

BEYOND THE INR: ROTEM AND TEG AS A COMPASS FOR SAFER CARE IN CIRRHOSIS

IRINA GÎRLEANU^{1,2}, ANA-MARIA SÎNGEAP^{1,2}, LAURA HUIBAN^{1,2}, CRISTINA MUZICA^{1,2*}, SIMONA JUNCU^{1,2},
RALUCA AVRAM^{1,2}, SEBASTIAN ZENOVIA^{1,2}, CAMELIA COJOCARIU^{1,2}, ANCA TRIFAN^{1,2}

¹“Grigore T. Popa” University of Medicine and Pharmacy, Iași, Romania

²Institute of Gastroenterology and Hepatology, “St. Spiridon” Emergency County Hospital, Iași, Romania

* Corresponding author: Cristina Muzica, e-mail: lungu.christina@yahoo.com

Received September 28, 2026

Liver cirrhosis challenges the equation between abnormal coagulation tests and bleeding or thrombosis risk. Rotational thromboelastometry (ROTEM) and thromboelastography (TEG) characterize whole-blood clot formation, strength and lysis, offering functional information beyond the International Normalized Ratio (INR). This narrative review examines their clinical value through the lens of multidisciplinary prevention and early recognition of hemostatic abnormalities. Randomized trials support reduced blood-component exposure during selected invasive procedures and gastrointestinal bleeding, whereas evidence for predicting bleeding, thrombosis or survival remains insufficient. Recent ROTEM trials and a meta-analysis reinforce transfusion stewardship but do not establish safety equivalence or universal treatment threshold. Interpretation must account for platform-specific assays, the incomplete representation of endothelial and anticoagulant pathways, and the dominant contribution of portal hypertension and clinical instability to many bleeding events. The most defensible application is selective, protocol-based testing integrated with hepatology, laboratory medicine, transfusion services, endoscopy and critical care. Future studies should compare viscoelastic strategies with contemporary restrictive blood transfusion strategies and validate longitudinal, multimodal models before routine screening is proposed.

Keywords: liver cirrhosis; thromboelastometry; thromboelastography; hemostasis; prevention.

INTRODUCTION

The assessment of coagulation in patients with liver cirrhosis remains particularly challenging, especially in those with decompensated disease. Conventional coagulation tests, such as prothrombin time (PT), International Normalized Ratio (INR), activated partial thromboplastin time (aPTT), and platelet count, provide only a limited representation of the complex changes occurring in the hemostatic system [1,2]. Although these parameters are frequently abnormal in advanced cirrhosis, they do not necessarily indicate an increased bleeding tendency, as both procoagulant and anticoagulant factors are simultaneously altered [3]. This concept has led to the recognition of a “rebalanced hemostatic system” in cirrhosis, which may nevertheless be fragile and easily disrupted by acute events such as infection, acute kidney injury, or acute-on-chronic liver failure [4].

The INR describes only part of the hemostatic abnormalities associated with liver cirrhosis. Reduced procoagulant factors synthesis coexists with depletion

of natural anticoagulants, increased von Willebrand factor and other compensatory changes; the resulting balance is fragile rather than uniformly hypocoagulable [1]. Consequently, an elevated INR is not a stand-alone indication for prophylactic plasma transfusion, and thrombotic risk persists despite apparently “abnormal” coagulation profile [5]. In this context, increasing attention has been directed toward global or viscoelastic tests of coagulation, particularly thromboelastometry (TEG) or rotational thromboelastometry (ROTEM). Unlike conventional coagulation tests, TEG and ROTEM evaluate the dynamics of clot formation in whole blood, providing information on clot initiation, propagation, cloth strength, and fibrinolysis. They therefore offer a more integrated assessment of hemostasis, incorporating the interaction between plasma coagulation factors, platelets, fibrinogen and fibrinolytic activity [6].

ROTEM and TEG provide dynamic whole-blood measurements that can support targeted therapy [7]. Their preventive promise should be framed precisely: avoiding unnecessary transfusion

is an established clinical objective, whereas early identification of a future bleeding or thrombotic event remains a separate validation problem.

WHAT THE TRACING MEASURES?

Both platforms follow clot initiation, propagation, maximum strength and subsequent lysis, but similar parameter names do not imply interchangeable numerical thresholds (Fig. 1). ROTEM FIBTEM and TEG functional-fibrinogen

assays estimate the fibrin-based contribution after platelet inhibition; they are not direct fibrinogen concentration measurements [6].

Clotting time (CT) and reaction time (R) describe initiation; clot formation time (CFT), K and the alpha angle describe propagation [8]. Maximum clot firmness (MCF) and maximum amplitude (MA) summarize strength, whereas ROTEM A5/A10 provide earlier amplitudes [8]. EXTE./INTEN use different activators; comparison with FIBTEM helps interpret reduced overall firmness without assuming isolated platelet deficiency [8].

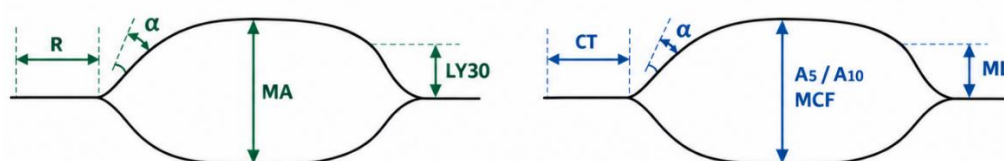


Fig. 1 – TEG/ROTEM parameters. CFT: clot formation time; CT: clotting time; R: reaction time; MCF: maximum cloth firmness; MA: maximum amplitude; ML: maximum lysis; LI: lysis index; LY30: lysis 30 minutes after MA

Their potential value is particularly relevant in decompensated cirrhosis, where conventional tests correlate poorly with the actual risk of bleeding and may lead to unnecessary transfusion of blood products. Viscoelastic testing has increasingly been investigated as a tool for guiding hemostatic therapy during active bleeding and invasive procedures, potentially allowing a more individualized and restrictive transfusion strategy. However, ROTEM and TEG also have important limitations and do not fully reproduce the *in vivo* hemostatic balance, particularly the contribution of endothelial factors and the protein C system. Consequently, current research continues to seek more comprehensive tests, or combinations of biomarkers and functional

assays, capable of accurately reflecting the delicate balance bleeding and thrombosis in patients with advanced and decompensated liver cirrhosis.

CLINICAL EVIDENCE: LESS TRANSFUSION, NOT PROVEN PREDICTION

The earliest procedural randomization evidence came from TEG, whereas dedicated ROTEM trials now support a similar blood-conservation role [9–11]. Table 1 summarizes procedural trials without treating their algorithms as universal transfusion thresholds.

Table 1

Selected randomized trials comparing viscoelastic strategy *versus* conventional-tests guided care

Study/platform	Sample size	Principal finding and interpretive limit
De Petri L, 2016/TEG [10]	60 patients	Blood products: 5/30 <i>versus</i> 30/30. One procedural bleed in the control arm; too few events to establish safety equivalence.
Kumar R, 2025/ROTEM [12]	40 patients	Blood products: 8/20 <i>versus</i> 20/20. No significant procedural bleeding. Early stopping and predominantly low-risk procedures limit inference.
Janko N, 2026/ROTEM [9]	57 randomized procedures	Prophylaxis: 20/28 <i>versus</i> 28/29 procedures. No major bleeding; 77% high-risk procedures. Less plasma but more cryoprecipitate.

A recent meta-analysis of six randomized trials, including 296 patients, reported less exposure to any blood product with viscoelastic guidance (risk ratio 0.33, 95% confidence interval 0.26–0.44) [13]. Procedure-related bleeding was uncommon (risk ratio 0.74, 95% confidence interval 0.24–2.31). These data neither proves bleeding prevention nor excludes

clinically important harm [13]. These trials frequently tested TEG/ROTEM guidance against liberal INR/platelet/fibrinogen based prophylaxis and they do not establish superiority over contemporary restrictive care without routine correction [10,12,13]. Routine testing before low-risk procedures is not currently recommended in liver cirrhosis [5].

BLEEDING MANAGEMENT AND PERIOPERATIVE CARE

In acute variceal bleeding, Rout *et al.* randomized 60 patients and demonstrated that 4/30 in the TEG group *versus* 30/30 in the control group received blood products transfusion [14]. Initial hemostasis profile was similar in both groups, 42-days rebleeding was lower in TEG group (10.0% *versus* 36.7%), although six-week mortality as not significantly different [14]. In non-variceal upper gastrointestinal bleeding, Kumar *et al.* randomized 96 patients. All blood components were administered in 26.5% of patients in viscoelastic group compared to 87.2% in the control group, without significant differences in bleeding control, rebleeding or mortality [15]. Portal pressure reduction and endoscopic source control remain central in variceal bleeding. Baveno VIII Consensus discourages fresh frozen plasma because of ineffective correction of coagulation parameters and the potential for volume overload and worsening portal hypertension; tranexamic acid is also not recommended routinely in this setting [16]. A prolonged clotting time must not become an automatic plasma trigger detached from the bleeding mechanism, and an abnormal lysis parameter alone should not override clinical context [16].

Liver transplantation provides the most established setting for repeated viscoelastic assessment during rapidly changing hemostasis [8]. However, transplant-derived thresholds cannot simply be transferred to stable cirrhotic patients, and evidence from transplantation should not be pooled uncritically with elective nonsurgical procedures [17]. In uncontrolled bleeding, testing should run alongside resuscitation and source control, never delay them.

ROTEM/TEG are *ex vivo* assays, not complete reconstruction of *in vivo* hemostasis, as they incompletely represent endothelial interactions, von Willebrand factor-mediated adhesion and the protein C pathway [1,8]. They can therefore underestimate compensatory hemostatic capacity, while apparently preserved clot strength does not include portal-hypertensive or mechanical bleeding [16]. Standard lysis measurements likewise do not fully characterize the complexity of fibrinolytic system [17]. The results depend on assay activation, platform generation, sample handling and the timing of analysis. Overall, clot strength integrates platelet and fibrin contributors and is not a specific platelet-function assay [8]. Accordingly, interpretation should use laboratory-verified, assay-specific procedures rather than an universal “hypocoagulable” cutoff.

In a prospective cohort of 162 patients, 19 developed bleeding within one year, lower EXTEM

MCF was associated with bleeding, but ROTEM did not predict thrombotic complications [18]. Only two bleeding events were procedure-related, limiting translation to preprocedural screening. In another prospective study of 42 patients with acute decompensation or acute-on-chronic liver failure, serial hypocoagulability tracked disease severity and mortality but not additional bleeding episodes (19). These observations distinguish prognostic association from a validated prediction rule. For the prevention and early-diagnosis theme, the realistic target is early recognition of changing hemostatic function in clinically deteriorating patients, not diagnosis of cirrhosis itself or population-wide thrombosis screening.

MULTIDISCIPLINARY IMPLEMENTATION AND RESEARCH PRIORITIES

Hepatologists should define disease severity and the contribution of portal hypertension, the endoscopists and interventional specialists should assess the procedure, avascular anatomy and feasibility of local hemostasis [16,17]. Laboratory and transfusion teams should verify preanalytics, quality control, turnaround time and assay-specific interpretation, while critical care teams address infection, renal dysfunction and physiological instability. This division of responsibilities prevents the tracing from being interpreted in isolation. A locally implemented program should audit whether testing changes an actual management decision, blood-component exposure, procedural delays, bleeding, thrombosis and total costs. Review of discordant clinical and laboratory findings should be built into the pathways. These are proposed implementation measures rather than evidence-based universal performance targets.

Future trials should compare ROTEM/TEG with restrictive contemporary care, stratify procedural risk and distinguish portal-hypertensive bleeding from hemostatic failure. They should be powered for patient-centered safety outcomes, document repeated procedures within individuals, and report transfusion-related harms alongside thrombosis. Product substitution also matters. Reduced plasma use accompanied by greater cryoprecipitate exposure (20) is not identical to avoiding all hemostatic treatment.

For early risk recognition, serial viscoelastic measurements should be evaluated alongside clinical severity, infection, renal function, fibrinogen and platelet count. Candidate models

require adequate event numbers, calibration, external validation and assessment of clinical utility, and mortality and transplantation should be considered as competing events where appropriate.

CONCLUSIONS

ROTEM and TEG offer a functional perspective that can reduce unnecessary blood-component exposure in selected patients with liver cirrhosis. Their contribution to multidisciplinary prevention is strongest when clinical context comes first, source control remains paramount, and abnormal traces do not trigger reflex correction. Early hemostatic phenotyping is promising, but routine predictive screening awaits robust validation.

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