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Viscoelastic Testing in Acute Lower Gastrointestinal Bleeding: Current Management, Evidence, and Unresolved Questions

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1. Abstract

1.1. Background

Acute lower gastrointestinal bleeding (LGIB) can present as self-limited haematochezia or as life-threatening haemorrhage that requires rapid localization and source control. Conventional coagulation tests assess isolated aspects of haemostasis, while thromboelastographic (TEG) and rotational thermoelectrometry (ROTEM) offer dynamic, whole-blood evaluation of clot initiation, propagation, strength, and breakdown.

1.2. Objective

To review current LGIB management, evaluate studies on viscoelastic testing, and assess whether evidence supports TEG-guided care in LGIB.

1.3. Findings

No randomized trial has tested a prespecified TEG- or ROTEM-guided transfusion algorithm exclusively in confirmed LGIB cases. The strongest randomized evidence is from patients with cirrhosis and upper gastrointestinal bleeding, where TEG-guided strategies reduced plasma and platelet use without affecting initial haemostasis. In contrast, retrospective mixed-GI cohorts found that patients undergoing TEG received more blood products, without consistent improvements in length of stay or other patient-centred outcomes. These differences likely reflect variations in patient populations, protocol adherence, timing, transfusion thresholds, and confounding by indication, rather than a direct effect of the assay.

1.4. Conclusion

Current evidence is insufficient to support routine TEG- or ROTEM-guided management of LGIB. The most defensible potential role for viscoelastic testing is as a targeted adjunct during severe ongoing haemorrhage when a specific non-red-cell component decision remains uncertain. Prospective LGIB-specific studies are needed to evaluate a complete viscoelastic-test-linked treatment pathway.

2. Introduction

Acute LGIB is a syndrome, not a single disease. Common causes include diverticular haemorrhage, Angio ectasia, ischemic and inflammatory colitis, post-polypectomy bleeding, colorectal neoplasia, anorectal disease, and small-bowel lesions. While many episodes resolve spontaneously, a significant minority result in shock, recurrent bleeding, transfusion dependence, or require endoscopic, radiologic, or surgical intervention [1-3]. Severe haematochezia may also originate above the ligament of Treitz, so an unstable patient should still be evaluated for an upper source.

The primary therapeutic challenge in LGIB is anatomical: identifying and controlling a bleeding vessel or mucosal lesion. This contrasts with trauma, cardiac surgery, liver transplantation, and some cirrhotic bleeding states, where systemic coagulopathy often drives haemorrhage. This distinction is important because an abnormal coagulation assay cannot replace computed tomography angiography (CTA), colonoscopy, transcatheter arterial embolization, or surgery. Nevertheless, haemostatic assessment may be complicated by antithrombotic exposure, massive blood loss, haemodilution, hypothermia, or liver disease. A substantial proportion of patients with LGIB receive anticoagulant or antiplatelet therapy for conditions such as atrial fibrillation, venous thromboembolism, coronary artery disease, or cerebrovascular disease. Conventional coagulation tests may not fully characterize clot function in these settings; however, standard viscoelastic assays also may not reliably detect clinically important direct oral anticoagulant or antiplatelet effects.

Viscoelastic haemostatic assays are valuable because they assess clot formation and lysis in whole blood, providing actionable bedside information. However, the term "TEG in gastrointestinal bleeding" often combines three distinct evidence domains: cirrhot-

ic upper-GI bleeding, mixed-source gastrointestinal haemorrhage, and true LGIB. This review distinguishes these domains, places older studies in context, and examines what can and cannot be inferred for current LGIB practice.

3. Review Approach

This narrative review evaluated society guidelines, randomized trials, systematic reviews, and observational studies available through July 2026. Targeted PubMed/MEDLINE searches were supplemented by citation tracing from relevant guidelines, reviews, and included studies. Search terms included lower gastrointestinal bleeding, haematochezia, gastrointestinal haemorrhage, thromboelastographic, rotational thermoelectrometry, viscoelastic testing, transfusion, plasma, platelets, fibrinogen, anticoagulants, antiplatelet agents, direct oral anticoagulants, reversal, colonoscopy, computed tomography angiography, embolization, rebleeding, length of stay, and mortality. Older studies were included if they clarified the evolution of LGIB management, highlighted key uses or limitations of viscoelastic testing, or provided essential context for current practice.

Evidence was grouped by clinical directness: (1) studies limited to patients with confirmed LGIB; (2) mixed-source gastrointestinal bleeding studies that included, but did not separately analyse, LGIB patients; (3) studies of cirrhosis-associated upper gastrointestinal bleeding; and (4) evidence from trauma, transplantation, cardiac surgery, or other major haemorrhage settings. Only the first category was considered direct evidence for LGIB. The other categories were treated as indirect evidence and not assumed to be generalizable to LGIB.

Studies were assessed based on population, bleeding source, haemostatic phenotype, viscoelastic platform, timing of testing relative to transfusion, use of a prespecified treatment algorithm, comparator, and risk of confounding by indication. Outcomes of interest included exposure to plasma, platelets, cryoprecipitate or fibrinogen, and red blood cells; achievement of haemostasis; rebleeding; need for endoscopic, radiologic, or surgical intervention; transfusion-related complications; intensive care and hospital length of stay; and mortality. Evidence on detecting anticoagulant or antiplatelet effects was considered separately from evidence on viscoelastic-test-guided transfusion, as standard TEG and ROTEM assays do not reliably identify all clinically significant medication effects.

This review was not registered as a systematic review. Study screening and data extraction were not performed independently by multiple reviewers, formal risk-of-bias tools were not used, and pooled effect estimates were not calculated. The aim was critical synthesis rather than a comprehensive systematic review or meta-analysis. The central question was whether a defined viscoelastic-test-linked treatment pathway improves component-specific transfusion stewardship or patient-cantered outcomes in patients with confirmed LGIB.

4. Contemporary Management of Acute LGIB

4.1. Initial Assessment and Risk Stratification

Management starts with hemodynamic assessment, large-bore intravenous access, targeted laboratory testing, resuscitation, and prompt recognition of severe ongoing bleeding. Initial haemoglobin may underestimate acute blood loss. Serial haemoglobin, lactate, perfusion status, vasopressor use, ongoing haematochezia, comorbidities, and transfusion trends offer further insight. Risk scores can support, but not replace, clinical judgment when determining discharge suitability or identifying patients at higher risk for intervention [1,3].

A key change from previous management is less emphasis on urgent colonoscopy for all hospitalized patients. A 2005 randomized trial found that urgent colonoscopy identified bleeding sources more often than standard care, but did not significantly improve mortality, length of stay, transfusion needs, rebleeding, surgery, or ICU admission [4]. The 2016 ACG guideline recommended colonoscopy within 24 hours for most patients after preparation [5]. The 2023 update now advises nonurgent inpatient colonoscopy for most hospitalized patients, as urgent colonoscopy has not improved major clinical outcomes [1]. This change also highlights an important principle for TEG: a test may enhance diagnostic detail without necessarily improving patient-cantered outcomes.

4.2 Transfusion and Antithrombotic Management

Red-cell transfusion decisions should consider active blood loss, hemodynamic, haemoglobin, ischemic risk, and comorbidities, rather than TEG. Restrictive RBC transfusion is suitable for most hemodynamically stable adults, but should be individualized for those with active ischemia, significant cardiovascular disease, or ongoing exsanguination [1,8]. Platelets, plasma, cryoprecipitate, fibrinogen concentrate, and specific reversal agents each require distinct decision criteria and should not be treated as interchangeable blood products.

A thorough medication history is essential. Vitamin K antagonists, direct oral anticoagulants, heparins, and antiplatelet agents each affect haemostasis differently. Conventional TEG channels may not detect clinically relevant concentrations of some direct oral anticoagulants and do not reliably measure all antiplatelet effects. Specific drug assays, medication timing, renal function, and guideline-based reversal criteria are still required. A normal tracing does not confirm the absence of an antithrombotic drug effect.

4.3 Localization and Source Control

CTA now plays a key role in severe, ongoing, hemodynamically unstable LGIB because it can quickly localize active extravasation and guide angiography [1–3]. A positive CTA in a patient with ongoing haemorrhage typically requires prompt interventional radiology consultation. Modern embolization offers high technical success, though rebleeding and ischemic complications remain significant and depend on technique, anatomy, and patient selection [7]. Colonoscopy is still important for diagnosis, therapy, and ruling out

other causes once the patient is prepared. Surgery is reserved for selected cases after precise localization when endoscopic and radiologic methods are unsuccessful or inappropriate.

TEG should be obtained early, ideally during the initial laboratory evaluation at hospital presentation, when results may influence management. Testing should occur alongside resuscitation, medication assessment, and source control, rather than being delayed until after these steps. In patients on anticoagulant or antiplatelet therapy, an abnormal tracing may reveal a coexisting factor, fibrinogen, or clot-strength deficit, supporting targeted non-red-cell component treatment. Standard TEG cannot reliably exclude all drug effects, so it should supplement, not replace, drug-specific assays and guideline-based reversal decisions [1,25]. TEG cannot localize or stop an arterial bleed, and obtaining it must not delay CTA, angiography, embolization, endoscopy, or surgery. Whether this early integrated approach improves transfusion exposure, rebleeding, length of stay, thrombosis, or mortality in LGIB remains unknown and requires a prospective clinical study.

5. What TEG and ROTEM Measure

TEG and ROTEM measure the changing mechanical properties of clotting whole blood [9,10]. Reaction time in TEG and clotting time in ROTEM broadly reflect clot initiation. K time, alpha angle, and related ROTEM measures describe clot propagation. Maximum amplitude in TEG and maximum clot firmness in ROTEM reflect the combined contribution of platelets, fibrin, and factor XIII to clot strength. Lysis measures estimate clot breakdown. Fibrin-specific channels can help separate the fibrin and platelet contributions to reduced clot strength.

These domains are physiologically intuitive, but results are not interchangeable across devices, activators, cartridges, reagents, or software. Factors such as sample underfilling, transport delay, heparin contamination, temperature, haematocrit, thrombocytopenia, recent transfusion, and timing between sampling and treatment can affect results. Reference intervals should not be used as transfusion thresholds. An effective clinical pathway must specify the platform, channel, trigger, product or drug, dose, reassessment interval, and stopping rule.

TEG presents three main limitations in LGIB. First, it cannot identify the bleeding source. Second, standard assays may not fully detect direct oral anticoagulants or antiplatelet agents. Third, abnormal results are not always causal, as factors such as profound anaemia, shock, hypothermia, dilution, liver disease, and prior transfusion can affect the tracing. Treating every abnormal value may lead to unnecessary transfusions.

Table 1: Contemporary LGIB management and the possible role of viscoelastic testing.

Clinical phase	Contemporary standard management	Potential TEG role and limitation
Initial stabilization	Hemodynamic assessment, intravenous access, serial hemoglobin and perfusion measures, RBC transfusion when indicated	Does not identify the source. If testing is planned, obtain it with the initial laboratory evaluation so that results are available early enough to inform a prespecified non-red-cell component decision.
Antithrombotic assessment	Identify drug, last dose, renal function, and severity; use guideline-based specific reversal when indicated	An abnormal result may reveal a coexisting factor, fibrinogen, or composite clot-strength deficit and inform component therapy. Standard TEG may miss DOAC or antiplatelet effects and must not replace indicated drug-specific reversal.
Clinical phase	**Contemporary standard management**	**Potential TEG role and limitation**
Localization	CTA for ongoing hemodynamically significant bleeding; consider upper source in unstable hematochezia	Should not delay CTA, angiography, or consultation. It provides no anatomical information.
Colonoscopy	Nonurgent inpatient colonoscopy for most hospitalized patients after adequate preparation; endoscopic therapy when appropriate	Does not replace preparation, visualization, or endoscopic hemostasis.
Escalating transfusion	Separate RBC decisions from plasma, platelets, fibrin, and drug reversal; reassess physiology and laboratory trends	A protocolized assay may help target non-RBC components during massive or complex hemorrhage; thresholds require local validation.
Definitive hemostasis	Transcatheter embolization or surgery for persistent bleeding when endoscopic management fails or is unsuitable	May support correction of a genuine systemic deficit, but cannot stop focal arterial or mucosal bleeding.

Table 2: Interpretation of common viscoelastic domains in acute bleeding.

Domain	Typical TEG / ROTEM measure	Interpretation and major caveat
Clot initiation	TEG R time; ROTEM clotting time	Prolongation may reflect factor deficiency, heparin, or an anticoagulant. It is not an automatic indication for plasma and is reagent dependent.
Clot propagation	TEG K time and alpha angle; ROTEM clot-formation time / angle	Slower propagation may indicate impaired fibrin generation. Temperature, hematocrit, and prior transfusion can influence the result.
Overall clot strength	TEG maximum amplitude; ROTEM maximum clot firmness	Low strength may reflect platelets, fibrin, or both. A single global value does not identify the deficient component.
Fibrin contribution	Functional fibrinogen; FIBTEM or analogous channel	Can support targeted fibrin replacement when concordant with clinical bleeding. Thresholds are platform and protocol specific.
Clot lysis	TEG LY30; ROTEM maximum lysis	Apparent hyperfibrinolysis requires confirmation and context. HALT-IT argues against automatic tranexamic acid use in GIB.
Normal tracing	All measured domains within the local interval	All measured domains within the local interval

Table 3: Studies most relevant to viscoelastic testing in gastrointestinal bleeding.

Study	Design / population	Main finding and relevance to LGIB
Green et al. [4]	Randomized trial; 100 patients with acute LGIB	Urgent colonoscopy improved source identification but not major outcomes; illustrates that a better test process may not improve outcomes.
De Pietri et al. [17]	Randomized trial; cirrhosis before invasive procedures	TEG sharply reduced prophylactic plasma/platelets without more bleeding; proof of stewardship, not active LGIB effectiveness.
Chi et al. [18]	Retrospective NSAID-associated GIB cohort; thromboembolism prediction	TEG parameters predicted thrombosis in a selected model; addresses prognosis, not TEG-guided transfusion, and cautions that GIB patients remain prothrombotic.
Kumar et al. [14]	Randomized trial; 96 patients with cirrhosis and nonvariceal upper-GI bleeding	Reduced component exposure and transfusion reactions without worse hemostasis; strong internal validity but indirect for non-cirrhotic LGIB.
Rout et al. [15]	Randomized trial; 60 patients with cirrhosis and variceal upper-GI bleeding	Reduced plasma/platelets and 42-day rebleeding, with no mortality difference; upper-GI cirrhotic physiology limits transferability.
Rizvi et al. [11]	Retrospective ICU mixed-GI cohort; TEG versus no TEG	More products and ventilation in the TEG group, without clear outcome benefit; likely substantial confounding by indication.
Nepal et al. [12]	Small propensity-matched ICU mixed-GI cohort	Greater total product volume and no LOS advantage with TEG; mixed sources, timing, and post-exposure adjustment limit inference.
Abdulelah et al. [13]	Single-center series; 226 severe GIB patients, all tested with TEG	Describes feasibility, high transfusion burden, and 39% mortality; no control group, predominantly cirrhotic/variceal phenotype.
Kvisselgaard et al. [19]	Updated systematic review of randomized bleeding trials	Across bleeding populations, component use may fall, but certainty is low and evidence is mostly perioperative; not direct LGIB evidence.

6. Evidence Relevant to LGIB

6.1. Direct LGIB Evidence

No randomized or prospective comparative study has limited enrolment to confirmed LGIB or tested a prespecified TEG- or ROTEM-linked treatment algorithm against current standard care. The mixed-source studies referenced below included patients with gastrointestinal haemorrhage but did not provide source-specific causal estimates for LGIB. Major LGIB guidelines address red-cell thresholds, platelet targets, anticoagulant reversal, CTA, colonoscopy, embolization, and resumption of antithrombotic therapy, but do not recommend routine viscoelastic testing [1,3,5]. As a result, the impact of a TEG- or ROTEM-linked strategy on transfusion practice, component exposure, time to haemostasis, rebleeding, length of stay, thrombosis, and survival in LGIB remains unestablished. Prospective, LGIB-specific studies are required to determine whether this strategy offers clinical benefit.

This uncertainty does not imply there is no effect. Patients with life-threatening LGIB and complications such as massive transfusion, dilution, hypothermia, acidosis, thrombocytopenia, low fibrinogen, or liver dysfunction may develop systemic haemostatic deficits similar to those in other critical bleeding states. Early viscoelastic testing could potentially identify these deficits and guide targeted haemostatic treatment. However, this hypothesis is based on data from other critical bleeding populations and requires direct evaluation in LGIB-specific clinical studies.

6.2. Older and Foundational Evidence

Earlier LGIB studies focused on the timing and methods of source localization, not on global haemostatic testing. The 2005 urgent-colonoscopy randomized trial increased diagnostic yield but did not improve major outcomes [4]. Later population-based research highlighted the varied causes and outcomes of LGIB [6]. These studies showed that process measures such as earlier testing or higher lesion detection do not necessarily lead to shorter hospital stays or better survival.

The older viscoelastic research focused on transplantation, surgery, trauma, and cirrhosis. A 2010 randomized liver transplantation study found that TEG-guided transfusion reduced intraoperative component use, though transplantation involves a controlled setting with serial testing, significant tissue injury, and standardized replacement protocols [21]. In 2016, De Pietri and colleagues demonstrated that TEG guidance significantly reduced prophylactic plasma or platelet transfusion before invasive procedures in cirrhosis, without increasing bleeding [17]. This provided important evidence for avoiding correction of abnormal INR or platelet count in rebalanced cirrhotic haemostasis, but did not address active LGIB.

A 2018 retrospective study of NSAID-associated gastrointestinal bleeding assessed TEG parameters as predictors of thromboembolism, not as a transfusion intervention [18]. The high reported discrimination should be interpreted with caution, as the study population and outcomes differ from those in acute LGIB, and retrospective modelling can be prone to overfitting. Nonetheless, the study is important because it highlights that patients with gastrointestinal bleeding may remain prothrombotic; focusing solely on "correcting coagulopathy" may overlook the risk of thrombosis.

6.3. Randomized Trials in Cirrhosis with Upper-GI Bleeding

Kumar and colleagues randomized 96 patients with cirrhosis and nonvariceal upper-GI bleeding to receive either TEG-guided or conventional blood-component therapy [14]. TEG guidance reduced component exposure without compromising bleeding control and resulted in fewer transfusion-related adverse events. Rout and colleagues randomized 60 patients with cirrhosis and acute variceal bleeding [15]. TEG guidance reduced plasma and platelet transfusions, maintained initial haemostasis, and was linked to a lower 42-day rebleeding rate, though mortality was unchanged.

These trials offer the strongest gastrointestinal evidence that a TEG-linked protocol can safely influence clinician behaviour. However, their relevance to LGIB is limited. Both studies enrolled cirrhotic patients with abnormal conventional coagulation tests, focused on upper-GI bleeding, used study-specific thresholds, and had relatively small sample sizes. Cirrhosis involves a rebalanced haemostatic system, where INR may overstate bleeding risk, supporting the avoidance of empiric plasma. In contrast, older patients with focal diverticular haemorrhage and no liver disease differ both biologically and clinically.

Meta-analyses of randomized trials in cirrhosis generally find that viscoelastic guidance reduces plasma and platelet exposure without demonstrating a mortality benefit [16]. This finding supports transfusion stewardship but does not show that TEG improves source control, length of stay, or survival in LGIB.

6.4. Mixed-Source Gastrointestinal Bleeding Studies

Rizvi and colleagues retrospectively compared ICU patients with gastrointestinal haemorrhage managed with or without TEG [11]. Patients in the TEG group received more blood products (9.10 versus 3.60 units) and more frequently required mechanical ventilation, without clear improvement in patient-cantered outcomes. These results do not prove that TEG causes harm. Clinicians are more likely to order TEG for patients with shock, ongoing haemorrhage, cirrhosis, or escalating transfusion, which represents classic confounding by indication.

Nepal and colleagues reported a propensity-matched ICU cohort with both upper and lower sources [12]. The TEG group received a higher total blood-product volume and did not have better length of stay or other clinical outcomes. Interpretation is limited by small sample size, mixed etiologist, uncertainty regarding algorithm adherence, and inclusion of variables that may have occurred after exposure in the matching strategy. Adjusting for post-exposure variables can introduce, rather than eliminate, bias.

A 2025 single centre series described 226 ICU patients who underwent TEG during severe gastrointestinal haemorrhage [13]. The population experienced profound shock, frequent alcoholic liver disease and variceal bleeding, high transfusion requirements, and 39% mortality. As all patients underwent TEG and there was no contemporaneous control group, the study demonstrates feasibility

and characterizes a high-risk phenotype but cannot estimate treatment effectiveness.

6.5. Broader Randomized Evidence

The 2026 Cochrane update reviewed 35 randomized trials with 3,096 participants, most of whom had elective cardiac surgery [19]. TEG- or ROTEM-guided algorithms may reduce exposure to fresh-frozen plasma and platelets and may lower rates of surgical reintervention or mortality. However, certainty was rated very low for all outcomes, and there was no clear reduction in red-cell requirements. These findings support the biological and operational plausibility of protocolized component stewardship, but do not demonstrate effectiveness in LGIB. Successful implementation requires rapid turnaround, repeated measurement when needed, clinician adherence, and a defined product algorithm. These conditions may not be present in routine LGIB care.

The HALT-IT trial highlights the risk of applying isolated haemostatic concepts without supporting outcome evidence. High-dose tranexamic acid did not reduce mortality from gastrointestinal bleeding and was associated with increased venous thromboembolism and seizures [20]. Therefore, a lysis signal on TEG should not automatically prompt antifibrinolytic therapy in GI bleeding unless used within a validated protocol.

7. Critical Analysis: why the Results Conflict

7.1. Directness of Evidence

The evidence hierarchy involves more than just distinguishing between randomized and observational studies; clinical relevance is also important. For example, a small, randomized trial in cirrhotic variceal haemorrhage may offer strong internal validity compared to a retrospective LGIB cohort, but its findings may not apply to noncirrhotic LGIB. Similarly, a mixed-GI observational study might include some LGIB patients but may not report the lower source, treatment pathway, or TEG algorithm separately. Neither study design directly addresses the primary research question.

7.2. The Exposure Problem

"TEG ordered," "TEG resulted," and "TEG-guided treatment" represent distinct exposures. Orders may be placed after multiple transfusions, and results may arrive after haemostasis or source control. Clinicians may also choose not to follow the recommended algorithm. Studies that group these scenarios as TEG use risk diluting biological effects and, depending on cohort design, may introduce time-dependent bias, reverse causation, and confounding by indication.

7.3. Confounding by Indication

Sicker patients are more likely to receive TEG, require more transfusions, have longer ICU stays, need ventilation, and experience higher mortality. Conventional multivariable adjustment may not account for bleeding rate, clinician concern, or evolving instability. As a result, increased product use in the TEG group may indicate appropriate selection of high-risk patients rather than treatment failure. Propensity methods cannot address this issue if key severity variables are unmeasured or if post-exposure variables are included.

7.4. Outcome Selection

RBC transfusion, plasma exposure, platelet exposure, cryoprecipitate, haemostatic intervention, rebleeding, thrombosis, and length of stay each measure distinct outcomes. A TEG algorithm is most likely to affect non-RBC component use, as its parameters do not directly correspond to factor, fibrin, and platelet deficits. It is less likely to impact RBC requirements, which are primarily determined by ongoing anatomical blood loss. Mortality and length of stay are influenced by factors such as source control, comorbidities, complications, and hospital workflow. Therefore, a study may show reduced plasma use without a shorter hospital stay and still provide meaningful stewardship benefits.

7.5. Population Heterogeneity

LGIB has varied causes. Diverticular haemorrhage is often brisk and arterial. Ischemic colitis may present with sepsis and organ dysfunction. Malignancy can lead to chronic or recurrent bleeding. Post-procedural bleeding typically has an identifiable lesion. Cirrhosis alters baseline haemostasis. Combining these phenotypes may obscure subgroup benefits or produce misleading associations. Upper and lower sources should not be combined unless source-specific results are reported.

7.5. Assay and Algorithm Heterogeneity

TEG and ROTEM platforms use different reagents and output variables. Thresholds established for liver transplantation or trauma require validation before use in other settings. Some protocols address prolonged initiation with plasma, weak clot strength with platelets, and low fibrin contribution with cryoprecipitate or fibrinogen. The same tracing may prompt different treatments at different institutions. Therefore, the intervention being evaluated is the entire assay-algorithm-clinician system, not just the machine.

8. Where TEG May fit in LGIB Care

Current evidence does not support routine or selective standard-of-care testing. If viscoelastic testing is used, its most defensible role is as a selective adjunct during severe ongoing haemorrhage, particularly when non-red-cell component decisions are unclear, conventional tests do not match the clinical picture, massive transfusion or dilution is occurring, or cirrhosis raises concerns about excessive INR-driven plasma transfusion. Samples should be collected early enough to inform prespecified decisions, and testing must not delay CTA, endoscopy, angiography, embolization, or surgical consultation.

TEG is unlikely to add value in cases of self-limited bleeding, stable patients managed without non-red-cell components, or when results will not affect treatment. For patients on anticoagulant or antiplatelet therapy, early testing may reveal a systemic haemostatic deficit and guide targeted component therapy. It should supplement, not replace, medication-specific assessment and reversal. Routine testing in all cases of haematochezia would increase costs and may lead to unnecessary transfusions due to incidental findings.

A robust clinical pathway should specify eligibility, sample timing, turnaround times, platform-specific thresholds, product dosing,

reassessment, and stopping criteria. It should also distinguish RBC decisions from non-RBC haemostatic therapy and document clinician response to results. Until such a pathway is prospectively evaluated in LGIB, viscoelastic testing should be considered an adjunct rather than standard care.

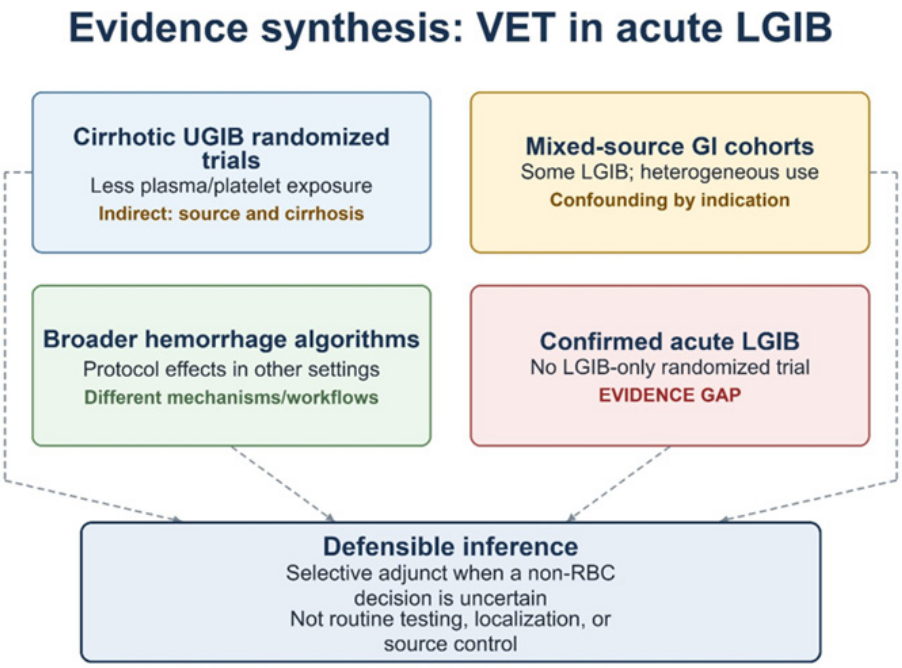

Figure 1: Evidence supporting viscoelastic testing in acute lower gastrointestinal bleeding: directness, limitations, and potential clinical role. The map separates clinical directness from causal certainty; direct LGIB evidence is sparse, and the strongest randomized evidence is indirect. This supports a selective adjunctive role rather than routine use or source-control therapy.

8.1. Antithrombotic-Associated LGIB: A High-Priority Evidence Gap

Antithrombotic exposure is frequent among hospitalized patients with LGIB. In a prospective UK audit of 2,528 cases, 29.4% of patients were on antiplatelet therapy and 15.9% were on anticoagulants [22]. While these figures do not indicate that most patients are exposed in all settings, they confirm that antithrombotic-associated LGIB represents a significant inpatient subgroup.

These patients face two main challenges: a focal bleeding lesion that requires localization and control, and medication effects that may alter systemic haemostasis. Key factors in reversal decisions include drug identity, timing of the last dose, renal function, thrombotic indication, platelet count, conventional tests, and, when available, drug-specific levels. Standard TEG may not detect clinically relevant factor Xa inhibitor activity [25]. TEG Platelet Mapping can identify aspirin- or P2Y12-related inhibition but results often overlap between treated and untreated patients [24]. Dedicated direct-thrombin-inhibitor and anti-factor Xa TEG channels have shown proof-of-concept correlation with drug concentrations, but only in small, nonbleeding cohorts [26].

The value of viscoelastic testing in this subgroup is not to replace medication-based reversal or lesion control. Instead, a drug-aware approach could use standard channels to identify deficits in factors, fibrinogen, or clot strength due to severe haemorrhage and resuscitation, while validated platelet- or anticoagulant-sensitive channels determine if a drug effect is present. This may reduce empiric plasma or platelet use and allow for more targeted transfusion of non-red-cell components. This is clinically relevant because empiric platelet transfusion in antiplatelet-associated gastrointestinal bleeding without thrombocytopenia has not reduced rebleeding and was linked to higher mortality in observational studies [23]. However, no prospective study has evaluated whether a TEG- or ROTEM-guided strategy improves transfusion exposure, rebleeding, length of stay, thrombosis, or mortality in antithrombotic-exposed LGIB.

9. Research Priorities

The first research priority is to identify a clinically coherent LGIB subgroup for whom a non-red-cell component decision is both plausible and time-sensitive. Antithrombotic-exposed patients should be a prespecified subgroup. Studies must distinguish standard global viscoelastic channels from platelet-function and anticoagulant-sensitive assays. They should also differentiate confirmed or highly probable LGIB from mixed-source gastrointestinal haemorrhage, and report bleeding source, cirrhosis, antithrombotic exposure, drug class and timing, baseline severity, assay platform, and timing relative to transfusion and source control. Early observational studies can establish feasibility and generate effect estimates, but ordering a test should not be equated with delivering a TEG-linked intervention. Clinical studies should compare a complete drug-aware pathway with guideline-based care, rather than simply comparing patients with and without a TEG order.

Second, future studies should measure process fidelity, including time from presentation to sampling, order-to-result time, products given before and after the result, algorithm concordance, repeat testing, and reasons for override. Without these data, a neutral result cannot distinguish between an ineffective assay and an inter-

vention that was delivered too late or not followed.

Ultimately, a multicentre prospective trial should compare a complete, prespecified viscoelastic-test-linked treatment pathway with contemporary conventional care in confirmed or highly probable LGIB. Outcomes should align with the proposed mechanism. Plasma, platelet, and fibrinogen-product exposure should be reported separately from red-cell use. Initial haemostasis, rebleeding, rescue intervention, transfusion complications, thrombosis, time to source control, intensive care use, hospital length of stay, and mortality should be measured to assess benefit and safety.

Finally, observational studies remain useful for feasibility and hypothesis generation but require a time-aware design. Baseline severity must be measured before TEG. Patients transfused before the assay should be analysed carefully. Source, cirrhosis, antithrombotic exposure, and source-control timing should be prespecified. Causal claims should be avoided unless the study design supports them.

10. Conclusion

Acute LGIB management focuses on stabilization, appropriate transfusion, rapid localization in cases of severe bleeding, and definitive source control. TEG and ROTEM provide dynamic insights into clot formation that conventional tests lack, but they do not identify or treat the bleeding source.

Current evidence does not support routine use of TEG or ROTEM for LGIB. Favourable randomized data are mainly from cirrhosis-associated upper-GI bleeding and show reduced plasma and platelet exposure, but not consistent survival benefits. Retrospective studies with mixed sources show no clear clinical advantage and are limited by confounding, timing, and lack of protocolization. Existing publications do not clarify whether TEG or ROTEM improves LGIB management in patients on antiplatelet or anticoagulant therapy. This subgroup warrants further study, but standard TEG cannot reliably exclude significant direct oral anticoagulant or antiplatelet effects, limiting its use as a reversal test. The most appropriate role for TEG or ROTEM is targeted assessment during severe haemorrhage when non-red-cell component decisions remain unresolved. Prospective studies should assess a drug-aware, LGIB-specific pathway that integrates medication history, drug-specific testing when available, viscoelastic-guided component decisions, and continued prioritization of localization and source control.

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