Clinical Critical Care

E-ISSN 2774-0048

VOLUME 33 NUMBER 1

JANUARY-DECEMBER 2025

Clinical Critical Care
eISSN 2774-0048

Clinical Critical Care
Volume 33, Article ID e250010, 8 pages
https://doi.org/10.54205/ccc.v33.273141

REVIEW

Viscoelastography interpretation

Karuna Chavalertsakul[1], Jakkrit Laikitmongkhon[2]

[1]Division of Critical Care Medicine, Department of Medicine, Faculty of Medicine Ramathibodi Hospital, Mahidol University, Bangkok, Thailand, 10400;
[2]Division of Pulmonary and Pulmonary Critical Care Medicine, Department of Medicine, Faculty of Medicine Ramathibodi Hospital, Mahidol University, Bangkok, Thailand, 10400

OPEN ACCESS
Citation:
Chavalertsakul K, Laikitmongkhon J. Viscoelastography interpretation. Clin Crit Care 2025; 33: e250010.

Received: January 14, 2025
Revised: March 16, 2025
Accepted: March 24, 2025

Data Availability Statement:
The data and code were available upon reasonable request (Jakkrit Laikitmongkhon, email address: tontenbreak@gmail.com)

Funding:
No source of financial support and funding relevant to this article was reported.

Competing interests:
No potential conflict of interest relevant to this article was reported.

Corresponding author:
Jakkrit Laikitmongkhon
Division of Pulmonary and Pulmonary Critical Care Medicine, Department of Medicine, Faculty of Medicine Ramathibodi Hospital, Mahidol University, Bangkok, Thailand, 10400
Tel: (+66) 2-201-1301
E-mail: tontenbreak@gmail.com

ABSTRACT:

The application of viscoelastography, which includes both Thromboelastography (TEG) and Rotational Thromboelastometry (ROTEM), has become increasingly prominent across various clinical disciplines. Its utility is well-documented in surgical settings, trauma care, postpartum care, and critical care medicine, where patients often experience complex and dynamic disturbances in hemostasis. Viscoelastic testing offers a rapid, real-time, point-of-care evaluation of the entire coagulation process, providing valuable insights into the distinct phases of clot initiation, clot strength, and subsequent fibrinolysis. The understanding and timely interpretation of viscoelastography results enhance the precision of hemostatic management by guiding the judicious use of appropriate blood components and hemostatic agents. This targeted approach reduces the risks associated with transfusion-related complications and contributes to improved overall clinical outcomes.

Keywords: Viscoelastography; Thromboelastography; Rotational thromboelastometry

INTRODUCTION

Viscoelastography, encompassing Thromboelastography (TEG) and Rotational Thromboelastometry (ROTEM), offers substantial advantages by providing rapid, real-time, point-of-care evaluation of the entire coagulation process. These technologies offer comprehensive insights into the distinct phases of hemostasis, including clot initiation, clot strength, and fibrinolysis.

Among trauma-related fatalities, post-traumatic exsanguination and trauma-induced coagulopathy are recognized as major contributors to early mortality[1]. To address these critical issues, the sixth edition of the European guideline on the management of major bleeding and coagulopathy following trauma, as well as recommendations from the American Association for the Surgery of Trauma, advocate for a goal-directed strategy in coagulation management. This strategy emphasizes the use of either conventional coagulation assays or viscoelastic monitoring techniques[2, 3]. Beyond trauma care, the application of viscoelastography is well established in other clinical settings such as surgical procedures, critical care, or postpartum hemorrhage[4-6], where patients often present with complex and dynamic coagulation abnormalities.

Multiple studies have demonstrated that the implementation of viscoelastic hemostatic assays in guiding treatment is associated with substantial clinical benefits. These include a reduction in unnecessary blood component transfusions, decreased bleeding volume, a lower incidence of acute kidney injury, and a diminished need for invasive hemostatic interventions[7-10].

THE HEMOSTASIS

The primary hemostasis is initiated immediately following vascular injury. Vasoconstriction, mediated by local tissue factors and the autonomic nervous system, reduces initial blood loss. Simultaneously, platelet adhesion begins at the injury site due to the absence of prostacyclin and nitric oxide, normally secreted by intact endothelium to inhibit adhesion. Subsequently, platelet aggregation and plug formation are triggered by the exposure of collagen within the damaged vessel wall. This collagen exposure activates platelet factors, which attract and aggregate additional platelets to consolidate the hemostatic plug[11].

The secondary hemostasis, encompassing the coagulation cascade, reinforces the primary platelet plug through the generation of a stable fibrin mesh. This process is initiated by the exposure of tissue factor, a procoagulant released from extravascular tissues upon vascular injury. Two principal models currently describe the mechanisms underlying this complex cascade[11, 12] (Figure 1).

The classical cascade model posits two distinct pathways initiating coagulation: the intrinsic pathway, triggered by contact activation, and the extrinsic pathway, initiated by tissue factor. Both pathways converge on a common pathway, culminating in the activation of downstream coagulation factors and ultimately, fibrin clot formation[13] (Figure 2).

The cell-based model describes coagulation as a dynamic process consisting of three overlapping phases. It begins with the initiation phase, where tissue factor-mediated activation of coagulation factors occurs at the site of vascular injury, leading to the generation of a limited amount of thrombin. This is followed by the amplification phase, during which thrombin activates additional coagulation factors, significantly increasing thrombin production. Finally, in the propagation phase, a thrombin burst converts fibrinogen into fibrin monomers, which polymerize to form a stable fibrin mesh. This fibrin mesh, in combination with platelet plugs, establishes the blood clot and achieves hemostasis[14] (Figure 3).

Following fibrin clot formation and hemostasis, then fibrinolysis begins, initiated by plasminogen activators. This process degrades fibrin, breaking down the blood clot to restore blood flow and facilitate wound healing. However, hyperfibrinolysis, which is excessively rapid fibrinolysis, can lead to uncontrolled bleeding[15] (Figure 1).

KEY MESSAGES:

- A goal-directed coagulation management strategy, utilizing either conventional coagulation assays or viscoelastic monitoring, is recommended.
- Viscoelastography, TEG, and ROTEM, provide a rapid, real-time, point-of-care assessment to identify the specific abnormal phases of coagulopathy.
- TEG and ROTEM characteristic graphs resemble a horizontal chili pepper, with the stem, body, and tail representing the initiation, strength, and lysis phases of clot formation, respectively.
- Viscoelastography guided management has demonstrated several benefits, including reduced excessive blood component transfusions, decreased bleeding volume, a lower incidence of acute kidney injury, and a diminished need for invasive hemostatic interventions.

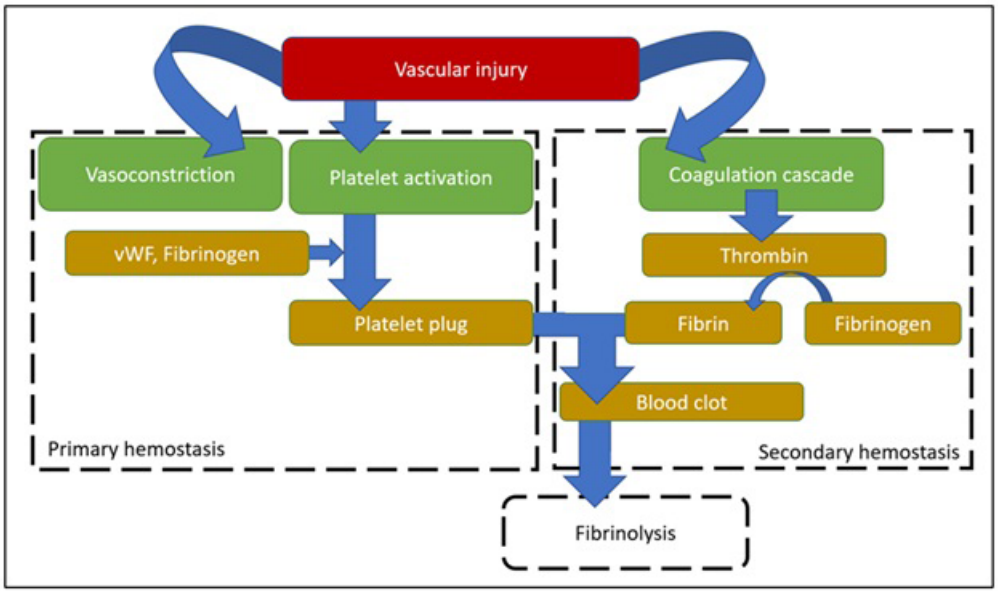

Figure 1. Hemostasis, initiated by vascular injury, consists of three phases. Primary hemostasis involves vasoconstriction and platelet activation, leading to the formation of a platelet plug. Secondary hemostasis entails the generation of thrombin, which activates the conversion of fibrinogen to fibrin. Together with the platelet plug, this results in the formation of a definitive blood clot. Subsequently, fibrinolysis aims to restore vascular circulation and facilitate wound healing. **Abbreviation:** vWF: von Willebrand factor.

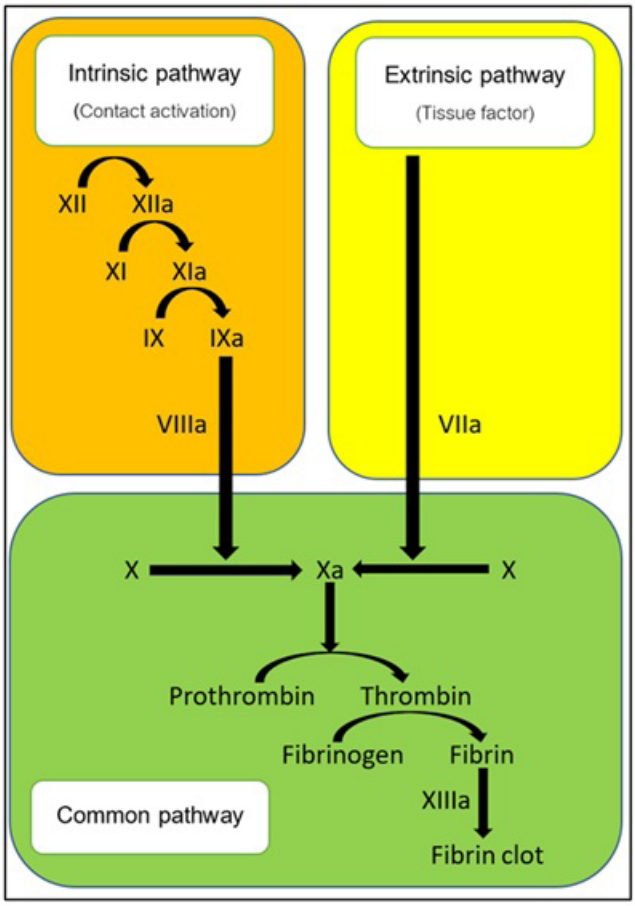

Figure 2. Cascade model of secondary hemostasis.

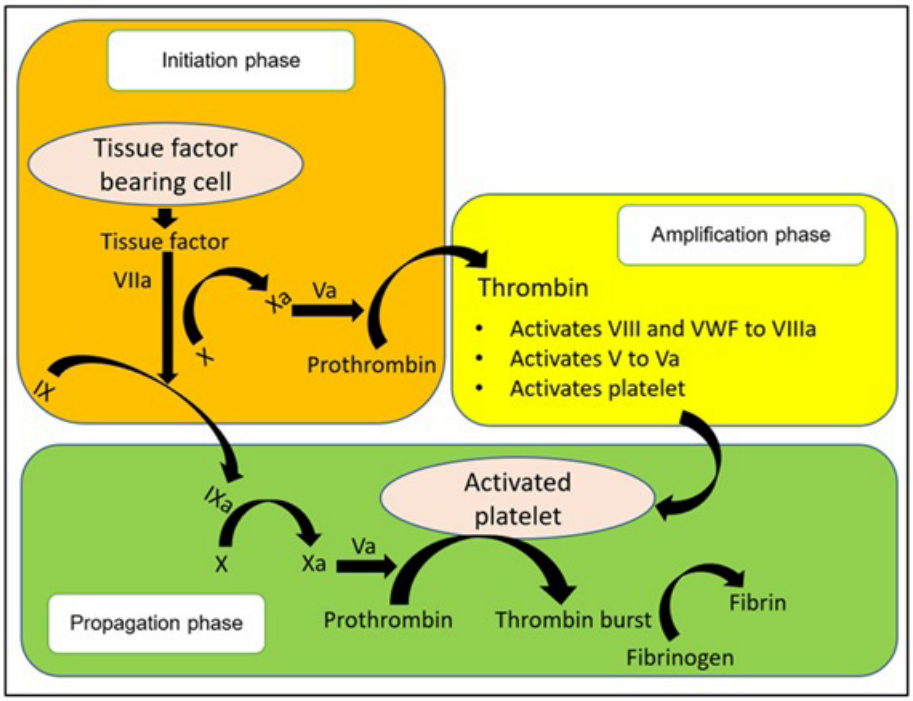

Figure 3. Cell-based model of secondary hemostasis.

VISCOELASTOGRAPHY

Viscoelastography provides a rapid, real-time, point-of-care assessment of viscoelastic properties using a minimal whole blood sample, enabling a swift turnaround time of approximately 20 minutes compared to the 60 minutes required for standard coagulation tests such as prothrombin time (PT), activated partial thromboplastin time (aPTT), activated clotting time (ACT), and platelet or fibrinogen count[16]. This technique measures changes in viscosity as the sample clots, using a transducer that continuously monitors viscosity through rotational movement. The resulting graph depicts distinct phases of clot formation, including initiation, strength, stabilization, and clot lysis[17]. By analyzing specific abnormalities in coagulation phases, viscoelastography supports the optimization of blood component transfusion strategies and minimizes unnecessary transfusions[8].

TEG uses a cylindrical cup containing 340 µL of whole blood, which oscillates at 4°45' every 5 seconds. A pin suspended on a torsion wire within the blood sample detects changes in viscoelastic strength. As clot strength increases, greater rotational force is transmitted to the wire, which is measured by an electromagnetic transducer. TEG enables simultaneous analysis of two samples but requires manual pipetting and is highly sensitive to vibrations, necessitating a stable platform for optimal performance.

In contrast, ROTEM features a fixed cylindrical cup also containing 340 µL of whole blood. A pin suspended on a ball-bearing mechanism oscillates at 4°75' every 6 seconds under constant force. Clot formation restricts pin rotation, which is detected optically via a charge-coupled device image sensor. ROTEM allows for the simultaneous analysis of four samples with automated pipetting, offering enhanced efficiency[17] (Figure 4).

INTERPRETATION

TEG and ROTEM use different nomenclatures to describe the same parameters identifying coagulation phases. Both generate characteristic graphs resembling a horizontal chili pepper, with time in minutes plotted on the x-axis and clot strength amplitude in millimeters on the y-axis. The stem of a chili pepper represents the initiation of clot formation, the body represents clot strength, and the tail represents clot stabilization or the fibrinolysis process. A normal result is depicted by a short stem, a plump body, and a short or absent tail of a chili pepper shape (Figure 5). In addition to the standard test assays, TEG and ROTEM offer specialized tests that incorporate specific reagents to further elucidate the underlying causes of coagulopathy. These adjunctive tests, which will be discussed in detail within each phase of clot formation, enhance the diagnostic utility of viscoelastic monitoring by identifying specific deficiencies or abnormalities in the coagulation process.

CLOT INITIATION

The graphic representation of a chili pepper stem symbolizes the initiation of coagulation. In TEG, this phase is quantified by the reaction time (R), which represents the time required to achieve a 2 mm amplitude. The kinetics time (K) then measures the duration needed for the clot amplitude to increase from 2 to 20 mm. Finally, the alpha angle reflects the slope of the curve, indicating the rate of fibrin build-up and cross-linking. Analogous parameters in ROTEM are clotting time (CT), clot formation time (CFT), and the alpha angle, respectively[18] (Figure 5).

Prolonged R or CT values indicate impaired clot initiation, potentially due to deficiencies in coagulation

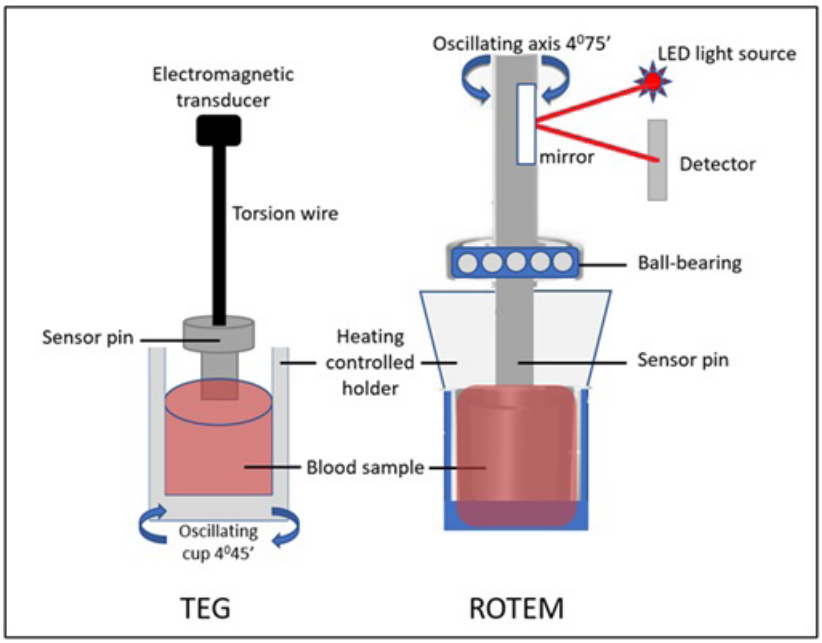

Figure 4. TEG and ROTEM device.

factors, the presence of anticoagulants, or contaminants (Figure 6). Further testing is required to identify the underlying cause. Citrate-kaolin TEG (CK-TEG), which uses kaolin-activated whole blood, can differentiate intrinsic pathway deficiencies. Prolongation of the R value in CK-TEG suggests an intrinsic pathway abnormality, analogous to an abnormal aPTT or ACT. Rapid TEG (rTEG), incorporating both kaolin and tissue factor, aids in identifying coagulation factor deficiencies more broadly. Heparinase-containing TEG (HTEG) neutralizes unfractionated heparin; normalization of the R value in HTEG facilitates the detection of heparin contamination[19].

Similarly, ROTEM assays provide a further comprehensive evaluation of coagulation factor abnormalities. INTEM assesses the intrinsic pathway using a contact activator. Prolonged CT value in INTEM analogous to an abnormal aPTT or ACT. EXTEM evaluates the extrinsic pathway with tissue factor. Prolonged CT value in EXTEM analogous to an abnormal PT. HEPTEM, similar to HTEG, neutralizes heparin to detect contamination, which is demonstrated by the normalization of the CT value in HEPTEM. However, the diagnostic utility of ROTEM, INTEM, and EXTEM in detecting impaired clot initiation may be limited in patients receiving warfarin therapy or low molecular weight heparin[16].

Prolonged K or CFT value, coupled with a shallow alpha angle and prolonged R or CT values, collectively suggests impaired clot initiation.

In cases of coagulation factor deficiencies, administration of fresh frozen plasma (FFP) or prothrombin complex concentrate (PCC) is recommended to correct the coagulopathy. For anticoagulants or contaminants, appropriate antidotal therapy should be implemented, potentially in combination with FFP administration.

CLOT STRENGTH

The maximum amplitude (MA) in TEG and maximum clot firmness (MCF) in ROTEM represent the peak clot strength[18], graphically analogous to the body of a horizontal chili pepper (Figure 5). Subnormal values indicate impaired clot strength, which may result from thrombocytopenia, platelet dysfunction, or hypofibrinogenemia. Further investigation of platelet count and fibrinogen levels is warranted (Figure 7). Function-

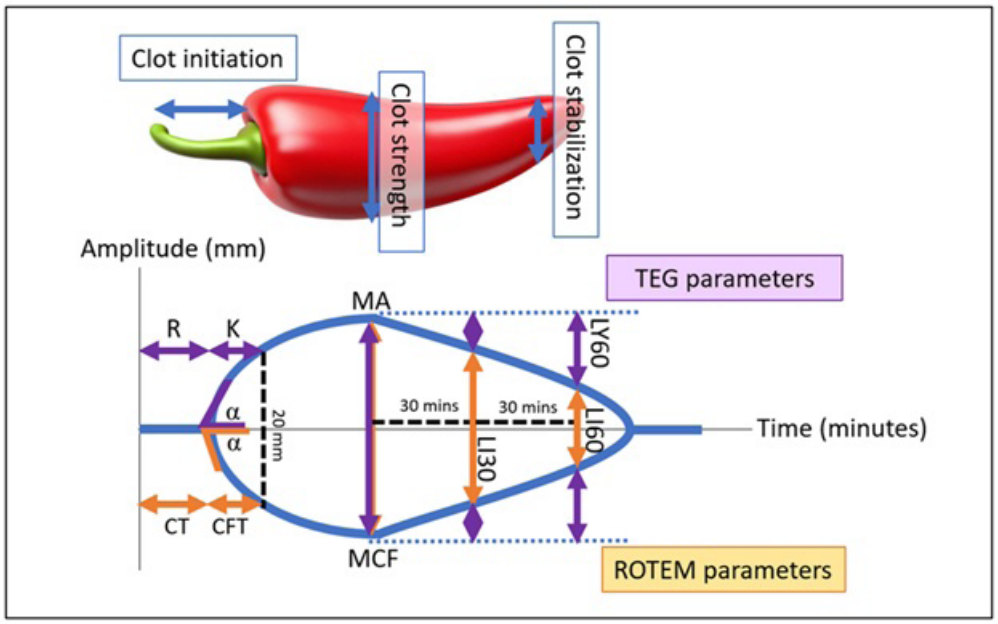

Figure 5. TEG (purple arrows) and ROTEM (orange arrows) parameters include R or CT represent clot initiation. MA or MCF represent clot strength and LY30, 60 or LI30, 60 represent clot stabilization. **Abbreviations:** R: reaction time; K: kinetic time; MA: maximum amplitude; LY30, LY60: lysis at 30 and 60 minutes; CT: clotting time; CFT: clotting formation time; MCF: maximum clot firmness; LI30, LI60: lysis index at 30 and 60 minutes.

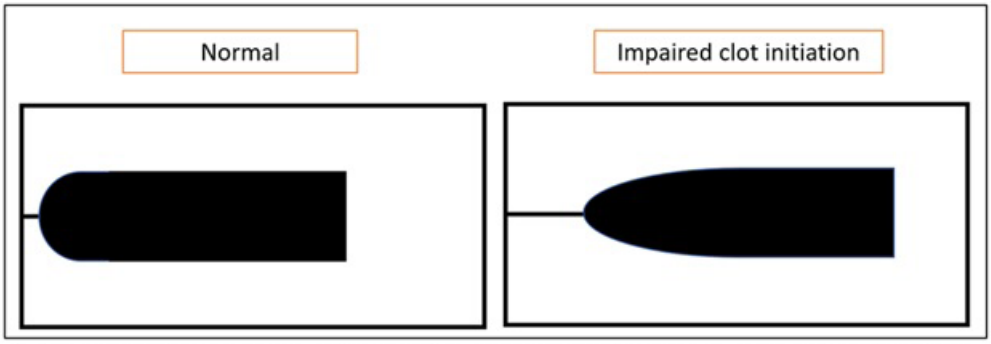

Figure 6. The image shows a tracing with a prolonged R time (TEG) or CT value (ROTEM), resembling a long stem of a chili pepper, which represents impaired clot initiation. **Abbreviations:** R: reaction time; CT: clotting time.

al fibrinogen assays, such as TEG fibrinogen testing or FIBTEM in ROTEM, use reagents to inhibit platelet contribution. Abnormal TEG fibrinogen testing or FIBTEM allows differentiation of fibrinogen-related coagulopathy from platelet-related causes (Figure 8). Additionally, platelet mapping in TEG, which employs agonists like adenosine diphosphate or arachidonic acid to directly activate platelets, can assess both platelet function and quantity. However, the diagnostic utility of TEG and ROTEM is limited in patients with von Willebrand disease (vWD) or those receiving antiplatelet therapy[16]. Management of these conditions depends on the underlying cause. Thrombocytopenia or platelet dysfunction necessitates platelet transfusion, while hypofibrinogenemia requires administration of fibrinogen or cryoprecipitate.

A prolonged K or CFT value, combined with a shallow alpha angle, despite a normal R or CT value but with a reduced MA or MCF value, indicates impaired clot strength.

CLOT STABILIZATION

The morphology of the chili pepper tail serves as a visual analogy for understanding clot stability and fibrinolysis. A short body with a long tail suggests rapid fibrinolysis. In TEG, parameters such as lysis at 30 and 60 minutes (LY30, LY60) represent the percentage reduction in clot amplitude from the MA at these time points. Similarly, in ROTEM, the lysis index at 30 and 60 minutes (LI30, LI60) reflects the percentage of clot remaining from the MCF, presenting an inverse relationship to TEG lysis values (Figure 5). Elevated LY30 and LY60 values, or conversely depressed LI30 and LI60 deviating from established normal ranges, indicate hyperfibrinolysis[18] (Figure 9). ROTEM includes an assay known as APTEM, which utilizes aprotinin to inhibit fibrinolysis. When standard ROTEM results demonstrate reduced LI30 or LI60 values that normalize in the APTEM assay, this pattern supports the diagnosis of hyperfibrinolysis. However, low LI30 or LI60 values observed in standard ROTEM alone are generally considered sufficient for diagnosing hyperfibrinolysis. In such cases, administration of antifibrinolytic agents, such as tranexamic acid, is indicated to correct the underlying coagulopathy.

Although TEG and ROTEM are rapid tools for detecting specific phases of coagulopathy, the identification of fibrinolysis typically takes at least 30 minutes, potentially delaying antifibrinolytic treatment. However, some studies suggest that early indicators, such as the amplitude

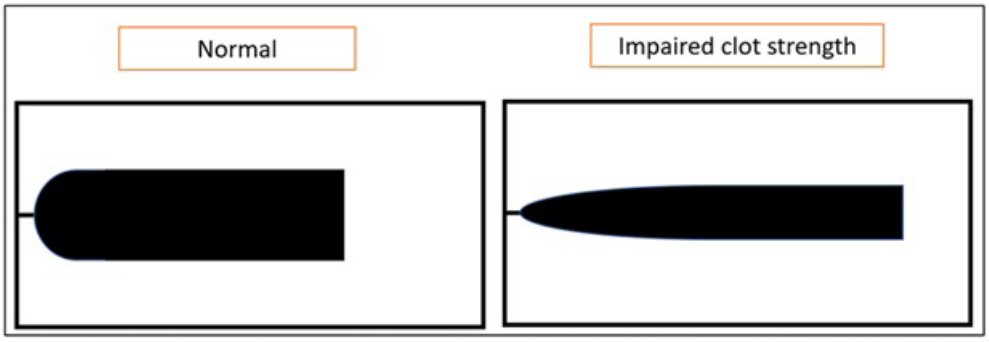

Figure 7. The image shows a tracing with a reduced MA (TEG) or MCF (ROTEM) value, resembling a slim body of a chili pepper, which represents impaired clot strength. **Abbreviations:** MA: maximum amplitude; MCF: maximum clot firmness.

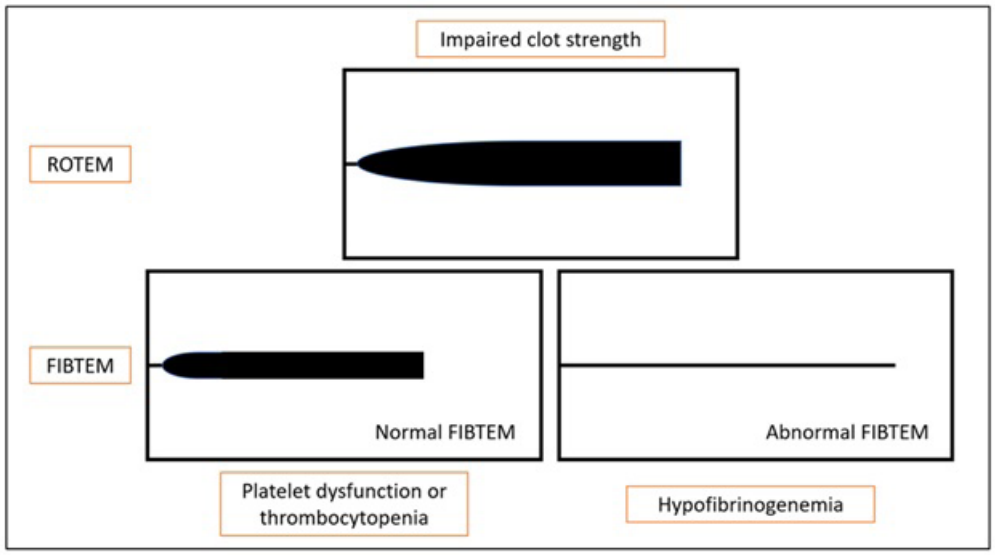

Figure 8. The FIBTEM tracing in a ROTEM specimen demonstrates impaired clot strength. A normal FIBTEM result suggests a platelet-related coagulopathy, such as platelet dysfunction or thrombocytopenia. Conversely, an abnormal FIBTEM indicates hypofibrinogenemia.

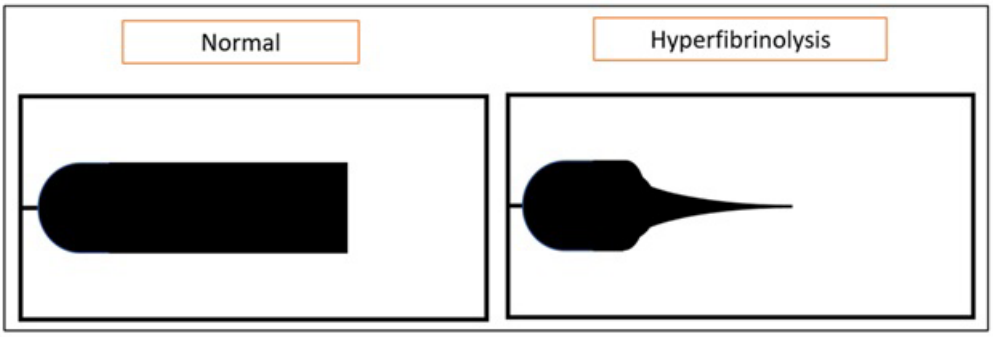

Figure 9. The image shows a tracing with an elevated LY30 or LY60 (TEG) or reduced LI30 or LI60 (ROTEM) value, resembling a long tail of a chili pepper, which represents impaired clot stabilization from hyperfibrinolysis. **Abbreviations:** LY30, LY60: lysis at 30 and 60 minutes; LI30, LI60: lysis index at 30 and 60 minutes.

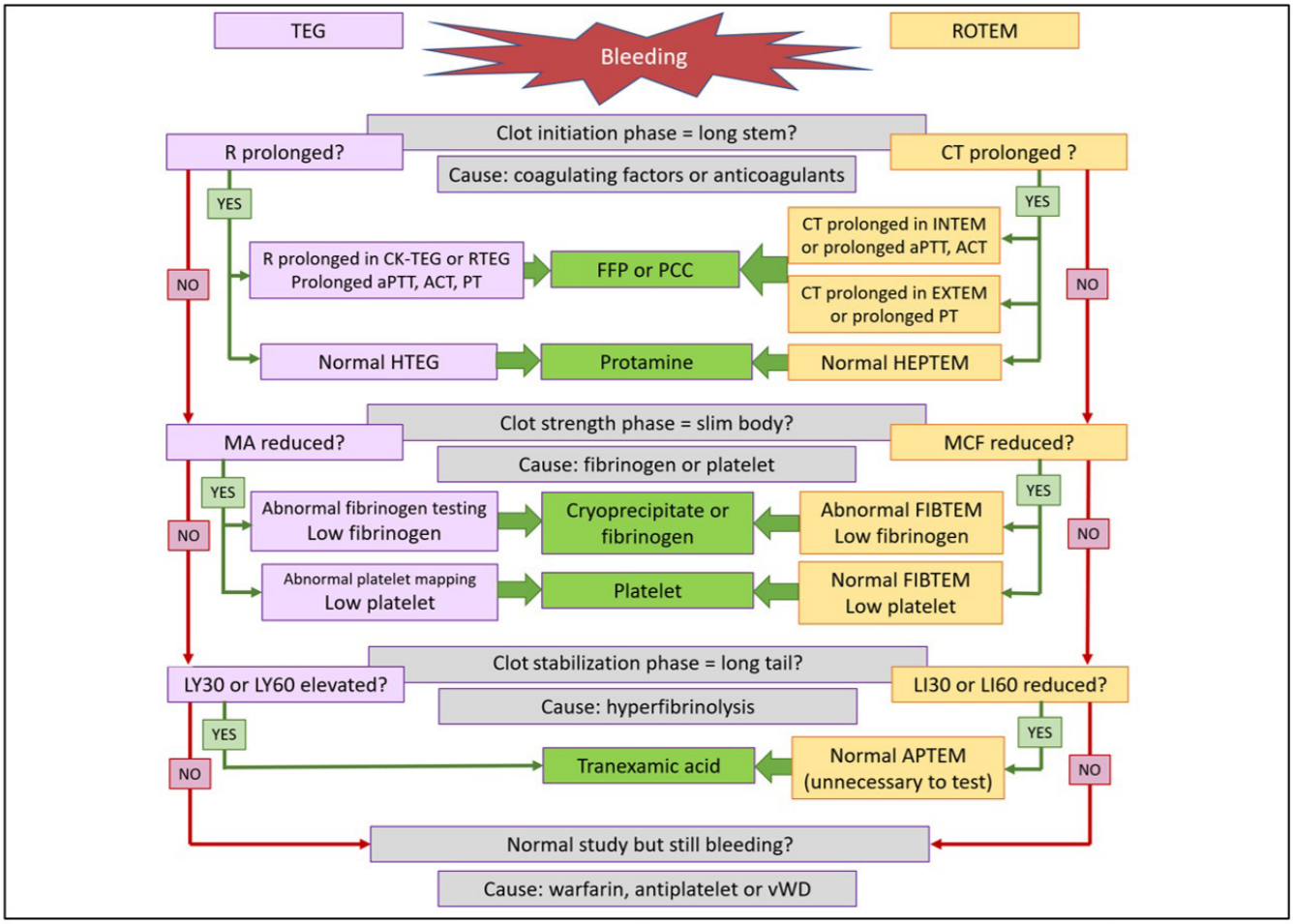

Figure 10. Summary of TEG and ROTEM interpretation. **Abbreviations:** R: reaction time; CT: clotting time; aPPT: activated partial thromboplastin time; ACT: activated clotting time; PT: prothrombin time; FFP: fresh frozen plasma; PCC: prothrombin complex concentration; MA: maximum amplitude; MCF: maximum clot firm-ness; LY30, LY60: lysis at 30 and 60 minutes; CFT: clotting formation time; LI30, LI60: lysis index at 30 and 60 minutes; vWD: von Willebrand disease.

at 5 minutes after clotting time (A5) in EXTEM or the CT value in FIBTEM, can help identify a high likelihood of excessive fibrinolysis. These early markers may support the earlier administration of tranexamic acid, even before definitive LI30 or LI60 values are available, which usually take 30 to 60 minutes to be obtained[20-22].

LIMITATION

Viscoelastography offers several advantages in assessing the underlying causes of coagulopathy and guiding patient management. However, it also has several limitations. It is operator-dependent, not universally available in all healthcare facilities, and is associated with high costs. While the test can detect abnormalities in various phases of hemostasis, it does not quantify the severity of coagulopathy or provide precise guidance on the required treatment dosage. Nonetheless, it can be useful for monitoring treatment responses over time. Additionally, detecting fibrinolysis may require a prolonged testing period, potentially delaying the initiation of antifibrinolytic therapy. There are also specific limitations inherent to the assays themselves; for example, ROTEM, EXTEM, and INTEM have poor sensitivity to detect impaired clot initiation in patients receiving warfarin and low molecular weight heparin. While FIBTEM cannot identify platelet dysfunction contributing to impaired clot strength in patients with vWD or those receiving antiplatelet therapy[16].

CONCLUSION

Due to the advantages of rapid, real-time assessment offered by viscoelastography, accurate and timely interpretation of its results is crucial for guiding targeted treatment of the underlying causes of coagulopathy (Figure 10). This approach ensures precise management while preventing the unnecessary overuse of blood component transfusions, thereby reducing potential risks and improving patient outcomes.

ACKNOWLEDGEMENT

None

REFERENCES

1. Cole E, Weaver A, Gall L, West A, Nevin D, Tallach R, et al. A decade of damage control resuscitation: New transfusion practice, new survivors, new directions. Ann Surg. 2021;273:1215-20.
2. Rossaint R, Afshari A, Bouillon B, Cerny V, Cimpoesu D, Curry N, et al. The European guideline on management of major bleeding and coagulopathy following trauma: sixth edition. Crit Care. 2023;27:80.
3. LaGrone LN, Stein D, Cribari C, Kaups K, Harris C, Miller AN, et al. American association for the surgery of Trauma/American college of surgeons committee on trauma: Clinical protocol for damage-control resuscitation for the adult trauma patient. J Trauma Acute Care Surg. 2024;96:510-20.
4. Drumheller BC, Stein DM, Moore LJ, Rizoli SB, Cohen MJ. Thromboelastography and rotational thromboelastometry for the surgical intensivist: A narrative review. J Trauma Acute Care Surg. 2019;86:710-21.
5. Saner FH, Abeysundara L, Hartmann M, Mallett SV. Rational approach to transfusion in liver transplantation. Minerva Anestesiol. 2018;84:378-88.
6. Khanna P, Sinha C, Singh AK, Kumar A, Sarkar S. The role of point of care thromboelastography (TEG) and thromboelastometry (ROTEM) in management of Primary postpartum haemorrhage: A meta-analysis and systematic review. Saudi J Anaesth. 2023;17:23-32.
7. Wikkelso A, Wetterslev J, Moller AM, Afshari A. Thromboelastography (TEG) or thromboelastometry (ROTEM) to monitor haemostatic treatment versus usual care in adults or children with bleeding. Cochrane Database Syst Rev. 2016;2016:CD007871.
8. Fahrendorff M, Oliveri RS, Johansson PI. The use of viscoelastic haemostatic assays in goal-directing treatment with allogeneic blood products - A systematic review and meta-analysis. Scand J Trauma Resusc Emerg Med. 2017;25:39.
9. Bugaev N, Como JJ, Golani G, Freeman JJ, Sawhney JS, Vatsaas CJ, et al. Thromboelastography and rotational thromboelastometry in bleeding patients with coagulopathy: Practice management guideline from the Eastern Association for the Surgery of Trauma. J Trauma Acute Care Surg. 2020;89:999-1017.
10. Santos AS, Oliveira AJF, Barbosa MCL, Nogueira J. Viscoelastic haemostatic assays in the perioperative period of surgical procedures: Systematic review and meta-analysis. J Clin Anesth. 2020;64:109809.
11. Scridon A. Platelets and Their Role in Hemostasis and Thrombosis-From Physiology to Pathophysiology and Therapeutic Implications. Int J Mol Sci. 2022;23.
12. Gale AJ. Continuing education course #2: current understanding of hemostasis. Toxicol Pathol. 2011;39:273-80.
13. Dahlback B. Blood coagulation. Lancet. 2000;355(9215):1627-32.
14. Hoffman M, Monroe DM, 3rd. A cell-based model of hemostasis. Thromb Haemost. 2001;85:958-65.
15. Risman RA, Kirby NC, Bannish BE, Hudson NE, Tutwiler V. Fibrinolysis: an illustrated review. Res Pract Thromb Haemost. 2023;7:100081.
16. Solomon C, Sorensen B, Hochleitner G, Kashuk J, Ranucci M, Schochl H. Comparison of whole blood fibrin-based clot tests in thrombelastography and thromboelastometry. Anesth Analg. 2012;114:721-30.
17. Whiting D, DiNardo JA. TEG and ROTEM: technology and clinical applications. Am J Hematol. 2014;89:228-32.
18. Srivastava A, Kelleher A. Point-of-care coagulation testing. Continuing Education in Anaesthesia Critical Care & Pain. 2012;13:12-6.
19. Chavez JJ, Foley DE, Snider CC, Howell JC, Cohen E, Muenchen RA, et al. A Novel Thrombelastograph® Tissue Factor/Kaolin Assay of Activated Clotting Times for Monitoring Heparin Anticoagulation During Cardiopulmonary Bypass. Anesthesia & Analgesia. 2004;99:1290-4.
20. Drotarova M, Zolkova J, Belakova KM, Brunclikova M, Skornova I, Stasko J, et al. Basic Principles of Rotational Thromboelastometry (ROTEM(R)) and the role of ROTEM-Guided fibrinogen replacement therapy in the management of coagulopathies. Diagnostics (Basel). 2023;13.
21. Harr JN, Moore EE, Chin TL, Chapman MP, Ghasabyan A, Stringham JR, et al. Viscoelastic hemostatic fibrinogen assays detect fibrinolysis early. Eur J Trauma Emerg Surg. 2015;41:49-56.
22. Wannatoop T, Kittivorapart J, Kittisares K, Werawatakul W, Ruchutrakool T, Permpikul P, et al. Implementation of Viscoelastic Hemostatic Assay-guided Therapy to Evaluate and Manage Trauma-related Bleeding: A Pilot Study from a Level 1 Trauma Center in Bangkok, Thailand. Siriraj Medical Journal. 2022;74:294-304.