



**GASTROINTESTINAL BLEEDING: ENDOSCOPIC AND SURGICAL APPROACHES
AND THE RISK OF REBLEEDING**

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ABSTRACT

Gastrointestinal bleeding (GIB) is one of the most frequent, time-critical clinical syndromes in emergency medicine, gastroenterology, and surgery. The bleeding source may be upper (esophagus–stomach–duodenum) or lower (small/large bowel); the clinical presentation (hematemesis, melena, hematochezia), hemodynamic status, and comorbidities determine the management strategy. Contemporary care integrates early resuscitation and risk stratification with timely endoscopy; interventional radiology (CT angiography and selective embolization) and surgery serve as escalation options when endoscopic control is inadequate. This article synthesizes evidence-based principles for stepwise management of GIB, selection of endoscopic hemostasis techniques, practical differences between variceal and non-variceal bleeding, indications for surgical intervention, and strategies for stratifying and preventing rebleeding.

Keywords:gastrointestinal bleeding, UGIB, LGIB, variceal bleeding, NVUGIB, endoscopic hemostasis, clip, thermal coagulation, OTSC, band ligation, CT angiography, embolization, surgery, rebleeding, Forrest classification, PPI.

INTRODUCTION

Gastrointestinal bleeding is a common condition with substantial resource utilization and heterogeneous etiologies, including peptic ulcer disease, erosive gastritis/duodenitis, Mallory–Weiss tears, angiodysplasia, diverticular hemorrhage, neoplasms, inflammatory bowel disease, and portal-hypertension-related variceal bleeding. In many healthcare systems, GIB is among the leading reasons for hospital admission, and professional society guidance (e.g., ACG) emphasizes its burden on emergency departments and inpatient services.

In the emergency setting, the first priority is to correct life-threatening hemodynamic disturbances even before the bleeding source is definitively identified. A practical approach begins with ABCDE assessment (airway, breathing, circulation, neurological status, and full examination). At least two large-bore intravenous lines are established, crystalloid fluids are initiated, and key laboratory tests are obtained (hemoglobin/hematocrit, platelets, INR/aPTT, creatinine, liver enzymes, lactate). The ACG 2021 UGIB guideline supports a restrictive transfusion strategy commonly anchored around a hemoglobin threshold of 7 g/dL, while recognizing the need for individualized decisions in myocardial ischemia, severe hypoxemia, or ongoing shock. The goal of this phase is to restore perfusion and enable safe transition to diagnostic and therapeutic steps such as endoscopy or CT imaging.

The second core issue is risk stratification. In upper GIB, scoring tools such as the Glasgow-Blatchford Score (GBS) can identify low-risk patients who may be managed with outpatient follow-up and expedited specialist consultation; the ACG guideline notes that selected patients with very low risk (e.g., GBS 0–1) may be managed without hospital admission. Risk stratification influences not



only admission decisions but also endoscopy timing (urgent vs early), ICU needs, and anticipated probability of rebleeding.

Endoscopy is both diagnostic and therapeutic in GIB. In non-variceal upper GIB (NVUGIB), endoscopic stigmata (Forrest classification) correlate with rebleeding, need for surgery, and mortality. Active spurting (Ia) or oozing (Ib) and a visible vessel (IIa) are considered high risk; an adherent clot (IIb) indicates intermediate risk; a flat pigmented spot (IIc) and a clean base (III) are low risk. In lower GIB, common sources include diverticular bleeding, angiodysplasia, colitis, neoplasms, and anorectal pathology (hemorrhoids, fissures). Because presentations can overlap, rapid clinical localization (e.g., melena is more often upper; massive upper bleeding may also present with hematochezia) and correct sequencing of diagnostic steps are crucial.

The clinical relevance of this topic is tightly linked to rebleeding. Even after apparent hemostasis, ulcer pathophysiology (H. pylori, NSAIDs, steroids), antiplatelet/anticoagulant therapy, coagulopathy, comorbidities, and high-risk endoscopic stigmata can precipitate recurrent hemorrhage. Rebleeding prolongs hospitalization, increases transfusion requirements, raises ICU utilization, and elevates the likelihood of embolization or surgery. Accordingly, this article presents a practical chain: stabilization → endoscopic hemostasis → prevention of rebleeding → escalation.

METHODS

Methods. This work is an evidence-informed narrative review. Sources were selected using three criteria: (1) authoritative clinical guidelines and consensus statements (ACG 2021 UGIB, ESGE 2021 NVUGIB, ACG 2023 LGIB); (2) classical foundations for rebleeding risk assessment (Forrest classification and prognostic tables); (3) high-reliability reviews and educational resources addressing endoscopic hemostasis, interventional radiology, and surgical escalation. The synthesis was organized around the ‘bleeding management pathway’: initial resuscitation and medication/comorbidity assessment; endoscopic localization and hemostasis; CT angiography and selective embolization if endoscopy fails or is insufficient; and surgical management when required. Findings are presented as decision points supported by conceptual figures and summary tables. The aim is not to create a local protocol, but to provide an actionable ‘decision map’ for clinicians.

RESULTS

Results. Evidence synthesis consolidated GIB management into three actionable domains: (A) optimal selection of endoscopic hemostasis, (B) the role of interventional radiology and surgery, and (C) stratification and prevention of rebleeding.

A) Endoscopic approaches (diagnosis + hemostasis). Endoscopy is effectively ‘dual-purpose’: it identifies the bleeding source and provides definitive therapy. In NVUGIB, stigmata guide therapy: high-risk lesions require endoscopic treatment, whereas low-risk stigmata often do not warrant aggressive intervention. ESGE 2021 emphasizes that epinephrine injection is typically not definitive as monotherapy; instead, it is used as an adjunct (‘bridge’) to mechanical or thermal therapy. Practically, clinicians often first improve visualization and reduce active bleeding, then secure durable hemostasis.

Injection therapy: epinephrine 1:10,000 is injected in small aliquots around the lesion, producing vasoconstriction and a tamponade effect. Its primary role is to reduce active bleeding and facilitate clip placement or thermal contact. Sclerosants and tissue adhesives may be used in selected circumstances, but their adverse-event profile (necrosis, thrombosis) requires careful case selection.

Mechanical therapy: through-the-scope (TTS) clips are widely used for visible vessels, Dieulafoy lesions, and diverticular bleeding. The key advantage is mechanical vessel closure without thermal injury. Over-the-scope clips (OTSC) provide stronger compression and may be useful in



fibrotic ulcer bases or as rescue therapy after standard clips/thermal methods fail. In variceal bleeding, the principal mechanical method is band ligation, which is first-line for esophageal varices.

Thermal therapy: bipolar coagulation, heater probes, and argon plasma coagulation (APC) are commonly used. Thermal methods are effective in angiodysplasia and bleeding stigmata, but energy dosing must be controlled to reduce deep tissue injury and perforation risk. APC is non-contact and can be advantageous for superficial vascular lesions. Topical therapy: hemostatic powders and spray/gel systems can provide rapid control in diffuse or multifocal bleeding and in complex cases under anticoagulation. Their practical value is speed and the ability to stabilize the situation; however, they may serve as a temporary solution until definitive etiologic control or planned re-intervention. Variceal bleeding warrants explicit mention. In portal hypertension, ruptured esophageal varices are managed with endoscopic band ligation plus pharmacologic therapy (vasoactive agents) and infection prophylaxis. If bleeding persists, balloon tamponade or esophageal stents can function as bridge therapies, while definitive options such as TIPS may be considered. Although this paper does not provide a full variceal protocol, the ‘endoscopy + escalation’ logic remains applicable.

In lower GIB, colonoscopy enables source identification and endoscopic therapy. Diverticular bleeding is commonly treated with clipping; angiodysplasia often with thermal therapy; post-polypectomy bleeding with clips or coagulation; and suspected tumors require biopsy and oncologic referral. The ACG 2023 guideline highlights that after resuscitation and clinical evaluation, CT angiography should be considered when the source is not identified or bleeding is ongoing, followed by endoscopic or radiologic hemostasis. B) When endoscopy is insufficient: interventional radiology and surgery (escalation). If bleeding is uncontrolled or rebleeding risk remains high, CT angiography can localize active hemorrhage and direct angiographic embolization. Selective embolization provides hemostasis without laparotomy, particularly when minimal hemodynamic stability is achieved and localization is adequate. Surgery is considered when: (1) endoscopic and radiologic methods fail or are unavailable; (2) massive ongoing bleeding persists; (3) anatomical indications exist (tumor, perforation, necrosis); (4) the source is clear and segmental resection is expected to be beneficial. In upper GI ulcer bleeding, operations range from vessel ligation/oversewing to selective resection; in lower GI bleeding, options may include segmental colectomy or subtotal colectomy when the source is unclear. Although decisions depend on local resources, delays can worsen outcomes; thus, ‘escalation readiness’ should be continuous. C) Rebleeding risk: prognosis and prevention. Rebleeding has two layers: endoscopic stigmata and clinical context. By Forrest classification, active spurting/oozing and visible vessels carry high risk, while low-risk stigmata carry minimal risk. Prognostic tables consistently show higher rebleeding rates in Forrest Ia–Ib lesions and substantially lower rates in Forrest III lesions. Clinical variables act as amplifiers: hemodynamic instability, large ulcer size, depth of anemia, anticoagulants, renal/hepatic failure, older age, and previous bleeding. Prevention strategies fall into three phases. First, post-endoscopic pharmacologic reinforcement: in NVUGIB, PPI therapy in high-risk lesions reduces rebleeding risk. Second, etiologic control: *H. pylori* testing and eradication, discontinuation of NSAIDs or selection of safer alternatives, and planning for resumption of antiplatelet/anticoagulant therapy based on a risk–benefit balance. Third, monitoring and reassessment: trends in vital signs and hemoglobin, recurrent melena, or renewed hematemesis/hematochezia should trigger repeat endoscopy or escalation to IR/surgery.

To visualize the pathway, Figures 1–4 and Tables 1–3 are provided: Figure 1 outlines the ED-to-endoscopy algorithm; Figure 2 summarizes hemostasis techniques; Figure 3 provides a simplified rebleeding matrix; Figure 4 presents an escalation ladder; Table 3 provides simplified rebleeding probability ranges by Forrest class.

Figure 1. Conceptual management algorithm from the emergency department to endoscopy

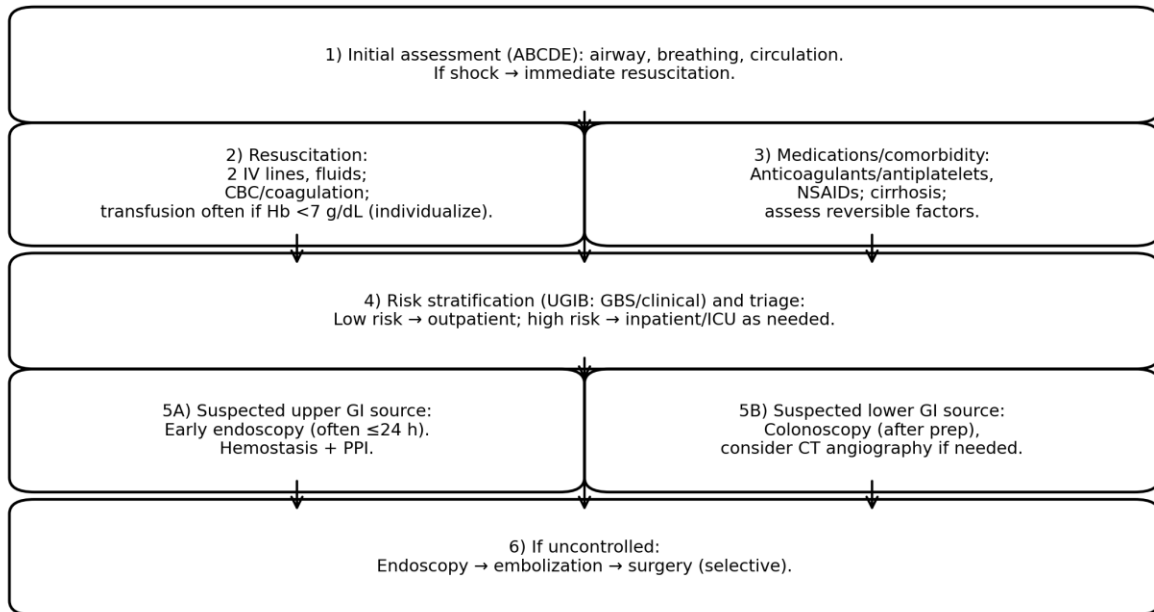


Figure 2. Endoscopic hemostasis toolbox (conceptual)

Endoscopic hemostasis toolbox (conceptual):

Injection	Mechanical	Thermal	Topical / Combination
Epinephrine 1:10,000 (rarely as monotherapy) Selected sclerosants	TTS clips OTSC Band ligation (varices) Endoscopic suturing (selected)	Bipolar coagulation Heater probe APC Other energy-based methods	Hemostatic powders Sprays/gels Combination: Epinephrine + clip/thermal

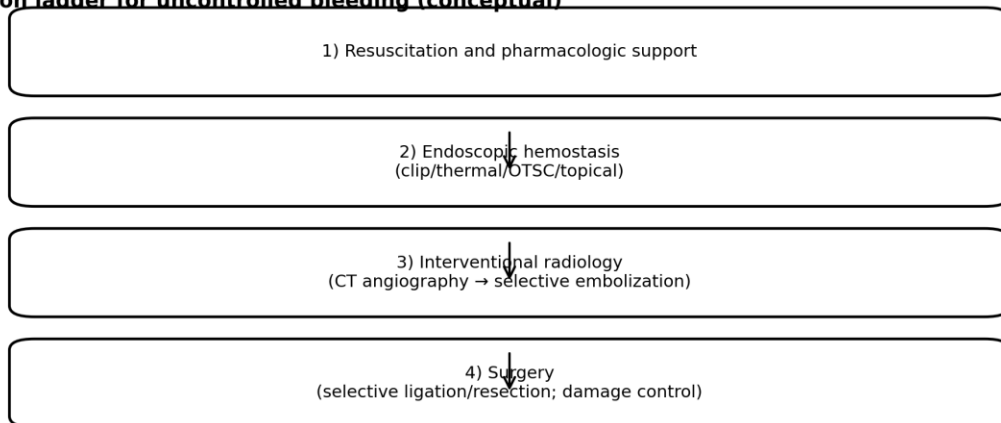
Note: In NVUGIB, epinephrine is usually adjunctive; definitive hemostasis is reinforced with mechanical or thermal therapy.

Figure 3. Rebleeding risk matrix: stigmata and clinical factors (simplified)

	Low clinical risk	Intermediate clinical risk	High clinical risk
High-risk stigmata (Forrest Ia-IIa)	Outpatient follow-up	Outpatient + optimize risk factors	Inpatient work-up guided by etiology
Intermediate (IIb)	PPI + monitoring	Monitoring + repeat endoscopy as indicated	Monitoring + IR/2nd endoscopy ready
Low-risk (IIc-III)	Endoscopy + PPI	Endoscopy + close monitoring	ICU + plan early re-intervention

Figure 4. Escalation ladder for uncontrolled bleeding (conceptual)

Escalation ladder for uncontrolled bleeding (conceptual)



Selection depends on: source, resources, stability, rebleeding risk, endoscopic success.

Table 1. Endoscopic hemostasis methods: indications and practical notes

Method	Typical use	Advantages	Limitations/risks
Epinephrine injection	Diffuse oozing; improves visualization; in combination	Fast; clears the field	Not durable as monotherapy; temporary effect
Clip (TTS)	Visible vessel; Dieulafoy lesion; diverticular bleeding	Mechanical closure; minimal thermal injury	Lower success in difficult positions
OTSC	Fibrotic base; failure of clips/thermal methods	Strong compression; durable hemostasis	Cost; training; availability



Band ligation	Esophageal varices	Rapid and effective; lowers recurrence	Limited view in active bleeding; may require bridge escalation
Thermal coagulation	High-risk stigmata; angiodysplasia	Widely available; effective	Risk of deep injury/perforation; dosing required
Topical powder	Multifocal/diffuse bleeding; 'bridge' therapy	Rapid; non-contact	Rebleeding possible; follow-up plan needed

Table 2. Rebleeding risk factors and prevention strategies

Risk factor	Mechanism/notes	Prevention/decision
Forrest Ia–IIa	Open/recently bleeding vessel	Hemostasis + close monitoring + PPI regimen
Anticoagulants/antiplatelets	Impaired coagulation	Risk–benefit review; reversal when indicated; resumption plan
NSAIDs	Reduced mucosal protection	Stop NSAID; choose alternatives + gastroprotection
H. pylori	Ulcer recurrence and bleeding risk	Test + eradication; confirm eradication
Shock/large ulcer/comorbidity	Hypoperfusion and vessel exposure	Resuscitation; early endoscopy; escalation readiness

Table 3. Estimated rebleeding probability by Forrest class (simplified ranges)

Forrest class	Endoscopic appearance	Rebleeding (approx.)
Ia	Active spurting bleeding	55–100%
Ib	Active oozing	≈35%
IIa	Visible vessel	40–50%
IIb	Adherent clot	20–30%
IIc	Flat pigmented spot	≈10%
III	Clean base	≈5%

Note: ranges are simplified and reflect typical values reported in prognostic tables and clinical teaching resources.

DISCUSSION

Discussion. A frequent clinical error in GIB is prioritizing source hunting over resuscitation. In practice, stabilization precedes definitive diagnostics and hemostasis. This does not imply that endoscopy should proceed regardless of deterioration; rather, a minimum level of perfusion and airway safety should be ensured to make endoscopy safe. In severe hematemesis, aspiration risk is substantial and the need for intubation must be assessed individually.

Timing of endoscopy matters. ESGE 2021 notes that very urgent endoscopy (≤ 6 hours) in NVUGIB does not consistently improve outcomes and may be harmful in certain settings; in many cases, 'early' endoscopy within 24 hours represents a reasonable balance. ACG 2021 emphasizes



using risk stratification to determine which patients require earlier endoscopy and which may be managed as outpatients.

Regarding technique selection, the practical logic is ‘strong and precise’. In visible-vessel ulcers, temporary bleeding control with epinephrine injection followed by clipping or thermal coagulation yields more durable hemostasis. APC is often optimal for angiodysplasia; Dieulafoy lesions may be managed with clips or banding; post-polypectomy bleeding commonly responds to clips, coagulation, or endoloops. In variceal bleeding, band ligation is first-line and, when combined with pharmacologic measures and prophylaxis, reduces rebleeding and supports improved outcomes

When endoscopy fails, IR and surgery should be viewed not as optional alternatives but as integral components of a complete system. Embolization is selective and minimally invasive, but vascular anatomy, collateral flow, and ischemic complications must be considered. Surgical goals are definitive source control (ligation/resection) and reversal of shock physiology. In unclear sources, extensive resection may be overly traumatic; therefore, diagnostic precision (endoscopy/CT angiography) should be maximized whenever feasible.

Rebleeding prevention can be treated as a quality indicator. In high-risk stigmata, monitoring intensity should be higher, PPI regimens should be strictly implemented, etiologic factors (H. pylori, NSAIDs) corrected promptly, and antithrombotic resumption should follow a coordinated plan with cardiology/neurology as appropriate. In low-risk stigmata, unnecessary repeat endoscopy and prolonged admission can often be avoided, allowing resources to be focused on higher-risk patients.

Limitations. This is a narrative review rather than an original randomized study. Availability of OTSC, hemostatic powders, CT angiography, and angiography suites may be limited in some settings; implementation may benefit from defining a resource-adapted ‘minimal package’ versus an ‘ideal package’.

CONCLUSION

Conclusion. Effective management of gastrointestinal bleeding requires an integrated pathway: stabilization → risk stratification → endoscopic hemostasis → rebleeding prevention → escalation (interventional radiology/surgery). In NVUGIB, selecting appropriate mechanical or thermal therapy according to Forrest stigmata and reinforcing post-procedure care with PPIs reduces rebleeding; in variceal bleeding, band ligation and bridge escalation options are critical. In lower GIB, colonoscopy and, when needed, CT angiography are central for localization and planning hemostasis. Preparing escalation options in parallel, systematically assessing rebleeding risk, and addressing etiologic drivers improve clinical outcomes.

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