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WHAT'S NEW IN INTENSIVE CARE

How to interpret abnormal standard coagulation tests at the bedside in critically ill patients?

Julie Helms[1,2,3*], Ling Liu[4] and Lene Russell[5,6]

Coagulation abnormalities are common in the intensive care unit (ICU) and are consistently associated with worse outcomes. They include sepsis-induced coagulopathy (SIC), disseminated intravascular coagulation (DIC), trauma-induced coagulopathy, liver-related hemostatic alterations, and anticoagulation-related abnormalities, all potentially contributing to bleeding, thrombosis, organ dysfunction, and mortality.

This short review provides a pragmatic approach to interpret abnormal standard coagulation tests in common ICU contexts (Table 1). Advanced hemostatic assays are beyond the scope of this review.

A key principle for bedside interpretation

Abnormal coagulation tests should not be corrected in the absence of a clinical correlate, such as active bleeding or thrombosis. Isolated abnormalities, like prolonged clotting times or elevated D-dimer, are common in critically ill patients and, by themselves, do not justify corrective interventions. This principle should guide interpretations across all ICU conditions.

What standard coagulation tests can—and cannot—tell us

Standard coagulation tests like platelet count, prothrombin time (PT/INR), activated partial thromboplastin time (aPTT), fibrinogen, and D-dimer were developed to detect factor deficiencies and were not designed to reflect global hemostatic balance in critically ill patients. Their interpretation in the ICU has important limitations.

For example, prolonged aPTT may reflect lupus anticoagulant in a prothrombotic context rather than bleeding risk, whereas inflammation may shorten aPTT through factor VIII elevation. Prolonged PT/INR may reflect reduced hepatic synthesis, hemodilution, or consumption, rather than the intake of vitamin K antagonists. Conversely, normal PT/aPTT do not exclude clinically relevant bleeding or thrombosis.

Fibrinogen is an acute-phase reactant, often normal or elevated despite consumption. D-Dimer is almost universally increased in critically ill patients and should therefore be interpreted dynamically.

Interpretation across major ICU clinical contexts

SIC and DIC

SIC, defined by abnormal platelet count, PT/INR, and organ failure [1], affects ~45% of septic patients and is associated with increased mortality [2]. Progression to overt DIC further increases mortality (approximately threefold vs no DIC) [3].

Standard coagulation tests reflect a complex interplay between inflammation, endothelial activation, and coagulation. Thrombocytopenia and prolonged PT are frequent but non-specific, while fibrinogen is usually elevated in sepsis due to the acute-phase response [4]. DIC diagnosis is supported by scoring systems (e.g. ISTH) [5]. However, these scores have limitations, as platelet count, PT, fibrinogen, and D-dimer are influenced by liver dysfunction, inflammation, hemodilution, and medications.

DIC management is mainly supportive and symptom-driven; in sepsis, it predominantly addresses a prothrombotic phenotype with little overt bleeding [6–8]. When

*Correspondence: Julie.helms@chru-strasbourg.fr
[3] Hôpitaux Universitaires de Strasbourg, Service de Médecine Intensive-Réanimation, Nouvel Hôpital Civil, Strasbourg, France
Full author information is available at the end of the article

Table 1 Coagulation tests

Test (reference range)	Description	Limitations	Interpretation
Platelet count (150–450 × 10^9/L)	Number of platelets in whole blood	Fragmented erythrocytes or cytoplasmic fragments from malignant lymphoid or myeloid cells can be mistaken for platelets causing a falsely high count Pseudo-thrombocytopenia might be caused by in vitro platelet clumping at room temperature EDTA-induced aggregation of platelets may be seen; using citrated samples usually resolves the clumping	Thrombocytopenia: Seen in multiple conditions including: Infections, sepsis Hemodilution Preeclampsia, HELLP TTP; HUS CAPS HIT Trauma DIC Thrombocytosis: Reactive thrombocytosis driven by an increase in the production of thrombopoietin Chronic myeloproliferative disease
Prothrombin time/INR) (12–14 s/0.8–1.4)	Measures the extrinsic pathway (F VII) and common pathway (F I, II, V and X); the time it takes for plasma to clot after addition of tissue factor, phospholipid and calcium	Acidosis and hemolysis can prolong INR Incompletely filled sample tubes may result in a prolonged INR Elevated hematocrit can result in a falsely high INR INR is insensitive to antithrombin anticoagulation	Increase PT/INR in: Treatment with vitamin K antagonists Conditions with consumption of coagulation factors (e.g. DIC, massive bleeding) Prothrombin (FII) deficiency Severe fibrinogen (FI) deficiency DOAC may cause prolonged PT/elevated INR to a varying and unpredictable degree Liver disease
aPTT (< 34 s)	Measures the intrinsic pathway (mainly F VIII, IX, XI and XII) and the common pathway (F I, II, V and X); the time it takes plasma to clot after addition of calcium and an activator, such as kaolin	High bilirubin levels and free hemoglobin may result in a falsely prolonged aPTT Lupus anticoagulant (which is associated with thrombosis) can cause prolongation of aPTT Acidosis and hemolysis can prolong aPTT	Increased aPTT in: Conditions with consumption of coagulation factors (e.g. DIC, massive bleeding) Treatment with unfractionated heparin or argatroban (also causes prolonged INR) Hemophilia (FVIII or FIX deficiency, acquired or congenital) Direct oral anticoagulants (DOACs) and low molecular weight heparin can cause prolonged aPTT to varying and unpredictable degrees
Fibrinogen (1.4–4.0 g/L)	The amount of circulating fibrinogen in plasma; most commonly measured by the Clauss assay, which measures the formation of fibrin after the addition of purified thrombin	Synthetic colloids such as HES may result in falsely increased levels of fibrinogen	Reduced fibrinogen levels are seen in: Consumption and loss due to bleeding/hyperfibrinolysis DIC (except septic DIC where usually elevated) Congenital fibrinogen deficiency (afibrinogenemia) Liver disease Increased fibrinogen levels are seen in acute inflammatory conditions, including sepsis
Antithrombin (0.80–1.20 kU/L)	The amount of antithrombin in plasma	Treatment with thrombin inhibitors may lead to overestimation of antithrombin levels High levels of bilirubin or triglycerides can affect antithrombin measurements A high hematocrit can give a falsely high antithrombin level	Low levels are seen in: Genetically determined or acquired AT deficiency Sepsis, infections, DIC nephrotic syndrome, liver cirrhosis and preeclampsia Prolonged treatment with unfractionated heparin High values have no clinical significance

Table 1 (continued)

Test (reference range)	Description	Limitations	Interpretation
D-Dimer (< 0.5 mg/L)	The amount of the fibrin degradation product D-dimer in plasma	D-Dimer levels increase with age High levels of bilirubin or triglycerides may falsely increase D-dimer levels An elevated serum rheumatoid factor level may falsely increase D-dimer levels	The negative predictive value for thrombosis is high (> 90%), which means that the analysis can be used to rule out deep vein thrombosis and pulmonary embolism. However, a high value is non-specific and is seen not only in deep vein thrombosis and pulmonary embolism, but also in DIC, trauma, infection/inflammation, thrombolysis treatment and pregnancy, among other conditions

aPTT activated partial thromboplastin time, *AT* antithrombin, *CAPS* catastrophic antiphospholipid syndrome, *DIC* disseminated intravascular coagulation, *DOAC* direct oral anticoagulant, *EDTA* Ethylenediamine tetra-acetic acid, *F* factor, *HELLP* hemolysis, elevated liver enzymes, and low platelets, *HIT* heparin-Induced thrombocytopenia, *HUS* hemolytic uremic syndrome, *INR* international normalized ratio, *TTP* thrombotic thrombocytopenic purpura, *TP* prothrombin time

no bleeding is present, routine correction of coagulation abnormalities is therefore not recommended, and standard thromboprophylaxis should be maintained [9].

Distinguishing thrombotic microangiopathies from DIC

Thrombocytopenia is the most common coagulation abnormality, seen in about half of ICU patients. Sepsis, liver dysfunction, medication effects, and bone marrow suppression are among the most frequent causes [10]. Thrombocytopenia is a strong marker of severity in critical illness and is consistently associated with worse outcomes, particularly in patients with septic shock [11]. Although often associated with bleeding, it may indicate prothrombotic conditions, which require urgent treatment. TMA and DIC overlap clinically but differ mechanistically: TMA causes microangiopathic hemolytic anemia with schistocytes, severe thrombocytopenia, and renal/neurological injury, while coagulation tests are often normal. Conversely, DIC involves consumption with prolonged PT, increased fibrinogen in sepsis, and markedly elevated D-dimer. Thrombotic thrombocytopenic purpura results from severe ADAMTS13 deficiency, whereas hemolytic uremic syndrome is Shiga toxin-mediated or complement-driven [12]. Distinguishing TMA from DIC is crucial, as failure to do so delays etiology-specific therapy. Blood smear and targeted testing are essential.

Trauma-induced coagulopathy

Trauma-induced coagulopathy is characterized by an initial hypocoagulable phase increasing bleeding risk, followed by a hypercoagulable, prothrombotic state. Although TIC has classically been defined as a prothrombin ratio of 1.2 or higher, it is associated with a complex series of coagulation changes, including thrombocytopenia, low fibrinogen, and elevated D-dimer levels, which reflect consumption and fibrinolysis. Hemodilution from fluid and blood product resuscitation contributes to low fibrinogen and prolonged PT/INR and should be considered when interpreting coagulation results in trauma patients.

Liver disease and cirrhosis

Patients with liver disease often present with abnormal coagulation tests due to reduced synthesis of both procoagulant and anticoagulant factors, as well as thrombocytopenia related to reduced thrombopoietin and splenic sequestration. Despite prolonged PT/INR and aPTT, stable patients with cirrhosis are often in a rebalanced hemostatic state. This balance can be disrupted by sepsis, bleeding, or hemodynamic instability, leading to a phenotype that may resemble DIC [13].

Extracorporeal organ support

Extracorporeal organ support profoundly alters hemostasis through blood-surface interactions, inflammation, and anticoagulation exposure. Thrombocytopenia, elevated D-dimer, and prolonged clotting times are common and reflect ongoing coagulation activation (and the use of anticoagulant therapy), rather than isolated bleeding risk. Serial monitoring of D-dimer levels in ECMO patients shows that trends over time, not absolute values, may provide insight into ongoing vascular injury, fibrin turnover, and bleeding risk [14, 15].

Monitoring anticoagulation in these patients is challenging, as anti-Xa and aPTT values may be confounded by hyperbilirubinemia, hemolysis, hypertriglyceridemia, antithrombin deficiency, factor VIII elevation, hemodilution, and assay interference. Discordance between laboratory tests and clinical anticoagulant effect is frequent, underscoring the need for cautious interpretation and integration of laboratory results with clinical context and circuit-related events.

Practical bedside considerations

No single test captures the dynamic nature of coagulation in critical illness [16]. Interpretation should rely on global patterns, clinical context, and serial measurements rather than isolated values. Integrating test trends with the patient's trajectory helps distinguish adaptive responses from pathological dysregulation. In patients with unexplained bleeding or thrombocytopenia, standard coagulation tests should prompt further targeted investigations rather than empirical correction, as immune-mediated and acquired factor deficiencies cannot be excluded based on PT/INR or aPTT alone.

The way forward

Abnormal standard coagulation tests are common in critically ill patients and reflect complex interactions between inflammation, endothelial dysfunction, organ failure, and therapeutic interventions. Understanding their limitations and interpreting them within the clinical context—while avoiding correction in the absence of a clinical correlate—is essential for rational bedside decision-making. A mechanism-informed, phenotype-oriented approach remains key to balancing bleeding and thrombosis risks in the ICU.

Author details

[1] INSERM (French National Institute of Health and Medical Research) UMR 1260, Regenerative Nanomedicine (RNM), Strasbourg, France. [2] Université de Strasbourg (UNISTRA), Faculté de Médecine, Fédération Hospitalo-Universitaire (FHU) TARGET, Strasbourg, France. [3] Hôpitaux Universitaires de Strasbourg, Service de Médecine Intensive-Réanimation, Nouvel Hôpital Civil, Strasbourg, France. [4] Jiangsu Provincial Key Laboratory of Critical Care Medicine, Department of Critical Care Medicine, Zhongda Hospital, School of Medicine, Southeast University, Nanjing, China. [5] Copenhagen University Hospital Herlev-Gentofte, Copenhagen, Denmark. [6] Department of Clinical Medicine, University of Copenhagen, Copenhagen, Denmark.

Data availability statement

NA.

Declarations

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