



Acute Nonvariceal Gastrointestinal Bleeding: A Comprehensive Review and Approach for an Interventional Radiologist

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Abstract

Gastrointestinal bleeding (GIB) is one of the most common leading life-threatening conditions requiring prompt diagnosis and rapid endoscopic and interventional radiology (IR) management. Endoscopy is the first line of management for upper GIB, while it has a limited role in lower GIB, especially in acute clinical settings, due to poor bowel preparation. Patients with failed and refractory endoscopic management necessitate emergent computed tomography angiography (CTA) evaluation. CTA is crucial in assessing underlying causes and planning transarterial embolization (TAE). It has been almost three decades since IR gained popularity by virtue of increased technical experience, availability of sophisticated hardware, and evolving techniques. Newer embolization agents and catheters, as well as the established role of CTA, have expanded and enhanced the role of IR in the management of GIB. TAE has been proven to be a safe, rapid, effective, and minimally invasive procedure alternative to surgery when endoscopic management fails to control GIB. We present a comprehensive approach for managing nonvariceal GIB, including CTA protocol, anatomical variants, visceral to visceral collateral pathways, and specific embolization techniques. This article will help readers get an insight into TAE that will help better management of patients with GIB.

Keywords

- ▶ nonvariceal
- ▶ gastrointestinal bleeding
- ▶ embolization
- ▶ embolic materials

Introduction

Acute nonvariceal gastrointestinal bleeding (GIB) is a common life-threatening clinical condition in 1 to 2% of hospitalized patients, which carries a high degree of mortality (25%) and requires timely intervention.^{1,2}

Prompt identification and management of bleeding sources may significantly reduce morbidity and mortality.¹ The severity of bleeding and location of the potential source in

the GI determine the variable diagnostic and therapeutic strategies for further management.¹

Initial endoscopic evaluation is an essential diagnostic tool in upper GIB (UGIB) with high sensitivity (92–98%) and variable specificity (3–100%) and helps in the effective management of GIB in a majority of cases.³ Lower GIB (LGIB) resolves spontaneously in 80 to 85% of patients and remains challenging for endoscopic evaluation due to poor bowel preparation.^{4,5}

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Further, surgery and transarterial angioembolization (TAE) are the essential options in refractory acute nonvariceal GIB patients who do not respond to initial resuscitation and endoscopic intervention. Surgery is typically reserved for the hemodynamically unstable patient. However, TAE has gained popularity and has almost replaced surgery due to significantly low procedure-related morbidity compared to surgery.^{6,7}

Diagnostic computed tomography angiography (CTA) study is essential in planning and executing TAE. CTA is an accurate examination for identifying the source of acute GIB. A meta-analysis of data from 672 patients with moderate-to-severe UGIB and/or LGIB revealed an overall sensitivity of 85% and a specificity of 92% for the detection of the bleeding site.⁵

In addition, knowledge of various anatomical variants, arterial collateral pathways, embolization techniques, and appropriate selection of embolic agents in a proper clinical setting is vital for a successful TAE.^{5,7}

Various forms of embolization techniques have not been described in detail in the available literature. This article aims to highlight the role of TAE in non-variceal GIB, including initial clinical presentation, and management algorithm; planning of TAE; diagnostic evaluation, including CTA protocol and anatomical considerations; embolization techniques and selection of embolizing material and their classification

Clinical Presentation and Management Algorithm

Management of acute GIB varies widely and depends on the patient's clinical severity and hemodynamic status. Patients with overt or acute GIB with signs of hemodynamic instability, massive blood loss, and ongoing resuscitation require urgent surgical intervention.^{8–10}

Hemodynamically stable patients with nonvariceal UGIB with limited blood loss usually undergo UGI endoscopy, which is considered the diagnostic and therapeutic modality of choice. Endoscopic hemostasis can be achieved by epinephrine injection, sclerotherapy, clipping, hemostatic powder, thermal therapy, or ligation. Studies have shown excellent clinical success in achieving primary hemostasis in up to 90% of patients undergoing this procedure.^{11,12} Endoscopy in an acute lower GIB setting becomes challenging. It has poor outcomes in identifying active bleeding sources due to poorly prepared bowel, hemodynamic instability, old age, and ongoing bleeding^{13,14} (► Fig. 1).

TAE Planning and Diagnostic Evaluation

Robust planning is of utmost importance as these patients may become unstable if not attended timely. Intensive care and anesthetist support should be sought wherever required. CTA and endoscopic findings must be discussed with clinical colleagues for risk stratification of rebleeding and bowel ischemia.

Protocol of CTA

There is no consensus for a single CTA protocol for acute nonvariceal GIB; however, arterial and venous phase imaging

of the abdomen is an integral part of CTA. CTA is typically performed as a part of TAE procedure planning, especially during active GIB or acute clinical presentation. It helps in anatomical localization of the source of bleeding, defines the underlying disease process, and gives us a road map for TAE by depicting various anatomical arterial variants and collateral pathways.⁵

Our institution typically performs three-phase abdominal CT scans without oral contrast. It includes a noncontrast scan, followed by an arterial angiographic phase after 20 to 25 sec of intravenous contrast injection, and a venous phase follows after 70 to 80 seconds of contrast injection covering the entire abdomen, extending from the dome of the diaphragm to the lesser trochanter of femur. CTA with all possible reconstructions is vital to identify the source of bleeding.

Delayed imaging is not routinely performed at our institute; however, in case with slow rates of bleeding contrast leak may be identified only in delayed images.¹⁵ Noncontrast scan delineates the pre-existing hyperdensities like ingested food particles, tablets, or metallic clips placed by endoscopy. These hyperdensities could be misinterpreted as active arterial bleeding within the GI lumen.¹⁵

CTA Interpretation and Anatomical Considerations

CTA signs of active arterial GIB vary according to underlying etiologies, including active contrast material extravasation, focal contrast outpouching (pseudoaneurysm), vasospasm, vessel wall irregularities, abrupt vessel cutoff, expanding hematomas, or early filling of draining vein (Arterio-venous fistula).¹⁵

UGIB and LGIB confine to the celiac axis, superior and inferior mesenteric arteries (SMA and IMA), and their branches. Chronic occlusion of any major GI arterial branches may open arterial collateral pathways, which serve as the main arterial supply to the involved GI tract. Their importance further increases in cases of GIB as these pathways prevent potential acute GI ischemia/infarction after angioembolization but at the same time augments the chances of rebleeding. Major collateral pathways are described here:

1. Superior and inferior pancreaticoduodenal arteries are branches of the celiac axis and SMA.¹⁶ Anterior and posterior divisions of these arteries form rich arterial collateral surrounding the pancreatic head, known as the "pancreaticoduodenal arcade." Arterial collateral loops of this arcade become prominent and tortuous if the origin of celiac or SMA is chronically occluded¹⁷ (► Fig. 2).
2. Dorsal pancreatic artery, a branch of the splenic artery, also communicates with the pancreaticoduodenal arteries and distal splenic artery through "the pancreaticoduodenal arcade"¹⁷ (► Fig. 3).
3. Arc of Buhler is a standalone persistent embryological anastomotic connection between celiac and SMA, failing to regress.¹⁸

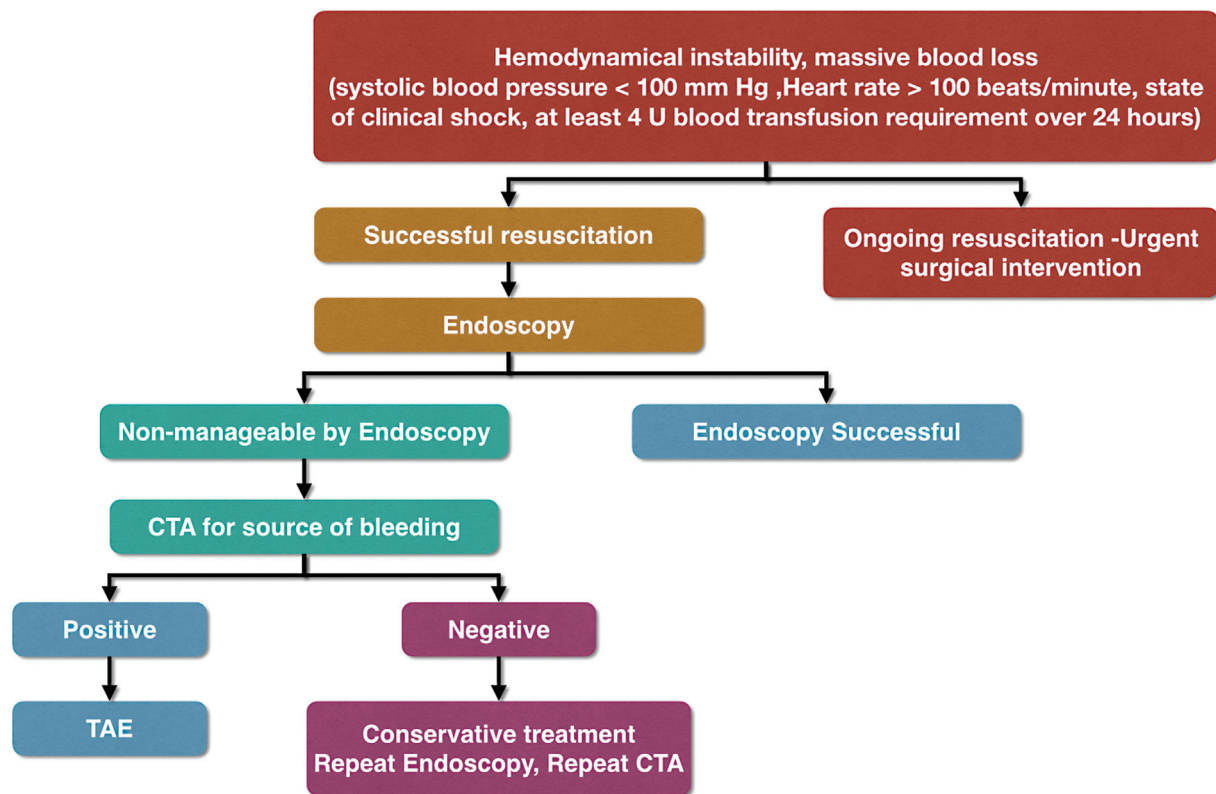


Fig. 1 Nonvariceal gastrointestinal bleeding patient management algorithm. CTA, computed tomography angiography; TAE, transarterial embolization.

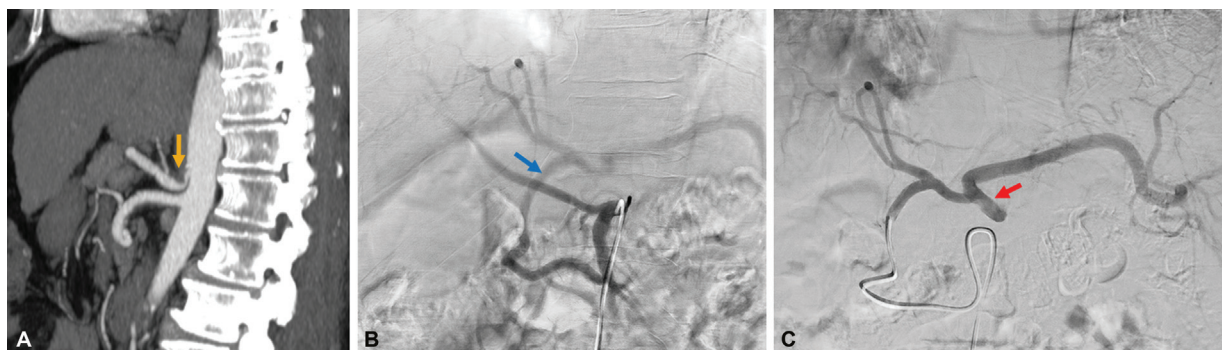


Fig. 2 Pancreaticoduodenal arcade; Superior–inferior pancreaticoduodenal arterial collateral pathway in a 66-year-old patient with celiac axis stenosis; (A) Sagittal computed tomography angiography shows severe stenosis of celiac axis origin (yellow arrow). (B) Selective angiography of superior mesenteric artery shows tortuously dilated inferior pancreaticoduodenal artery forming pancreatic arcade with retrograde filling of proper/common hepatic arteries via gastroduodenal artery (GDA) (blue arrow). (C) Selective catheterization and angiography of GDA shows complete retrograde visualization of celiac axis (red arrow) and its branches.

4. Arc of Barkow is an omental collateral connection between terminal branches of the right gastroepiploic artery (branch of gastroduodenal) and the left gastroepiploic artery (branches of the splenic artery)¹⁸ (► Fig. 4).
5. Right gastric artery is a small branch and usually arises from the proper hepatic artery and finally anastomoses with terminal branches of the left gastric artery^{19,20} (► Fig. 5).

TAE is challenging in lower GIB due to typical end arterial anatomy as it may lead to bowel ischemia, although it reduces the possibility of rebleeding. Some authors describe small bowel bleeding as a separate entity as mid-GIB, and

bleeding distal to the ileocecal valve is considered lower GIB.⁴ Major arteries involved in lower GIB are SMA and IMA. The marginal artery of Drummond is a major collateral pathway that runs along the mesenteric border of the colon. It communicates between SMA's middle colic branch and IMA's left colic branch¹⁷ (► Fig. 6).

The Arc of Riolo is an anastomotic channel that lies deep to the mesentery and has a radial course. It provides collateral flow between SMA and IMA in the presence of significant chronic occlusion of either origin²⁰ (► Fig. 6).

Superior and middle rectal arteries also form the arterial collateral arcades between IMA and internal iliac arteries.^{19–22}

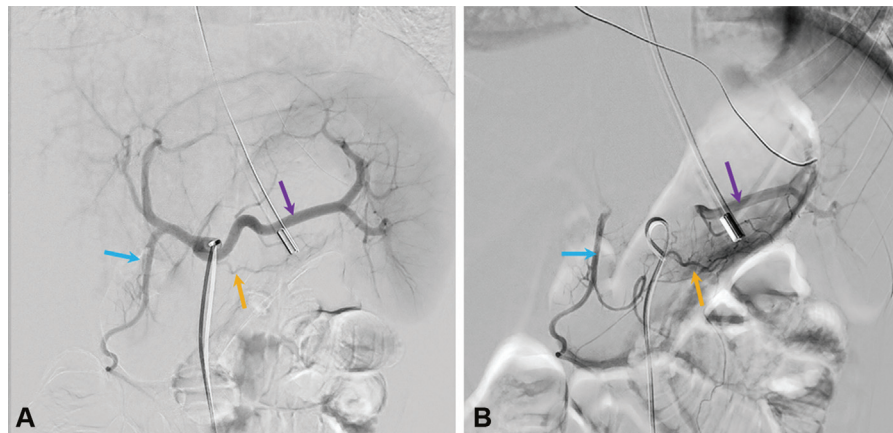


Fig. 3 Collateral pathways between dorsal pancreatic artery (branch of splenic artery) with anterior/posterior pancreaticoduodenal arteries (branch of superior mesenteric artery) (A) Selective angiography of celiac axis shows normal angiographic arterial anatomy including splenic artery (violet arrow) and gastroduodenal artery (GDA) (blue arrow). (B) Inadvertent catheterization of dorsal pancreatic artery (yellow arrow) shows pancreatic parenchymal blush and pancreatic arcade with retrograde filling of GDA and splenic artery via collaterals.



Fig. 4 Arc of Barkow: Collateral pathway between right-left gastroepiploic arteries. (A) Selective angiography of celiac axis shows normal angiographic arterial anatomy including hepatic artery (violet arrow) and gastroduodenal artery (blue arrow). Note anastomosis between right and left gastroepiploic arteries (yellow arrows). (B) Maximum intensity projection coronal computed tomography angiography image of another patient shows anastomosis between right and left gastroepiploic arteries along the greater curvature of stomach (yellow arrows).

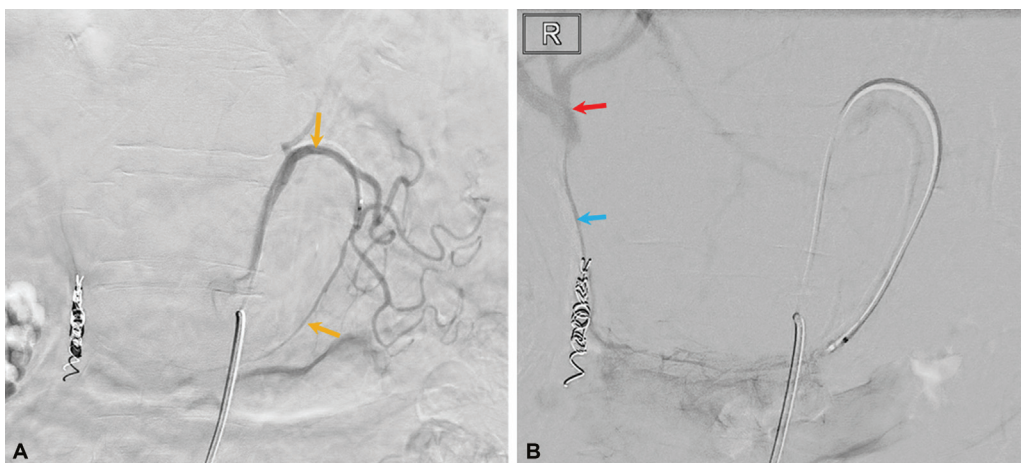


Fig. 5 Collateral pathway between right-left gastric arteries: (A) Superselective angiography of left gastric artery (LGA) and (B) distally placed microcatheter into LGA (yellow arrows) demonstrates communication with the right gastric artery (blue arrow) and opacification of hepatic arteries (red arrow).

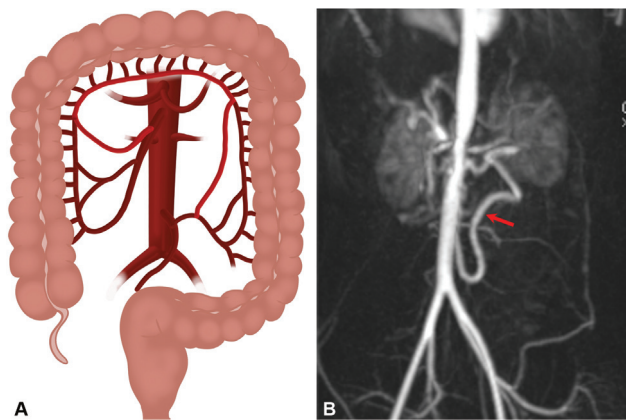


Fig. 6 Superior and inferior mesenteric arterial anastomotic collaterals: (A) Marginal artery of Drummond (yellow arrows). (B) Coronal maximum intensity projection abdominal aortic magnetic resonance angiography in an 18-year-old male patient with midaortic syndrome demonstrates prominent arc of Riolo.

Anatomical Variants

As a general rule, right-sided replaced and accessory hepatic arteries have aberrant origin from SMA. In contrast, left-sided

arteries arise from the left gastric artery, and literature suggests that these are equally common in frequencies of 20% population.^{23–26}

Important diagnostic clues may prompt the diagnosis on CTA as the aberrant right hepatic artery courses posterior to the main portal vein (→ **Fig. 7**). In contrast, the left aberrant hepatic artery can be easily identified in the fissure of ligamentum venosum (→ **Fig. 8**).

The replaced hepatic artery supplies the entire liver lobe, while the accessory hepatic artery supplies partial liver volume. These anatomical variants should be kept in mind as these could be potential source of GIB.^{7,8} Other anatomical variants are infrequent and not relevant here.

TAE Technique

Catheterizing major mesenteric vessels of the aorta and negotiating the catheter through smaller branches are essential technical and skill based processes. Diagnostic conventional aortograms for arterial mapping can be avoided if prior CTA is available, reducing unnecessary time delay, contrast use, and radiation dose. Accurate superselective placement of a microcatheter within a distal artery and its



Fig. 7 Replaced right hepatic artery in a 44-year-old male patient; (A) Axial computed tomography angiography image at the level of superior mesenteric artery (SMA) shows replaced right hepatic artery coursing behind the main portal vein. (B) Selective angiography of SMA demonstrates replaced right hepatic artery arising directly from proximal SMA. (C) Selective celiac axis angiography in the same patient shows proper hepatic artery continue as left hepatic artery (blue arrow).

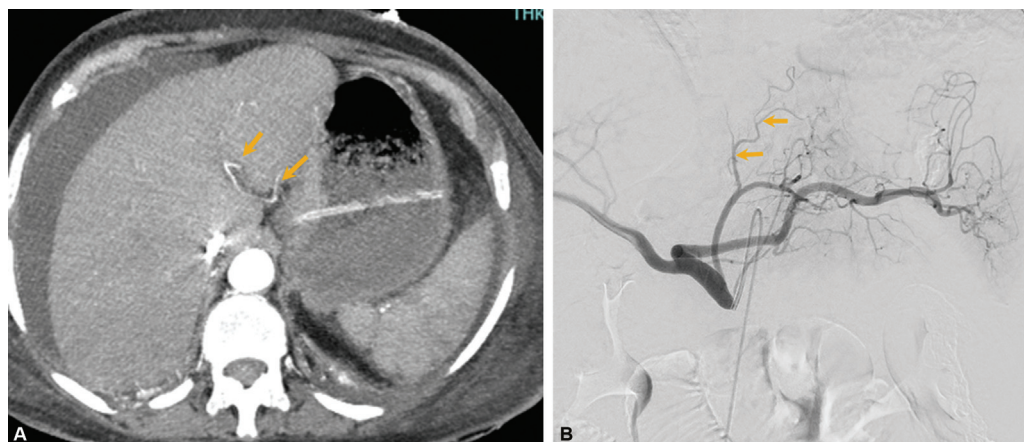


Fig. 8 Replaced left hepatic artery in a 55-year-old male patient; (A) Axial maximum intensity projection computed tomography angiography image at the level of gastrohepatic ligament shows replaced left hepatic artery coursing through the ligamentum venosum (yellow arrows). (B) Selective angiography of celiac axis demonstrates replaced left hepatic artery arising directly from left gastric artery.

parenchymal distribution may be confirmed on a cone-beam CT scan.²⁷

CTA has a high sensitivity (85%) and specificity (92%) for detecting bleeding sites. CTA can detect at least 0.1 to 0.5 mL/min bleeding rate, whereas DSA is less sensitive and detects a minimum of 0.5 mL/min bleeding rates.²⁸

Less often, arterial vasospasm becomes a challenge to access distal branches and can conceal the active bleeding sites; however, this caveat may be overcome by a wait and watch strategy or use of intra-arterial 200 µg nitroglycerin into the culprit vessel. This technique of injecting intraarterial nitroglycerine is also effective in challenging cases to demonstrate bleeding.²⁹ Provocative mesenteric angiography techniques has also shown promising role in detection of occult and obscure bleeding sites. Kohanteb et al has used heparin (5000 units), TPA (mean: 18.9 mg), and nitroglycerin (mean: 263 µg) for the localization and detection of GIB.²⁹

Embolization Techniques

Achieving hemostasis at the bleeding vessel remains the main objective for a successful embolization procedure. Anatomical complexities and bleeding artery hemodynamics greatly influence the embolization approach and embolic material delivery.

1. Superselective/localized embolization

Meticulous planning with prior CTA, experience, and efforts of interventional radiologist are of utmost importance to catheterize the bleeding point and embolic material delivery. Superselective embolization helps significantly in reducing rebleeding or ischemic events³⁰ (►Fig. 9).

A recent study by Kodani M et al showed that superselective embolization of 1 vasa rectum in lower GIB reduces ischemic events and shortens hospitalization. Surgical intervention for ischemic complications is more frequently required if more than or equal to 2 vasa recta are embolized³⁰ Long segment (3–4 cm) of marginal artery can be embolized if superselective catheterization of the vasa recta cannot be achieved. It reduces the perfusion to a bleeding point, but rebleeding tendencies remain high due to collaterals. Rebleeding secondary to extensive collaterals can also be minimized by superselective catheterization in UGIB.³¹

2. Sandwich embolization

This technique is used for cases in which there is possibility of filling of the bleeding vessel in retrograde manner and cases with pseudoaneurysm. To prevent the rupture of the pseudoaneurysm and its refilling, it is imperative to embolize the proximal and distal doors of the pseudoaneurysm and any active bleeding site, also known as “front door/back door packing.” (►Fig. 10). Check angiogram should be performed to demonstrate any back door filling of pseudoaneurysms or active site of bleeding.^{32,33}

3. Segmental embolization

A segment of an artery can be embolized if there are multiple tiny arterial bleeding points that cannot be cath-

eterized individually or in cases of profuse mucosal inflammatory bleeding. Segmental embolization results in hypoperfusion and may induce clotting. However, rebleeding may occur due to rich collateral supply or deranged coagulation³⁴ (►Fig. 11).

4. Proximal embolization

Proximal embolization with liquid/particulate agents is also feasible in patients with complex arterial anatomy, failed superselective catheterization, or arteriovenous malformation. Liquid embolic material may percolate deep to arteriolar or capillary levels depending on the concentration of the embolic material³⁴ (►Fig. 12).

Embolic Materials

Choice and selection of appropriate embolic agents is of paramount importance since it depends on various factors like need of permanent or temporary embolization, chances of rebleeding, visceral ischemia, desired level of embolization, etiology of GIB, radiologist's experience, availability of embolic materials, and site and size of the bleeding artery.^{35–37}

A wide range of embolic materials are available commercially; however, the selection and choice of different embolic materials do not have any consensus/guidelines and are still debatable. The possible reason may be attributed to variable safety profile and delivery techniques.³⁵ (►Table 1)⁴

Factors Affecting Selections of Embolic Materials

1. Anatomical level of embolization and GIB etiology

Underlying bleeding etiology (inflammatory, tumor, vascular) and level of vascular anatomy (artery, arterioles, capillary) usually determine the selection of embolic agents. Inflammatory or tumor bleeding usually requires coil embolization if bleeding arteries can be easily accessed and targeted, while in patients with profuse or multiple discrete bleeding sites can be managed by a combination of coils, particles, and liquid agents. Vascular etiologies like arteriovenous malformation need closure of nidus by liquid agents.^{35–37} Desired levels of arterial anatomical embolization and their selective embolic agent of the first choice have been shown in the illustration (►Fig. 13).

2. Permanent vs. temporary agents

Gelfoam (Pfizer, New York, United States) is a cheap, temporary, compressed form of bio-absorbable embolic material that can be used in the form of particles, scrapings, slurry, and mixed with saline and contrast for its delivery. Gelfoam is not used alone in GIB. Few studies suggest that it may be used in combination with coils as “A Gelfoam sandwich” technique to expedite the clotting within the fibrin scaffolding of the coil. A major drawback of gelfoam embolization is its uncontrolled distal migration/regurgitation into nontargeted vessels. This

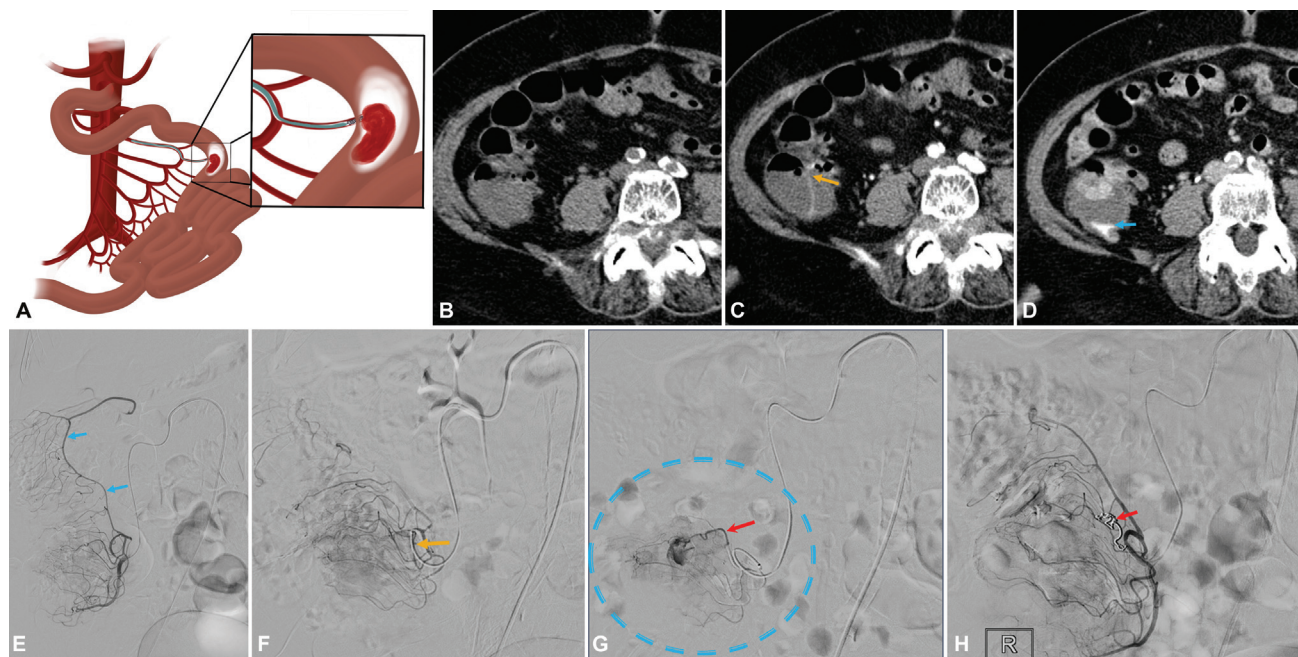


Fig. 9 Superselective/localized embolization (illustration A) and its importance; (B) noncontrast, (C) arterial and (D) venous phase axial computed tomographic images in a 58-year-old female at the level of cecum reveal active arterial contrast extravasation (yellow arrows) and pooling of contrast within cecum (blue arrow). Ileocolic artery superselective angiography; (E) ileocolic artery proximal to marginal artery (blue arrows) (F) distal to the marginal artery (yellow arrows). Note that embolization proximal to the marginal artery may cause rebleeding; however, nonsuperselective embolization beyond the marginal artery may lead to ischemia; (G) and (H) pre- and post-superselective successful embolization at vasa recta level (red arrows) without ischemia.

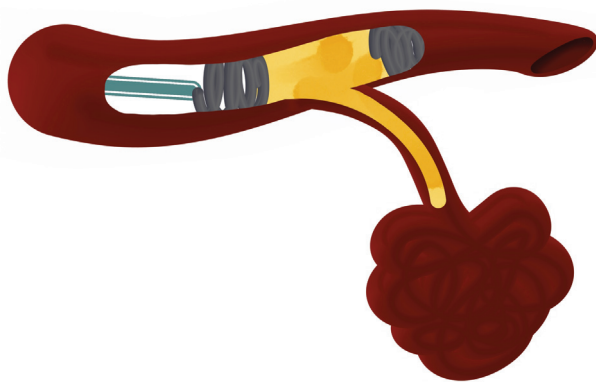


Fig. 10 Sandwich embolization: an illustration shows a pseudoaneurysm with superselective coil embolization of feeding artery proximal and distal to pseudoaneurysm.

may occlude submucosal arteriolar plexus beyond vasa recta level and ultimately lead to increased bowel ischemia.^{38–41} However, recanalization of a vessel after gelfoam embolization may happen within days to weeks, which also remains a concern for rebleeding.^{36,42–44} GI tumors with profuse surface bleeding may be appropriate candidates for gelfoam embolization if surgery is contemplated within 24 to 48 hours of the procedure.^{45–48}

Metallic coils and polyvinyl alcohol particles (PVA) are the most commonly used permanent embolic agents of choice in GIB. Metallic coils are available in various configurations and morphology. The fibrin component of coils triggers the thrombotic process within the lumen. Coils should be deployed as much as possible to reduce recurrent bleeding. It preserves the microcirculation of the GI tract and thus reduces the risk of ischemia. Certain clinical situations



Fig. 11 Segmental embolization: (A) Illustration depicts the segmental embolization of an artery. (B) Superselective catheterization of gastroduodenal artery (GDA) in a 67-year-old female patient with a history of peptic ulcer disease shows abnormal active contrast extravasation (red arrow) without any obvious feeding artery (C) Postsegmental GDA embolization by multiple metallic coils.

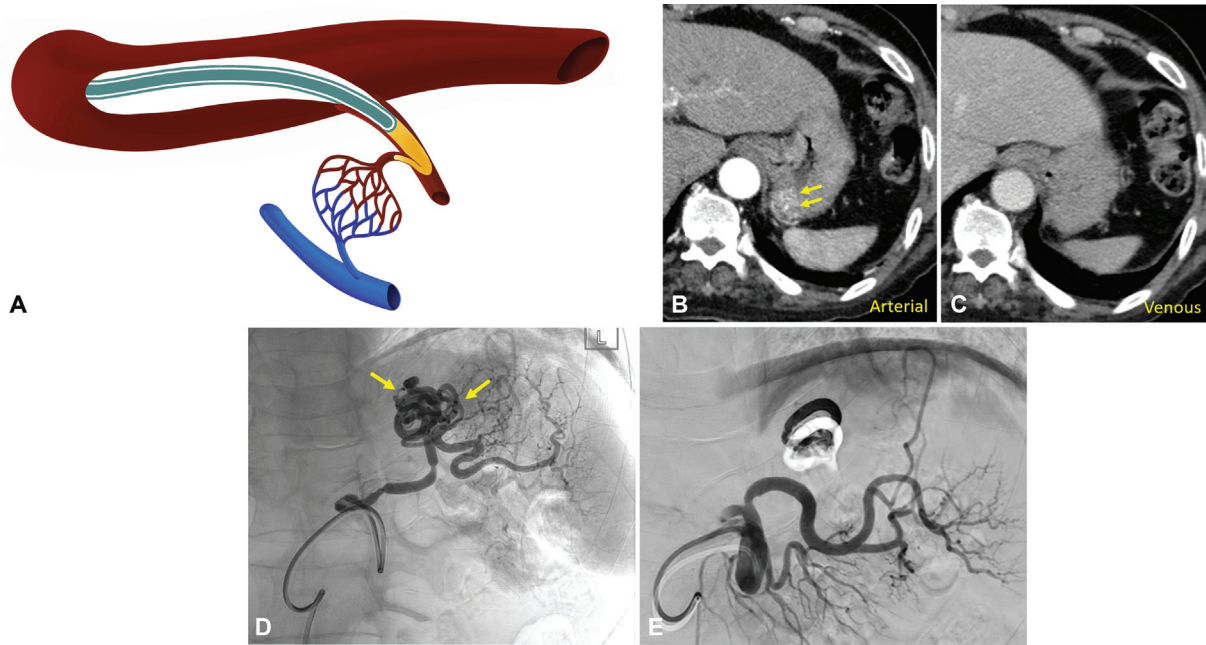


Fig. 12 Proximal embolization shown in illustration (A); proximal embolization of vascular malformation (“Dieulafoy’s lesion”) in a 71-year-old male patient with a history of recurrent upper gastrointestinal bleed; (B) arterial and (C) venous phase axial computed tomography angiography (CTA) at gastric fundus level shows multiple intramural tortuous abnormal arterial vascular channels without active arterial extravasation (arrows). (D) Superselective catheterization of left gastric artery confirms the CT findings. (E) Complete occlusion of vascular malformation after proximal embolization of left gastric artery by liquid embolic agents.

Table 1 Various embolic agents and their mechanism of action

Duration of embolization	Category	Agents	Mechanism of action
Temporary		Gelfoam (pledgets/particles), autologous clot, oxidized cellulose, microfibrillar collagen.	Mechanical occlusion
Permanent	Mechanical devices	Coils (pushable, detachable), Detachable balloon and plugs	Mechanical occlusion + fibrin induced thrombotic reaction
	Particles	Polyvinyl alcohol particles (PVA). embospheres	Mechanical occlusion + endothelial damage-coagulative necrosis-inflammation and fibrosis of the vessel wall
	Liquid polymers	N-Butyl cyanoacrylates (glue), ethylene vinyl alcohol copolymer (Onyx)	Polymerization and cast formation
	Liquid sclerosants	Absolute alcohol, sodium tetra-decyl sulphate (STS)	Endothelial damage-coagulative necrosis-inflammation of the vessel wall

like old age, massive blood loss, cirrhosis, and coagulopathy have been associated with recurrent bleeding after metallic coils embolization. In such clinical situations, gelfoam/PVA particles can also be used with metallic coils to expedite clotting.^{42,43}

N-butyl 2-cyanoacrylate (NBCA) glue has shown its significance and desirable results for GIB in many recent studies.^{37,48–52} It has several advantages like low cost, ability to occlude distal vasculature up to arteriole or capillary level beyond the reach of microcatheter, can be delivered by ultra-

microcatheter that is unsuitable for microcoils, excellent obliteration of bleeding points in complex anatomy and obliteration of vascular malformations.³⁵

NBCA should be used cautiously as it has a low safety profile and steep learning curve. It may polymerize quickly at the catheter tip and may adhere to the vessel wall. Prompt removal of catheter and immediate aspiration after the NBCA delivery is advisable for the same reason. Moreover, its distal uncontrolled nontargeted embolization and reflux may lead to bowel ischemia.⁴⁸ The rate of rebleeding after NBCA is

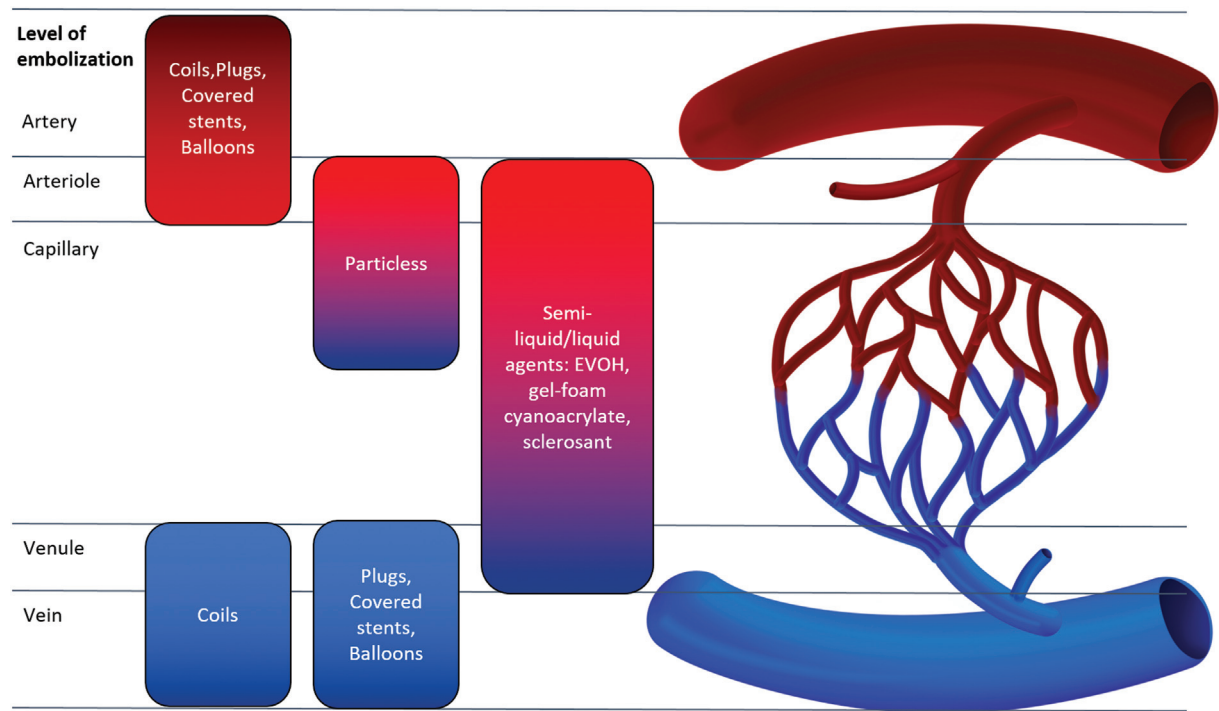


Fig. 13 Desired level of embolization and choice of embolic agent.

relatively lower (4–15%) as compared to that of coils or particles (0–26%).^{48–51}

Another relatively costly newer liquid agent is **Onyx (Micro Therapeutics, Inc., Irvine, California, United States)**, prepared with a mixture of ethylene-vinyl alcohol copolymer and dimethyl sulphoxide (DMSO) as solvent. It has a high degree of safety profile compared to NBCA as it does not adhere to vessel walls and takes longer time for solidification, resulting in a more predictable range of embolization. DMSO has certain disadvantages, including a requirement of DMSO-compatible catheters and excretion through sweat/respiration, which leaves diabetic ketoacidosis-like body odor for a few days.³⁵ The data on rebleeding rate after onyx embolization is limited and the available published meta-analysis has reported approximately 7.6% rebleeding rate.³⁶

Liquid sclerosant is not widely used in GIB. These agents are commonly used for variceal bleeding with endoscopic sclerotherapy.^{53,54} Glubran 2 is a synthetic surgical biodegradable cyanoacrylate-based glue, modified by the addition of a monomer into NBCA [Glubran has two components, NBCA + Metacrilisolfolane (MS)]; it is another potential liquid agent similar to NBCA that is equally safe and fast embolic material with high clinical success rates, ranging from 96 to 100% and rebleeding rate of 7.7%.⁵⁵

Outcome and Rebleeding

A summary of publications on GIB embolization shows that there is continuous improvement in technical success rates, which may attribute to preprocedure CTA planning, newer

microcatheters, and increased use of NBCA due to high clinical success rates (70–88%) and low rebleeding rates (4–15%) in comparison to other embolic agents.^{48–51,55} However, clinical success rates remain variable in literature due to variable underlying etiology and disease severity; for example, patients with malignant tumor bleed have shown low clinical success.³⁷

There was no significant difference between UGIB or LGIB-related success rates and mortality rates. One study has shown that²¹ clinical success rates were higher with diverticular disease-related bleeding than other etiologies.

Rebleeding rates 0 to 47% and mortality rates are reported in 3 to 29% of patients, while surgery surgery rates due to rebleeding (5–16%) are only reported in a few studies. The most common factor influencing rebleeding was coagulopathy; others include multisystem organ failure, massive blood transfusion, delayed embolization, and malignant tumor bleeding.^{1,7,15} Jeong et al¹⁸ have shown a significant reduction in rebleeding after using NBCA compared with other embolic agents since NBCA does not depend on coagulopathy¹⁵ (► **Table 2**).

Factors affecting mortality rates were multifactorial like massive blood transfusion, associated comorbidities, advanced age, rescue surgeries, cirrhosis, multiorgan failure, and malignancy patients. Overall survival rates were well correlated with clinical/technical failure, rebleeding, and major complications¹

Most studies have minor complications like access site hematoma, pseudoaneurysms, arterial dissection, or coil migration.^{9,11,15} Major complications include ischemia requiring surgeries. Few major anecdotal complications like

Table 2 Summary of recent publications on upper and lower GI embolization technical success, rebleeding, and mortality rates

Year	Author	Upper or lower GI etiology	Clinical success rate	Technical success rate	Rebleeding	Complication rates	Surgery rate	Mortality follow-up	Embolic material used
2004	Ripoll et al ⁵³	Upper GI			29		16.1	25.8	
2005	Khanna et al ⁵⁶	Lower GI		85	40				
2008	Larssen et al ⁵⁷	Upper GI	72	92	9			19	
2008	Eriksson et al ⁵⁸	Upper GI						3	
2010	Venclauskas et al ⁵⁹	Upper GI						20.8	
2011	Wong et al ⁶⁰	Upper GI		88.5	34.4			25	
2012	Jairath et al ⁵⁴	Upper GI			9		9	10	
2012	Ang et al ⁶¹	Various		80	46.7				
2012	Katano et al ⁶²	Both		80	0				
2013	Yap et al ⁶³	Both		98	23	4 ischemia, 3% coil migration		18	coils
2013	Teng et al ⁶⁴	Lower		84.6	7.7	7.7		19.2	
2015	Lee et al ⁶⁵	Upper GI		96.8	47	Hepatic artery dissection		16.7	
2015	Tanveer-Ul-Haq et al ⁶⁶	All	92	100	3	3 ischemia			
2017	Nykänen et al ⁶⁷	Upper		100	25	37.5		12.5	
2018	Lee et al ⁶⁸	Lower		100	27			29	
2018	Kwon and Han ⁶⁹	Both	72.2	100	16.7	16.7		16.7	NBCA
2018	Kwon and Han ⁶⁹	Both	68.2	100	22.7	16.7		32	Coils, particle, gelfoam
2018	Nykänen et al ⁷⁰	Lower		96	14	36		6	
2018	Senadeera et al ⁷¹	Lower	81	97	19	5.2 bowel ischemia	5.2	6.3	Ccoils +variable
2019	Muhammad et al ⁷²	Both	71.9	96.9	3	10 coil migration 3 access site hematoma		21.9	Coils=gelfoam

Abbreviation: GI, gastrointestinal.

septic shock, myocardial infarction, and death due to ischemia have also been reported.^{15,19}

Conclusion

CTA has been now accepted as quick, accurate, and robust imaging not only for diagnosing, but also helps in treatment planning and in most cases defines the etiology of GIB. The constant evolving advancement in the field of interventional radiology hardware and techniques has further improved success rate with minimal complication and better clinical outcome.

Ethical Approval Statement

Institutional review board (Medical ethical committee) approval was exempted.

Conflict of Interest

None declared.

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