Coagulopathy in Liver Disease

5

Naresh Shanmugam and Vimal Kumar

Liver plays an important role in coagulation. Apart from von Willebrand factor, all the other clotting factors are synthesized by the liver. Coagulopathy in liver disease is a reflection of synthetic liver failure. The normal coagulation cascade is outlined in Fig. 5.1. Always look for superimposed causes of coagulopathy such as vitamin K deficiency, infections, and renal failure.

The standard lab test for coagulation such as prothrombin time (PT)/international normalized ratio of PT (INR)/activated partial thromboplastin time (aPTT) assess only plasmatic events in hemostasis. They do not reflect how platelets and other cellular components contribute to coagulation. In liver disease there is concomitant decrease of both procoagulant and anticoagulant levels, platelets, etc.; and the hemostasis is altered. Using PT/aPTT as surrogate marker for bleeding risk is not advocated in liver disease. It can overrate bleeding risk resulting in administration of unneeded or even harmful prohemostatic factors. In liver disease there is concomitant decrease of both procoagulant and anticoagulant levels, and the hemostasis is rebalanced, and so these patients usually do not spontaneously bleed.

Hemostasis in liver disease is best assessed using thromboelastography (TEG). TEG is a point-of-care assay using a specialized machine that assesses clot formation in whole blood, including plasmatic and cellular components. TEG provides a graphical representation (Fig. 5.2) of assembly of a clot in whole blood and provides an assessment of overall hemostasis. Table 5.1 shows TEG parameters and its correlation with coagulation cascade. Anesthetist usually relies on TEG in choosing

N. Shanmugam
Department of Paediatric Liver, GI and Nutrition, Institute of Advanced Paediatrics, Dr. Rela Institute and Medical Centre, Chennai, India

Vimal Kumar (✉)
Department of Paediatric Haematology, Oncology, Blood and Marrow Transplantation, Institute of Advanced Paediatrics, Gleneagles Global Health City, Chennai, India

N. Shanmugam, A. Dhawan (eds.), *Pediatric Liver Intensive Care*,
https://doi.org/10.1007/978-981-13-1304-2_5

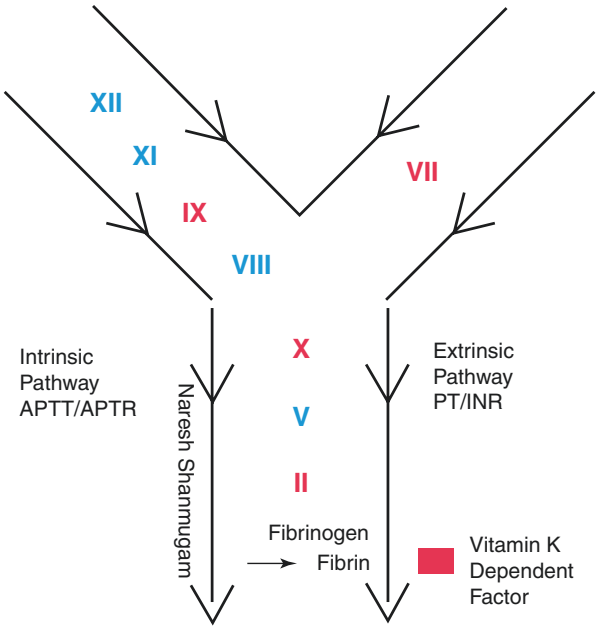

Fig. 5.1 Diagram showing factors involved in intrinsic and extrinsic pathway

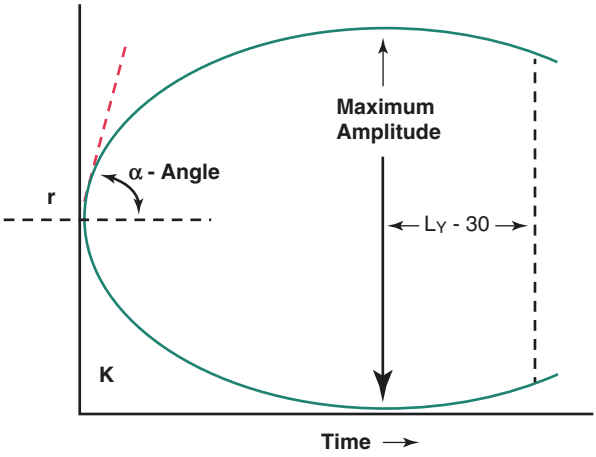

Fig. 5.2 Figure showing standard TEG in a normal person

the appropriate blood components for correcting coagulopathy during liver transplantation.

Tests prior to any surgical procedure:

- CBC, platelet count
- PT/INR, aPTT, fibrinogen
- FDP
- Thromboelastography

Table 5.1 TEG parameters and its correlation with coagulation cascade

TEG parameters	Normal range	Corresponds to	Correlates with
Reaction time in minutes (*r*)	2.5–7.5 min	Time between beginning of the clotting cascade to the initial formation of fibrin	Procoagulant factor levels, INR and aPTT
Kinetic time in minutes (*k*)	0.8–2.8 min	Time between initial fibrin formation to reach a specific clot firmness	Fibrinogen levels and platelet function/ number
α-Angle in degrees	55.2–78.4	Deals with kinetics of clot formation. Rate of fibrin formation and cross-linking of platelets	Fibrinogen levels and platelet function/ number
Maximum amplitude in mm	50.6–69.4	Measures the maximum clot strength	Fibrinogen levels and platelet function/ number
Clot lysis at 30 min (Ly-30; in percentage)	0.0–7.5	Percentage of clot dissolution within 30 min of maximum amplitude	Fibrin degradation products

- Blood group, antibody screen

Treatment options:

- Vitamin K: 2–5 mg IV OD for 3 days corrects vitamin K deficiency associated with decompensated liver disease.
- Platelet transfusion: if platelet count is <50,000/cu/mm in case of bleeding or prior to any invasive procedure.
- Fresh frozen plasma: advantages—contains all coagulation factors, inhibitors of coagulation, and fibrinolytic factors. Disadvantages: volume overload, exacerbation of portal hypertension, risk of infection, risk of transfusion-associated acute liver injury, and transient therapeutic improvement. This may be used when volume expansion is not a concern.
- Cryoprecipitate: hypofibrinogenemia (fibrinogen <100 mg/dL) treated with cryoprecipitate until normal fibrinogen levels reached.
- Recombinant factor VIIa: the most efficient use of this product is in intracranial pressure monitor placement. It may have efficient role in controlling active variceal bleeding when there is no clear endoscopic view. Disadvantages: thrombotic complications and high cost of the therapy.
- For treating local bleeding: aprotinin, tranexamic acid, epsilon aminocaproic acid.
- DDAVP: releases vWF and factor VIII. No benefit with variceal bleeding and after liver surgery.
- Red blood cell transfusion: note that transfusion should be minimum, not allowing Hb to increase more than 8–9 gm/dL.