**ORIGINAL ARTICLE** 

## Transcatheter arterial embolization for endoscopically unmanageable non-variceal upper gastrointestinal bleeding

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#### Abstract

**Objective.** Transcatheter arterial embolization (TAE) is a therapeutic option for endoscopically unmanageable upper gastrointestinal (GI) bleeding. We aimed to assess the efficacy and clinical outcomes of TAE for acute non-variceal upper GI bleeding and to identify predictors of recurrent bleeding within 30 days. Materials and methods. Visceral angiography was performed in 66 patients (42 men, 24 women; mean age,  $60.3 \pm 12.7$  years) who experienced acute non-variceal upper GI bleeding that failed to be controlled by endoscopy during a 7-year period. Clinical information was reviewed retrospectively. Outcomes included technical success rates, complications, and 30-day rebleeding and mortality rates. Results. TAE was feasible in 59 patients. The technical success rate was 98%. Rebleeding within 30 days was observed in 47% after an initial TAE and was managed with re-embolization in 8, by endoscopic intervention in 5, by surgery in 2, and by conservative care in 12 patients. The 30-day overall mortality rate was 42.4%. In the case of initial endoscopic hemostasis failure (n = 34), 31 patients underwent angiographic embolization, which was successful in 30 patients (96.8%). Rebleeding occurred in 15 patients (50%), mainly because of malignancy. Two factors were independent predictors of rebleeding within 30 days by multivariate analysis; coagulopathy (odds ratio [OR] = 4.37; 95% confidence interval [CI]: 1.25–15.29; p = 0.021) and embolization in  $\geq 2$  territories (OR = 4.93; 95% CI: 1.43–17.04; p = 0.012). Catheterization-related complications included hepatic artery dissection and splenic embolization. Conclusion. TAE controlled acute non-variceal upper GI bleeding effectively. TAE may be considered when endoscopic therapy is unavailable or unsuccessful. Correction of coagulopathy before TAE is recommended.

Key Words: angiography, embolization, gastrointestinal hemorrhage, hemostasis, therapeutic

## Introduction

Acute non-variceal upper gastrointestinal (GI) bleeding is a life-threatening condition accompanying various diseases. The average mortality rate in patients with upper GI bleeding is reported to be as high as 10% [1]. The most frequent cause of non-variceal upper GI bleeding is peptic ulcer, and other causes include malignant tumor, gastritis, duodenitis, vascular malformations, and Mallory–Weiss tears [2]. The initial treatment is based on the combination of medical therapy and endoscopic management [3]. However, failure of hemostasis occurs in up to 20% of cases [4], and such failure requires more effective treatment such as surgery or transcatheter arterial embolization (TAE). Although surgery is the traditional treatment of choice following failure of endoscopic hemostasis, it is an invasive procedure with a high mortality rate of up

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to 40% [5-7]. TAE is a reasonable and less hazardous alternative to surgery and has become the preferred therapeutic approach for the treatment of refractory non-variceal GI bleeding [8]. However, only a few studies have reported on the outcomes of TAE and the factors predicting embolization failure.

The aims of this study were to evaluate the effectiveness of TAE in patients with non-variceal upper GI bleeding when endoscopic hemostasis was unsuccessful or unable and to identify factors that can predict rebleeding within 30 days after TAE.

#### Materials and methods

#### **Patients**

This was a retrospective study performed at a single tertiary institution. We reviewed the medical records of all patients with acute upper GI bleeding who underwent visceral angiography for possible embolization during the 7 years from September 2006 to August 2013. Upper GI bleeding was defined as bleeding from an enteric source proximal to the ligament of Treitz [9]. Bleeding sites were identified either by endoscopic findings or clinical presentation. Patients with variceal bleeding, hemobilia, and bleeding into the peritoneal or retroperitoneal space were excluded. During the study period, visceral angiography was performed in 66 patients with acute upper GI bleeding. We collected data on patient demographics, clinical presentation, initial endoscopic findings, embolization procedures, and postembolization outcomes. This retrospective study was performed in compliance with the requirements of the institutional review board at our institution. Informed consent was not required.

#### Angiography and embolization technique

Angiography was performed with standard percutaneous transfemoral catheterization. The celiac trunk was examined first, splenic and superior mesenteric angiographic studies were performed using a 5-Fr selective catheter (Yashiro, Terumo, Japan), and the causative arteries were superselected using dedicated microcatheters (2.2 Fr, Asahi, Japan, and 2.4 Fr, Terumo, Japan) as necessary. When vascular abnormalities were demonstrated by angiography, embolic therapy was performed as selectively as possible. The embolic agents used were metallic coils ranging from 3 to 5 mm in diameter (Cook Medical, Bloomington, IN, USA), gelatin sponge (Spongostan, Ethicon, Inc., Somerville, NJ, USA), polyvinyl alcohol particles measuring 355-500 µm or 500-710 µm (Contour, Boston Scientific, Watertown, MA, USA), and cyanoacrylate surgical glue (Glubran; GEM SRL, Viareggio, Italy) mixed

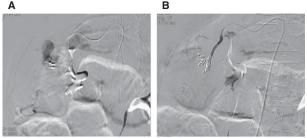


Figure 1. Images showing duodenal ulcer bleeding in a 54-year-old man. (A) Extravasation of contrast medium from the superior pancreaticoduodenal artery was noted, which was confirmed by two hemoclips. (B) The superior pancreaticoduodenal artery was selected with a 2.4 Fr microcatheter and embolized with multiple microcoils (Tornado  $3/2 \times 15$ , Vortx  $5/2 \times 1$ ).

with ultra-fluid lipiodol (Therapex; E-Z-EM, Montreal, Canada) in a 1:3 ratio. These agents were used singly or in combination. Figure 1 shows an example of the treatment of active bleeding from the pancreaticoduodenal artery.

We performed embolic therapy when there was contrast extravasation along with indirect signs of hemorrhage, including aneurysm/pseudoaneurysm, vessel irregularity, vessel cutoff and arteriovenous/arterioportal shunting, neovascularity, or increased vascularity from dilated arterioles [10]. We also used empirical or prophylactic embolization based on conclusive endoscopic identification of the source of bleeding. Postembolization arteriography was performed.

## Follow up

Follow-up information was available for all patients. Data on subsequent events were collected by reviewing the electronic medical records of patients. Coagulopathy was defined as an international normalized ratio >1.5, a partial thromboplastin time >45 s, or a platelet count <80,000/ml [11]. Initial hemostasis by endoscopy was defined as no evidence of bleeding from the treatment site after irrigation and 3 min of observation after endoscopic treatment [12]. Technical success was defined as immediate complete angiographic occlusion of all target vessels contributing to the area of hemorrhage [13]. Rebleeding was defined as bleeding within 30 days with >2.0 g/dl decrease in the hemoglobin level and/or lack of effectiveness of conservative medical treatment.

## Statistical analysis

Descriptive statistics were used to characterize the demographic features of the study population. For the univariate analysis, continuous variables are expressed as means (± standard deviation) or medians

Table I. Patient demographics (n = 66).

Measures	n (%)
Male	42 (63.6%)
Age (median and range, years)	61 (29-89)
Comorbidities	
DM	13 (19.7%)
HBP	18 (27.3%)
Heart failure	2 (3.0%)
IHD	3 (4.5%)
CVA	5 (7.6%)
Chronic liver disease	4 (6.1%)
Chronic renal failure	5 (7.6%)
Malignancy	41 (62.1%)
PUD	12 (18.2%)
Disease etiology	
Malignancy	36 (54.5%)
DU	14 (21.2%)
GU	6 (9.1%)
Post-surgery	6 (9.1%)
Angiodysplasia	1 (1.5%)
Pseudoaneurysm	1 (1.5%)
Dieulafoy's lesion	1 (1.5%)
Unknown cause	1 (1.5%)
Initial presentation	
Melena	27 (40.9%)
Hematemesis	16 (24.2%)
Melena + hematemesis	4 (6.1%)
Hematochezia	9 (13.6%)
Hematochezia + hematemesis	1 (1.5%)
Anemia	6 (9.1%)
Others	3 (4.5%)

Abbreviations: CVA = Cerebrovascular accident; DM = Diabetes mellitus; DU = Duodenal ulcer; GU = Gastric ulcer; HBP = High blood pressure; IHD = Ischemic heart disease; PUD = Peptic ulcer disease.

(interquartile range) and were compared using the Mann–Whitney U test. Categorical variables are expressed as the number (percentage) and were compared between groups using the chi-square test or the Fisher's exact test as appropriate. Variables with *p*-Values <0.10 by univariate analysis were included in a multivariate logistic regression model to identify the independent factors associated with early rebleeding. Statistical analysis was performed by using Statistical Analysis System software (version 8.02, SAS Institute, Cary, NC, USA). In all analyses, *p*-Values <0.05 were considered to be significant.

## Results

#### Patient demographics

Table I shows the demographics of all 66 patients. There were 42 men and 24 women, with a mean age of  $60.3 \pm 12.7$  years (range: 29–89 years). Most patients had a high surgical risk related to advanced age and comorbidities. Among them, 37 patients (56.1%) were older than 60 years, and 22.7% (15 of 66) were older than 70 years. Of the 66 patients, 22 (33.3%) had  $\geq 2$  comorbid conditions. Two predominant causes of bleeding were peptic ulcer bleeding (n = 20) and malignant bleeding (n = 36). The most common presenting symptom was melena (n = 27). A total of 33 patients (50%) exhibited shock (systolic blood pressure  $\leq 90$  mm Hg or pulse rate  $\geq 110$  beats/min) at the time of bleeding.

Endoscopy for therapeutic intervention was performed in 62 of the 66 patients (93.9%). More than half (56.5%) underwent emergency endoscopy. The bleeding source was localized in 56 of the 62 patients (90.3%). Of the 56 patients, 46 (82.1%) had active hemorrhage (spurting and oozing), and the others had only signs of recent hemorrhage (17.9%). Initial endoscopic hemostasis was achieved in 28 of the 62 patients (45.2%). However, recurrent bleeding after endoscopic intervention occurred after a mean period of 4.6 days (range: 1–15 days) and needed subsequent angiography.

Four patients did not undergo endoscopy before TAE. One patient strongly refused to receive endoscopy. One patient had advanced gastric cancer with malignant bleeding. His doctor in charge decided to perform angiography directly to control his bleeding. The other two patients had postsurgery bleeding. One showed bleeding at the afferent loop after gastrectomy as detected by a computed tomography (CT) scan. The other patient presented with GI bleeding at the antimesenteric border of the duodenal second portion after laparoscopic cholecystectomy.

#### Procedural and clinical outcomes

Among the 66 patients who had received angiography, 59 (89.4%) underwent embolization (Table II). Emergency (within 24 h) and urgent (24 h to 7 days) embolization was performed in 21 (35.6%) and 30 (50.8%) patients, respectively. Selective embolization was performed in 51 (86.4%) patients based on the findings of angiography, which were direct contrast extravasation (n=15, 25.4%) or indirect signs of hemorrhage (n=36, 61%). In the remaining 8 (13.6%) patients, no evidence of bleeding was identified by angiography, but the artery supplying the endoscopically identified bleeding site was embolized.

The artery in which embolization was performed most frequently was the gastroduodenal (or pancreaticoduodenal) artery (n = 25). Single-artery embolization was performed in 37 (62.7%) patients, two territories were embolized in 19 (32.2%) patients, and three or more territories were embolized in 3 (5.1%) patients. Multiple embolic agents were used more often than were single agents (37.3% vs. 62.7%).

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Table II. Summary of embolization procedures.

Measures	<i>n</i> = 59
Urgency of embolization	
Emergent (<24 h)	21 (35.6%)
Urgent (>24 h, <7 days)	30 (50.8%)
Remote (>7 days)	8 (13.6%)
Rationale for embolization	
Bleeding source visualized	
Contrast extravasation	15 (25.4%)
Indirect signs of hemorrhage	36 (61.0%)
Empiric or prophylactic	8 (13.6%)
Arteries embolized	
Left gastric	5 (8.5%)
Right gastric	1 (1.7%)
Gastroduodenal (or pancreaticoduodenal)	25 (42.4%)
Splenic	3 (5.1%)
Superior mesenteric artery – duodenal branch	3 (5.1%)
2 territories embolization	19 (32.2%)
$\geq$ 3 territories embolization	3 (5.1%)
Embolic agents	
Coils only	3 (5.1%)
Gelatin sponge only	7 (11.9%)
PVA particles only	6 (10.2%)
Histoacryl only	6 (10.2%)
Coils + gelatin sponge	8 (13.6%)
Coils + PVA particles	17 (28.8%)
Coils + histoacryl	6 (10.2%)
≥3 agents	6 (10.2%)

Abbreviation: PVA = Polyvinyl alcohol.

The combination of coils and gelatin sponge was used most often.

Embolization was technically successful in 58 of 59 patients, giving a technical success rate of 98.3% (Table III). In one patient, a pseudoaneurysm arising from the gastroduodenal stump was identified and treated with cyanoacrylate embolization, but this procedure failed because of migration of the cyanoacrylate. Coil embolization of the pseudoaneurysm was performed after stent placement at the junction between the common hepatic artery and proper hepatic artery. The patient did not experience any clinical sequelae, and his hospital course was uneventful.

All cases were divided into two groups, malignant bleeding versus nonmalignant bleeding, according to the causes of bleeding (Table III). Outcomes after embolization were compared between the two groups. Although technical success, rebleeding and overall mortality rate were not significantly different between the two groups, the proportion of deaths from underlying disease was significantly higher in the malignant bleeding group (p = 0.017).

Rebleeding occurred in 27 patients (46.6%) despite technically successful embolization after an initial TAE. The management of the patients with recurrent bleeding is summarized in Figure 2. Two of these patients underwent surgery and recovered well; five patients received endoscopic intervention, and one of them underwent palliative gastrectomy. Repeat embolization was performed in 8 of 27 patients with rebleeding and 1 achieved hemostasis by endoscopic intervention. Twelve of these patients received only conservative care for bleeding control, and most had malignant bleeding. The overall 30-day mortality was 42.4%. Among the deaths, seven were hemorrhagerelated deaths, and the others were caused by underlying disease.

#### Patients whose initial endoscopic hemostasis failed

The initial endoscopic hemostasis failed in 34 patients. The most frequent cause of bleeding was malignant bleeding (n = 21, 61.8%). Thirty-one (91.2%) patients underwent angiographic embolization, with a technical success rate of 96.8% (30 of 31). Rebleeding occurred in 15 patients (50%) and was managed by surgery (n = 1), repeat TAE (n = 4), and conservative care (n = 10). The overall 30-day mortality was 38.2%.

#### **Complications**

Complications from the procedure were one splenic artery embolization and one hepatic artery dissection. In the patient with splenic artery embolization, arterial occlusion caused a near-total infarction of the spleen. This patient had abdominal distension and left pleural effusion. After conservative treatment for 2 months, follow-up abdominal CT showed atrophic changes in the infarcted spleen, and his symptoms were resolved. In the patient with hepatic artery dissection, the distal portion of the coil had migrated into the hepatic artery. To remove the coil, we used a guiding sheath, catheter, and snare, but this failed, and hepatic artery dissection resulted, which led to hepatic artery occlusion. We placed a stent in the lumen of the hepatic artery to treat the coil occlusion and confirmed the preservation of hepatic artery blood flow using hepatic arteriography.

#### Predictors of early rebleeding

Patients who were treated successfully were compared with others to identify predictors of rebleeding (Table IV). By univariate analysis, rebleeding within 30 days was associated with coagulopathy (p = 0.045) and embolization of  $\geq 2$  territories (p = 0.024). The two factors were associated independently with early rebleeding in the multivariate analysis: coagulopathy (odds ratio [OR] = 4.37; 95% confidence interval [CI]: 1.25–15.29; p = 0.021), and embolization of  $\geq 2$  territories (OR = 4.93; 95% CI: 1.43–17.04; p = 0.012).

Measures		Etiology		
	All cases	Malignant bleeding	Non-malignant bleeding	Þ
Technical success	58/59 (98.3%)	36/36 (100%)	22/23 (95.7%)	0.207
Complications				
Splenic artery embolization	1		1	
Hepatic artery dissection	1		1	
Rebleeding (≤30 days)	27/59 (45.8%)	19/36 (52.8%)	8/23 (34.8%)	0.176
Mortality rate (≤30 days)	25/59 (42.4%)	17/36 (47.2%)	8/23 (34.8%)	0.346
Hemorrhage-specific	7/25 (28.0%)	2/17 (11.8%)	5/8 (62.5%)	0.017
Underlying disease	18/25 (72.0%)	15/17 (88.2%)	3/8 (37.5%)	

Table III. Postembolization outcomes.

\*Fisher's exact test.

## Discussion

Upper GI bleeding is one of the most serious challenges encountered by gastroenterologists in their clinical practice. Gastroduodenoscopy has been performed as a main tool of diagnosis and treatment for non-variceal upper GI bleeding [14,15]. With acid suppression by proton-pump inhibitors, endoscopic management has been shown to reduce rebleeding and mortality [16]. However, rebleeding rates have not significantly improved from longitudinal data in the past 15 years [17,18], which need salvage therapies such as radiological intervention or surgery. Treatment by angiographic embolization has been preferred over surgery because of improvements in interventional devices and embolic materials, and wider availability of experienced interventional radiologists. In this study, we evaluated the outcomes and rebleeding factors of TAE in patients with endoscopically unmanageable non-variceal upper GI bleeding.

In the present study, more than half of the included patients had cancer bleeding. That was the foremost reason for the rate of initial endoscopic hemostasis to appear quite low. In general, bleeding from upper GI tract cancer, such as gastric cancer, is not easy to control by endoscopic hemostasis. Patients with

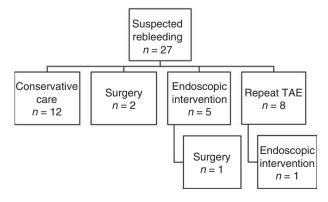


Figure 2. Flow diagram of subjects with suspected rebleeding within 30 days.

gastric cancer may benefit from acute surgery to control hemorrhage and may not be amenable to endoscopic intervention [19-21]. A previous study reported that initial endoscopic hemostasis was achieved in 10 of the 15 (67%) patients with upper GI tumor bleeding, among whom 8 patients experienced rebleeding [22]. Another report showed that endoscopic treatment was not effective as a primary treatment for bleeding caused by advanced gastric cancer [23]. In this study, initial hemostasis was achieved in only 14 of the 45 (31%) cancer patients. The authors concluded that, in the presence of large bleeding lesions (>2 cm) and unexposed vessels bleeding within a tumor, endoscopic hemostasis failure is predicted and TAE is recommended. Based on the findings of these studies, our institution considers TAE to be preferable as a rescue therapy for unmanageable cancer bleeding; our results are consistent with this practice.

TAE effectively managed non-variceal upper GI bleeding with high technical success (98.3%) in our study. Most patients had a high surgical risk related to advanced age and comorbidities. The initial embolization failed in only one patient because of migration of the cyanoacrylate. Many studies have reported technical success rates as high as 62% to 100% [24]. The causes of endovascular technique failure include difficult vascular anatomy, arterial dissection, vasospasm, false-negatively read angiograms, multiple bleeding, and tumor bleeding. Despite the technically successful embolization, the rebleeding rate within 30 days was relatively high at 46.6%. Many studies have reported rebleeding rates from 9% to 66% [24]. The mortality rate within 30 days was also somewhat high at 42.4%. One of possible explanations for such high rebleeding and mortality rate after TAE is that there was high proportion of malignant bleeding in the included cases. Whereas the rebleeding and mortality rate of non-malignant bleeding group were <35%, those of malignant bleeding group were 52.8% and 47.2%, respectively. Further, among a total 25 deaths

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Table IV. Predictors of rebleeding within 30 days of embolization.

Measures	Univariate analysis			Multivariate analysis		
	Clinical success $(n = 31)$	Rebleeding $(n = 27)$	Þ	Odds ratio	95% CI	Þ
Age (years)	$60.1 \pm 12.0$	$57.8 \pm 13.6$	0.49			
Male sex	16 (51.6%)	20 (74.1%)	0.08			
Coagulopathy	7 (22.6%)	13 (48.1%)	0.045	4.37	1.25-15.29	0.021
Anti-inflammatory drugs	8 (25.8%)	6 (22.2%)	0.70			
≥2 comorbidities	7 (22.6%)	12 (44.4%)	0.08			
Shock	17 (54.8%)	15 (55.6%)	0.96			
Hemoglobin <9.0 g/dl	19 (61.3%)	22 (81.5%)	0.10			
Initial endoscopic hemostasis	13/28 (46.4%)	11/26 (42.3%)	0.76			
Emergent embolization	11 (35.5%)	9 (33.3%)	0.86			
Positive bleeding source	27 (87.1%)	23 (85.2%)	0.83			
Positive extravasation	9 (29.0%)	6 (22.2%)	0.55			
≥2 territories embolization	7 (22.6%)	14 (51.9%)	0.024	4.93	1.43-17.04	0.012
Multiple embolic agent	20 (64.5%)	17 (63.0%)	0.90			
Malignant bleeding	16 (51.6%)	19 (70.4%)	0.15			
Number of PRBCs transfusion	$4.23 \pm 2.50$	$4.88 \pm 3.85$	0.48			

Abbreviation: PRBC = Packed red blood cell.

after TAE, 18 patients died by their underlying diseases, including malignancies.

Patients whose endoscopic hemostasis failed were also managed competently by TAE. Although the technical success rate was initially high, half of these patients experienced rebleeding afterward. The reason for this significant rebleeding rate also appears to be that >60% of them had malignant bleeding. A previous study reported that the clinical success rate of hemostasis by TAE for patients with unresectable gastric cancer was 52%, which was similar to our result [25].

We identified two independent rebleeding predictors by multivariate analysis: coagulopathy and embolization of  $\geq 2$  territories. Coagulopathy has been identified as a factor associated with rebleeding in previous studies, which showed ORs of 2.9-19.6 [11,26-28]. These results highlight the need for correcting coagulopathy before, during, and after embolization for upper GI bleeding. Embolization of  $\geq 2$  territories was associated with rebleeding. A possible explanation is that the site of bleeding could not be identified precisely in these patients; outcomes of blind embolization are controversial in published studies on this topic [9,29]. In the present study, 21 of 22 patients with embolization of  $\geq$ 2 territories did not show positive contrast extravasation but instead showed indirect signs of hemorrhage or a negative angiogram.

There is no single correct approach to resolve rebleeding after TAE, but surgery seems to play an important role. Six retrospective studies have compared outcomes of embolization versus surgery for continued or recurrent non-variceal upper GI bleeding after endoscopic hemostatic attempts [30]. Angiographic embolization was associated with a reduced rate of treatment-related complications. Mortality after either treatment was similar. In a pooled analysis, angiographic embolization was associated with a substantially higher rate of recurrent bleeding than was surgery.

There are limitations to this investigation. First, this study was designed as a retrospective study and was not randomized. Second, long-term follow up was not included in this study. There is a need for prospective, randomized, controlled studies comparing surgical and endovascular management of upper GI bleeding after endoscopic failure, although this task would be difficult to perform in the emergency setting. Third, as we mentioned previously, almost half of the patients had bleeding from upper GI malignancies. Although we can hardly think of non-variceal upper GI bleeding except malignancy, one should be more cautious to generalize the results in this study to nonvariceal upper GI bleeding cases.

In conclusion, TAE controlled acute non-variceal upper GI bleeding with high technical success and efficacy and should be considered when endoscopic therapy is not available or is unsuccessful. Angiographic failure can be predicted when embolization is required for  $\geq 2$  territories or coagulopathy is present. It is important to correct the coagulopathy before the embolization procedure.

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