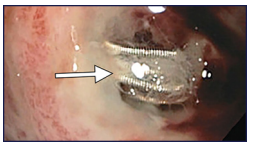

Outcomes of Transarterial Embolization for Acute Nonvariceal Upper Gastrointestinal Bleeding: Correlation with Periprocedural Endoscopy

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ABSTRACT

Purpose: To evaluate gastric and intestinal mucosal changes on postembolic endoscopy and mortality after transarterial embolization (TAE) for upper gastrointestinal bleeding (UGIB).

Materials and Methods: An institutional review board–approved retrospective review of patients who underwent arteriography for refractory UGIB at a multicenter health system from December 2003 to August 2019 was performed. Two hundred sixty-nine patients underwent TAE for UGIB. Data on etiology of bleeding, embolization technique, pre-embolic and postembolic endoscopic results, blood product requirements, and mortality were collected from the medical record. Endoscopy results were compared at the site of the target lesion before and after TAE. Multivariable logistic regressions were performed to assess predictors of new adverse mucosal responses and mortality.

Results: The most common etiology of UGIB was peptic ulcer. Twenty-five percent (n = 68) of the patients had clinical evidence of rebleeding after TAE, and the 30-day mortality rate was 26% (n = 73). Eighty-eight (32%) patients underwent post-TAE endoscopy, with only 15% showing new adverse mucosal changes after embolization. Procedural characteristics, including vascular territory and embolic choice, were not significantly predictive of increased risk of development of adverse mucosal response after TAE or increased mortality risk. No patients in the study were found to have bowel lumen stenosis at the time of post-TAE endoscopy or at 6 year follow-up.

Conclusions: TAE is a safe and effective intervention for patients with UGIB. Post-TAE endoscopy demonstrated that most patients had either stability or improvement in the target lesion after TAE, and only a minority of patients demonstrated adverse mucosal changes.

ABBREVIATIONS

CT = computed tomography, EGD = Esophagogastroduodenoscopy, GDA = gastroduodenal artery, GI = gastrointestinal, LGA = left gastric artery, MR = magnetic resonance, PEG = percutaneous endoscopic gastrostomy, TAE = transarterial embolization, UGIB = upper gastrointestinal bleeding

Acute, nonvariceal upper gastrointestinal bleeding (UGIB) is one of the most common gastrointestinal (GI) emergencies in the United States and carries considerable morbidity and mortality (1). Peptic ulcer disease is the most common etiology for UGIB, followed by malignancy, ischemia, gastritis, vascular malformation, Mallory-Weiss tear, trauma, and iatrogenic causes (2). Although endoscopy is considered the first-line management for evaluation and treatment of patients with UGIB, transarterial embolization (TAE) is often performed when endoscopy and medical therapy fail to control bleeding or if the patient is a poor surgical candidate (3,4).

TAE has been shown to be effective in the management of UGIB, with technical success (ie, successful embolization on arteriography) and clinical success (ie, absence of rebleeding within 30 days) rates ranging from 69% to 100% and 63% to 97%, respectively (5,6). TAE can be performed in multiple upper GI vascular territories with a variety of agents, including both permanent (eg, microspheres or platinum coils) and temporary (eg, gelatin sponge) embolic agents. TAE may be performed either empirically or when contrast extravasation is present (7). Although several

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RESEARCH HIGHLIGHTS

- A minority of patients demonstrated adverse changes on endoscopy after embolization for upper gastrointestinal bleeding (UGIB), with a majority demonstrating healing of the embolized lesion. No patients in the cohort were found to have bowel lumen stenosis at long-term follow-up.
- Procedural and clinical factors, including the vascular territory involved, the embolic agent used, and the etiology of UGIB, were not independently associated with an increased risk of adverse mucosal response after embolization.
- Preprocedural anemia in patients with UGIB was associated with increased mortality, emphasizing the importance of adequate resuscitation before intervention.

studies (5,8) have shown increased risk of rebleeding when either coils or gelatin sponge were used alone, the choice of embolic agent remains largely operator-dependent and vascular territory–dependent. In addition, despite the relatively high technical and clinical success rates, the 30-day mortality rate remains high, ranging from 4% to 46% (5).

Major complication rates for TAE in the setting of UGIB are generally low, ranging from 0 to 26% in major published series; however, the primary GI complications of TAE for UGIB, including embolization-related ischemia in the involved vascular territory, ulcer formation, and stenosis, carry significant morbidity (6,8,9–17). Patients with continued clinical concern for GI bleeding or with high-grade ulceration identified during initial endoscopy often undergo repeat endoscopy. Because the outlined complications primarily involve the gastric and intestinal mucosa, they may be visible at the time of follow-up endoscopy. Although it has been established that TAE for UGIB refractory to medical and endoscopic therapy is both safe and effective, there are limited data evaluating mucosal responses (ie, mucosal healing or induced injury) in the setting of TAE for UGIB. Studies published to date are characterized by small sample sizes, limiting the ability to inform patients, interventional radiologists, and other providers, including gastroenterologists, about the natural evolution of response to TAE (18,19). The purpose of this study was to evaluate the efficacy and safety of TAE for UGIB by examining mucosal changes identified on repeat endoscopy after TAE and to assess the associated mortality risk.

MATERIALS AND METHODS

Study Design

This retrospective study received approval from the institutional review board on human research at the Hospital of the University of Pennsylvania, and the requirement for informed consent was waived. Montage (Nuance Communications, Burlington, Massachusetts), a keyword search database for the health system picture archiving and communication system (PACS), was used to identify all patients who underwent arteriography for refractory UGIB from December 2003 to August 2019. Patients were excluded if they were younger than 18 years, the etiology of UGIB was secondary to trauma or varices, or the hemorrhage was determined to be distal to the ligament of Treitz.

STUDY DETAILS

Study type: Retrospective, observational, descriptive study

Level of evidence: 4 (SIR-D)

Procedural Technique for TAE

All embolizations were performed by fellowship-trained interventional radiologists (J.R.M.). Specific technique, catheter choice, and embolic agent were at the discretion of each interventionalist. In general, arterial access was achieved via the common femoral artery, and a vascular sheath was placed. Celiac arteriography was performed, and further selective diagnostic arteriography was performed through a microcatheter as indicated on the basis of those findings. When indicated, the microcatheter was placed in the desired position and embolization with the chosen agent was performed. Postembolic digital subtraction arteriography was performed to ensure stasis (20). After treatment of the intended vascular territory, the catheter and vascular sheath were removed, and hemostasis was achieved using manual compression or a vascular closure device.

Data Sources and Covariates

Demographic data, etiology of UGIB, 30-day mortality rates, and pre- and post-TAE transfusion requirements during the hospitalization as well as the international normalized ratio immediately before the start of TAE were recorded from the electronic medical record. Arteriography findings, embolization technique, and involved vascular territories were recorded from the procedure notes for TAE. Clinical evidence of rebleeding was defined as new onset of downtrending hemoglobin (drop > 1 g/dL) and/or new-onset hypovolemic shock (persistent tachycardia or hypotension unresponsive to fluid resuscitation) after TAE during the index admission (21–23). Performance of repeat TAE during the hospital admission was noted. Cause of death, when applicable, was recorded from the death certificate documentation.

Esophagogastroduodenoscopy (EGD) reports were collected immediately before and after TAE, where available. The etiology of bleeding, when noted in the report, was recorded or, otherwise, listed as unclear. Post-TAE endoscopy reports were evaluated and compared with pre-TAE endoscopy reports retrospectively. Post-TAE endoscopies performed within 90 days of TAE were included for analysis. Any new adverse sequelae of TAE that were not present on

initial EGD, including ischemia, stenosis, scarring, and edema, were recorded. The target lesion, when bleeding etiology was known, was compared with its appearance during pre-TAE EGD to assess for healing, defined as reduction in size or clinical reduction in inflammation as documented in the report. The clinical consequence of adverse mucosal changes was assessed within 90 days of follow-up EGD. Reports of follow-up imaging (including upper GI fluoroscopy, computed tomography [CT] of the abdomen, and magnetic resonance [MR] imaging of the abdomen) or EGD performed up to 6 years after TAE were evaluated to assess for the development of detectable bowel stenosis.

Statistical Analyses

Statistical analyses were performed using Prism version 9 (GraphPad Software, San Diego, California). Categorical variables were summarized as frequencies and percentages. Continuous variables were summarized as the mean ± SD or as the median with range, as indicated. Multivariable logistic regression models were used to analyze new adverse mucosal response on EGD and 30-day mortality, adjusting for multiple clinical and procedural factors. The regression modeling adverse mucosal response to TAE used the following cofactors: age of >60 years, male sex, use of coils, ulcer as UGIB etiology, TAE performed in the territory of the left gastric artery (LGA), TAE performed in the territory of the gastroduodenal artery (GDA), and active contrast extravasation. The regression modeling 30-day mortality used the following cofactors: age of >60 years, male sex, use of coils, use of gelatin sponge, ulcer as UGIB etiology, mass as UGIB etiology, iatrogenic UGIB etiology, TAE performed in the territory of the LGA, TAE performed in the territory of the GDA, a pre-TAE hemoglobin of <8 g/dL, and active contrast extravasation. An α level of 0.05 was used for statistical significance.

Cohort Characteristics

A total of 282 patients underwent arteriography for UGIB. Demographic data for the patient cohort are summarized in Table 1. The primary etiology of UGIB in this cohort was bleeding peptic ulcer (n = 132, 47%). Forty-two bleeding ulcers were esophageal or gastric in origin, and 90 bleeding ulcers were duodenal in origin. The next most common etiologies of UGIB were as follows: bleeding mass in 16% (n = 46), iatrogenic (including prior GI surgery) in 9% (n = 25), Dieulafoy lesion in 5% (n = 13), vascular in 2% (n = 6), Mallory-Weiss tear in 0.7% (n = 2), amyloid in 0.3% (n = 1), bleeding diverticulum in 0.3% (n = 1), and uncertain etiology in 20% (n = 56) of the patients.

Table 1. Demographic and Clinical Characteristics

Characteristic	Value (N = 282)
Demographic	
Age (y), median (Q1, Q3)	64 (52, 73)
Male sex	179 (63.5)
Race or ethnic group	
White	153 (54.3)
Black	100 (35.5)
Asian/Pacific Islander	8 (2.8)
Hispanic	6 (2.1)
Other/unknown	15 (5.3)
Clinical	
Etiology of UGIB	
Ulcer: gastric/esophageal	42 (14.9)
Ulcer: duodenal	90 (31.9)
Mass	46 (16.3)
Iatrogenic	25 (8.9)
Other or uncertain*	79 (28.0)
pRBCs required before TAE (units), mean (SD)	8.7 (7.5)
pRBCs required after TAE (units), mean (SD)	4.8 (6.6)
Clinical evidence of rebleeding	68 (24.1)
Mortality at 30 d	73 (25.9)
Underwent endoscopy after TAE	88 (31.2)

Note–Data are presented as frequencies and percentages, unless otherwise noted.
pRBC = packed red blood cell; TAE = transarterial embolization; UGIB = upper gastrointestinal bleeding
*Other included Dieulafoy lesion (13), Mallory-Weiss tear (2), vascular etiologies (6), amyloid (1), and diverticulum (1).

RESULTS

Procedural and Clinical Characteristics of the Cohort

Procedural characteristics for the patients are summarized in Table 2. A total of 235 (83%) patients underwent endoscopy before angiographic intervention. TAE was attempted in 277 (98%) patients and was technically successful in 269 (97%) patients. Seventy-one (30%) patients were found to have contrast extravasation at the time of arteriography, and 100% of these patients underwent successful embolization. Embolization was performed empirically in the remaining 198 (70%) patients. Angiography was performed in the territory of the GDA in 61% (n = 172) of patients and in the territory of the LGA in 34% (n = 96) of patients. In the vascular territory of the LGA, the most frequently used embolic agent was gelatin sponge alone (53 of 96 procedures), whereas in the vascular territory of the GDA, the most frequently used agents were coils with or without gelatin sponge (135 of 172 procedures). The mean international normalized ratio before TAE was 1.3 ± 0.2. Before arteriography, patients required a mean of 8.7 ± 7.5 units of packed red blood cells. After TAE, patients required a mean of 4.9 ± 6.8 units of packed red blood cells. Clinical evidence of rebleeding was present in 25% (n = 68) of patients. Seventeen (6%) patients underwent repeat TAE within the duration of their hospital admission for clinical suspicion of continued hemorrhage, with 6 (35.3%) demonstrating active extravasation at the time of repeat angiography.

Mucosal Response after TAE

Eighty-eight (31.2%) patients underwent EGD before and within 90 days after TAE. The median time to EGD after TAE was 5 days (Q1 = 3 days, Q3 = 16 days). Mucosal

Table 2. Procedural Characteristics

Characteristic	Value (N = 282)
Embolization performed	269 (95.4)
Vascular territory involved	
LGA	96 (34.1)
Active contrast extravasation	24 (25.0)
Repeat embolization	7 (7.3)
No embolization performed/technical failure	6 (6.2)
Coils ± gelatin sponge	33 (34.4)
Gelatin microspheres ± gelatin sponge	4 (4.2)
Gelatin sponge only	53 (55.2)
GDA	172 (61.0)
Active contrast extravasation	44 (25.6)
Repeat embolization	8 (4.7)
No embolization performed/technical failure	7 (4.1)
Coils ± gelatin sponge	135 (78.5)
Gelatin microspheres ± gelatin sponge	2 (1.2)
Gelatin sponge only	28 (16.3)
Other vascular territory	14 (4.9)
Active contrast extravasation	3 (21.4)
Repeat embolization	2 (14.3)
No embolization performed/technical failure	0 (0.0)
Coils ± gelatin sponge	10 (71.4)
Gelatin microspheres ± gelatin sponge	0 (0.0)
Gelatin sponge only	4 (28.6)

Note–Data are presented as frequencies and percentages. Other vascular territory included LGA and GDA or superior mesenteric artery.
GDA = gastroduodenal artery; LGA = left gastric artery.

changes at the site of the target lesion assessed at the time of follow-up EGD are summarized in Table 3. A total of 20 (23%) patients showed improvement in the target lesion after TAE, with findings including healing ulcer and resolved lesion. Thirty-eight (43%) patients showed no change in the target lesion at the time of follow-up EGD. New adverse findings, including ischemia, ulceration, inflammation, and scarring, were present in 15% (n = 13) of patients. Ongoing bleeding was present in 6% (n = 5) of patients. Twelve (14%) patients were noted only to have resolved bleeding at the time of follow-up because direct visualization of the lesion targeted at TAE was not achieved at the time of initial EGD. Of patients with new adverse findings, 23.1% (n = 3) demonstrated clinical consequences, with all 3 patients experiencing rebleeding. Each of these patients were found to have new or worsening ulceration after TAE, with 1 requiring repeat TAE.

Results of a multivariable logistic regression modeling new adverse EGD findings after TAE with clinical factors as predictors are summarized in Table 4. Bleeding etiology and procedural factors, including the target vessel distribution and embolic agent, were not significantly predictive of increased risk of adverse mucosal response. Active extravasation at the time of angiography approached but did not reach significance as a factor for increased risk of adverse mucosal response (odds ratio, 2.91; 95% CI, 0.86–9.86).

Pre- and post-TAE EGD findings from 3 patients from the cohort have been included to illustrate potential mucosal

Table 3. Mucosal Changes within 90 Days after Transcatheter Embolization.

Endoscopy findings at follow-up	Frequency (n = 88)	Percentage
Resolved bleeding: stable lesion	38	43.2
Resolved bleeding: improvement in lesion		
Healing ulcer	17	19.3
Resolved lesion*	3	3.4
Resolved bleeding: new adverse findings		
Ischemic changes	4	4.5
Ulceration	5	5.7
Inflammation/edema	2	2.3
Scarring/deformity†	2	2.3
Resolved bleeding alone‡	12	13.6
Ongoing bleeding	5	5.7

*Included resolved Dieulafoy lesion and resolved bleeding mass.
†Included extrusion of embolization coil in 1 patient and scarring in 4 patients.
‡Patients in this category underwent pre–TAE and post–TAE endoscopy; however, the target lesion was unable to be compared owing to profuse bleeding at the time of initial endoscopy.

Table 4. Multivariable Logistic Regression Assessing Predictors of Adverse Mucosal Response after Transarterial Embolization

Predictor variable (n = 88)	Odds ratio	*P* value	95% CI
Demographic data			
Age: ≥60 y	0.79	.68	0.25–2.51
Male sex	0.47	.28	0.12–1.8
Etiology of bleeding			
Ulcer	0.63	.46	0.19–2.12
Embolic agent used*			
Coils	0.47	.50	0.05–4.23
Vascular territory embolized			
Left gastric artery	2.38	.42	0.28–19.90
Gastroduodenal artery	1.59	.69	0.19–13.00
Active contrast extravasation at time of TAE	2.91	.08	0.86–9.86

TAE = transcatheter embolization.
*Coil category included any embolization performed using coils with or without other embolic agents. Gelatin sponge was used as the reference category for the embolic agent used.

outcomes after TAE, highlighted in Figure 1a–c, Figure 2a, b, and Figure 3a,b. Patient A, with imaging findings shown in Figure 1a–c, was a 66-year-old man with a history of a bleeding duodenal bulb ulcer refractory to endoscopic therapy who became hemodynamically unstable and subsequently underwent empiric GDA embolization with platinum coils. Follow-up EGD at 1 week demonstrated a healing duodenal bulb ulcer with exposed embolization coils (Fig 1a). Three months later, EGD was notable for re-epithelialization of the bowel lumen over the deployed coil (Fig 1b). Abdominal CT obtained 3 years after TAE for an unrelated indication was significant for embolization coils still in place in the territory of the GDA (Fig 1c). Demonstrated in Figure 2a, b, Patient B was a 69-year-old man with a history of abdominal aortic

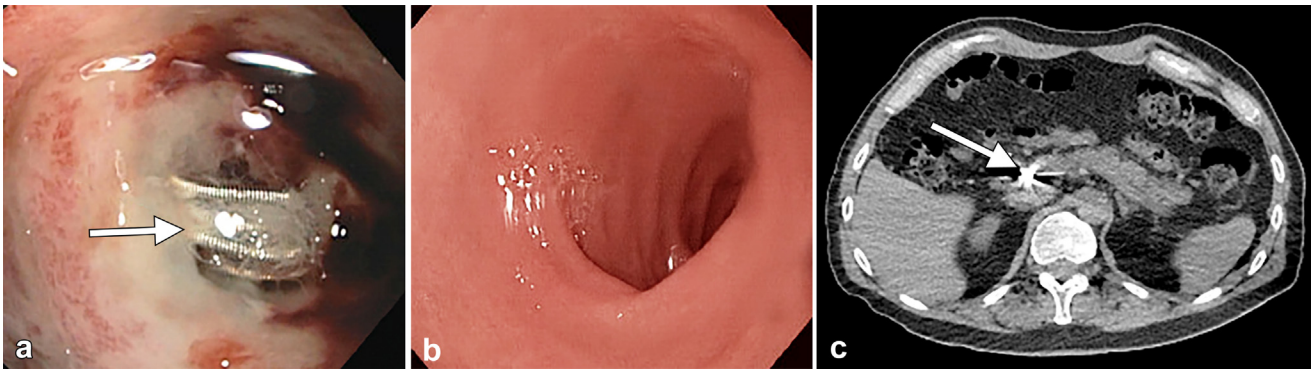

Figure 1. (a) Post-TAE endoscopy at 1 week demonstrated extrusion of an embolization coil through the bowel lumen (arrow) in the setting of a duodenal ulcer. **(b)** Post-TAE endoscopy 90 days later demonstrated re-epithelialization of the bowel lumen over the embolization coil and resolution of the index ulcer. **(c)** Computed tomography image of the abdomen obtained 3 years after embolization for an unrelated indication demonstrated the embolization coil still in place (arrow).

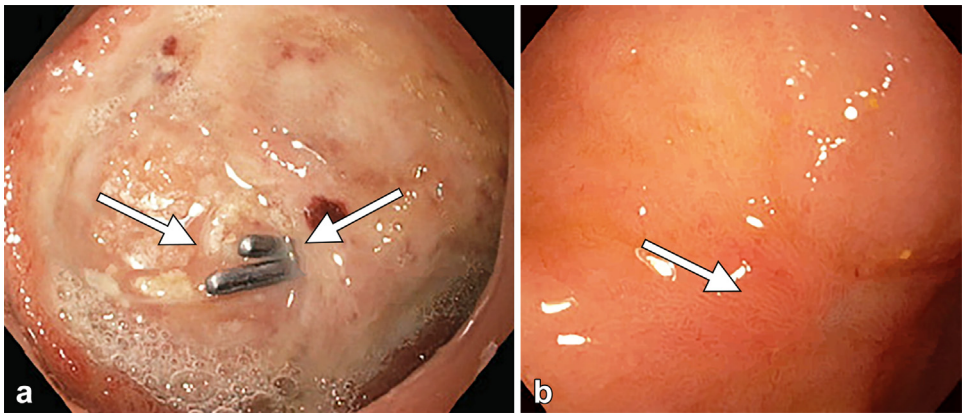

Figure 2. (a) Pre-TAE endoscopy demonstrated a large, deep, clean-based ulceration in the duodenal bulb, with 2 surgical clips from prior aortic surgery eroded into the bowel lumen (arrows). **(b)** Postembolic endoscopy 3 months later showed re-epithelialization and scarring of the duodenal lumen (arrow) over the ulcer and exposed surgical clip site.

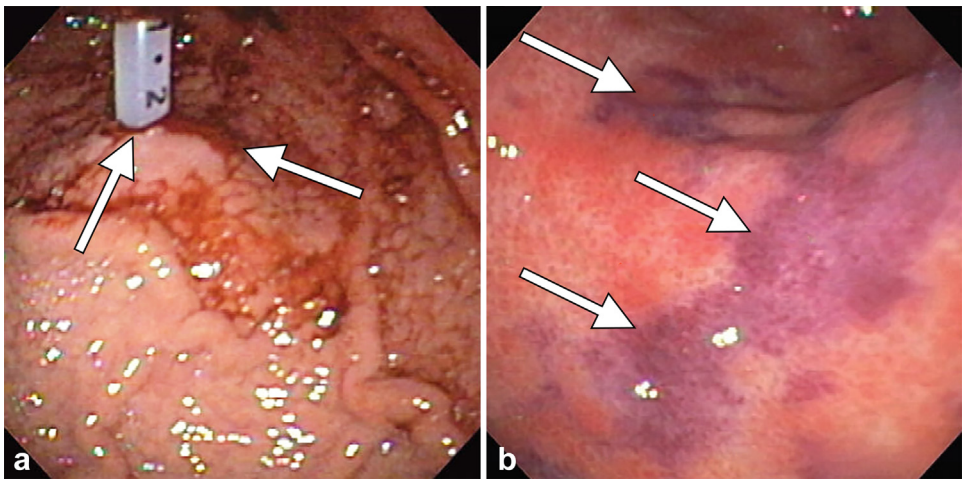

Figure 3. (a) Pre-TAE endoscopy showed active bleeding and friable mucosa at the site of a percutaneous endoscopic gastrostomy tube (arrows). **(b)** Endoscopy at 8 days post-TAE demonstrated new-onset ischemic changes (arrows) to the gastric mucosa.

Table 5. Multivariable Logistic Regression Assessing Predictors of 30-Day Mortality

Predictor variable (N = 282)	Odds ratio	*P* value	95% CI
Demographic data			
Age: ≥60 y	1.16	.62	0.65–2.07
Male sex	0.59	.13	0.30–1.17
Etiology of bleeding			
Peptic ulcer	1.16	.66	0.59–2.30
Mass	1.08	.87	0.45–2.59
Iatrogenic	0.36	.14	0.10–1.40
Embolic agent used*			
Coils	0.95	.93	0.31–2.97
Gelatin sponge	1.05	.94	0.31–3.50
Vascular territory embolized			
Left gastric artery	1.20	.68	0.50–2.86
Gastroduodenal artery	1.57	.24	0.74–3.35
Pre-TAE hemoglobin of <8 g/dL	1.30	.03	1.12–1.50†
Active contrast extravasation at time of TAE	0.78	.44	0.42–1.46

TAE = transcatheter embolization.
*Coil category included any embolization performed using coils with or without other embolic agents. Gelatin sponge category included any embolization using gelatin sponge alone without other embolic agents.
†Denotes a significant association.

aneurysm repair with subsequent aortoenteric fistula formation and duodenal ulceration with active UGIB. There was evidence of previously applied surgical vascular clips that had eroded into the duodenal lumen. The patient underwent empiric embolization of the GDA with coils. Postprocedural EGD at 3 months demonstrated healing of the ulceration with re-epithelialization of the lumen over the surgical clips. Finally, as illustrated in Figure 3a, b, Patient C was a 46-year-old man with a medical history significant for peptic ulcer disease and alcoholic cirrhosis complicated by duodenal ulcer perforation, necessitating surgical intervention with an extensive intensive care unit stay. The patient subsequently developed hepatic encephalopathy that required placement of a percutaneous endoscopic gastrostomy (PEG) tube complicated by refractory bleeding in the distal gastric antrum/proximal duodenum near the site of the PEG tube. The GDA was subsequently empirically embolized with coils. After TAE, EGD demonstrated discoloration of the gastric mucosa adjacent to the PEG site, indicative of post-TAE ischemic change; however, the patient remained asymptomatic and did not require any intervention.

Mortality

The 30-day mortality rate in the complete cohort was 26% (n = 73), including death as a direct result of UGIB and other causes. Results from a multivariable logistic regression modeling 30-day mortality rates are summarized in Table 5. Periprocedural anemia was predictive of an increased mortality risk (odds ratio, 1.30; 95% CI, 1.12–1.50). There was no association between patient demographics, the etiology of bleeding, the involved vascular territory, the choice of embolic, or the presence of active contrast extravasation at the time of arteriography with increased mortality.

Long-term Evaluation of TAE-Associated Complications

Of note, there was no evidence of bowel stenosis in the area of embolization in any patient at the time of follow-up EGD. Of patients in whom EGD was performed after TAE, 49 (56%) underwent imaging of the GI tract within 6 years after TAE, including CT of the abdomen (28), EGD (n = 13), oral contrast-enhanced upper GI fluoroscopy (n = 6), or MR imaging (n = 2). The median time to follow-up imaging was 162 days (range, 19–2,099 days). There was no evidence of bowel lumen stenosis noted on follow-up imaging reports of any patient in the cohort.

DISCUSSION

The presented large cohort study underscores the safety and effectiveness of TAE in the treatment of UGIB with high rates of technical success (97%) and low rates of post-embolic rebleeding (25%). Post-TAE endoscopy demonstrated a low incidence (15%) of adverse sequelae with most patients (80%) showing either no change or evidence of healing in the target lesion. Nonetheless, despite high technical and clinical success, the 30-day mortality rate remained high at 26%. These data hold important implications for informing interventional radiologists, patients, and other providers involved in their care about the evolution of the response to TAE and the role of follow-up EGD.

Most (73%) of the patients in our cohort underwent empiric embolization because they did not have evidence of contrast extravasation at the time of arteriography. Active contrast extravasation at the time of TAE was not predictive of increased mortality. Most of the patients underwent embolization in the territory of the LGA or the GDA, consistent with the fact that most patients undergoing TAE were found to have bleeding gastric or duodenal ulcers. Most patients received permanent embolic agents (ie, coils with or without gelatin sponge) in keeping with most patients experiencing bleeding in the GDA territory. Coils are often regarded as the preferred embolic agent in the GDA given the risks of nontarget embolization near the pancreatic circulation.

Bowel ischemia is a risk of TAE in the acute setting, resulting from interrupted perfusion. The evidence of this complication at the time of follow-up EGD was low, and TAE did not prevent mucosal healing. This is consistent with the rich collateral circulation of the upper GI system providing ample blood flow in support of healing. No specific independent associations between clinical or procedural factors and the development of adverse mucosal response were noted, suggesting that the choice of embolic agent does not adversely affect mucosal healing and that embolic choice should remain at the discretion of the interventionalist as dictated by the clinical scenario.

Furthermore, of patients with new adverse mucosal response, few (26%) required treatment, emphasizing the safety of TAE.

Because most of the patients who underwent GDA embolization received permanent embolic agents (ie, coils), long-term ischemia leading to bowel lumen stenosis remains a concern. At long-term follow-up, however, no patients with available imaging demonstrated evidence of bowel lumen stenosis, again highlighting the protective mechanism that the rich collateral circulation may provide. Underscoring these data, 1 patient in the present cohort showed evidence of embolization coil extrusion through the bowel wall on immediate EGD follow-up but subsequently experienced complete mucosal healing and re-epithelialization over the exposed coil on longer-term follow-up without any evidence of ischemic sequelae (Fig 1a–c). Because most of the patients demonstrated no active contrast extravasation at the time of arteriography, these EGD findings suggest that empiric embolization remains an effective strategy for treatment of UGIB that is safe for the GI mucosa. Moreover, requiring the presence of active extravasation on CT angiography to justify proceeding with embolization of UGIB may be unwarranted. In the authors' institution, CT angiography is used for the workup of lower GI bleeding but is not part of the therapeutic pathway for UGIB, noting that CT angiography may provide value in some clinical circumstances (eg, when the bleeding site remains unclear).

Although mortality remained high in this cohort, consistent with previous studies, no procedural factors were found to be associated with increased mortality. In the presented data, periprocedural hemodynamic instability was found to be associated with an increased mortality risk, underscoring the importance of adequate resuscitation of these patients in addition to interventional management.

This study has several limitations, which primarily issue from its retrospective nature. Endoscopic and embolization technique were not standardized, nor was periprocedural workup or care. Not all patients underwent post-TAE endoscopy or follow-up imaging, and the timing of follow-up was not standardized. Of those patients who did undergo post-TAE EGD, the indications for the procedure were not consistently available. This led to evaluation of GI mucosa at differing stages of healing in the analysis, limiting direct comparisons. In addition, procedures were performed by multiple different gastroenterologists and interventional radiologists, and outcomes may, in part, be related to the operator's experience and expertise, which could not be controlled for in this type of study. In regard to long-term follow-up, the assessment of bowel stenosis using cross-sectional imaging (eg, CT or MR imaging of the abdomen) may have been limited by the sensitivity of these modalities for this condition without the use of specialized protocols. Finally, the small sample size in many procedural subcategories, including choice of embolic agent and vascular territory, limited the statistical power required to perform subgroup analyses.

Overall, this study found TAE to be a safe and effective treatment for UGIB refractory to endoscopic management. Most of the patients who underwent TAE showed either no change or improvement in the appearance of the target mucosa at the time of follow-up EGD. There were no procedural or clinical factors associated with adverse mucosal response after TAE in this cohort. No patients in the cohort were found to have bowel lumen stenosis at the long-term follow-up.

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