OPEN ACCESS Freely available online

ISSN: 2155-9864
Journal of Blood Disorders & Transfusion

Perspective

Coagulation Cascade Dysregulation: Biological Disturbances in Hemostatic Control

Chris Reeder*

Department of Hematology and Transfusion Medicine, Southern Pacific University, Wellington, New Zealand

DESCRIPTION

The human circulatory system depends on a carefully balanced process that prevents excessive blood loss while maintaining uninterrupted blood flow through vessels. This balance is maintained through a sequence of biochemical reactions commonly known as the coagulation cascade. Under normal circumstances, coagulation factors, platelets, vascular structures, and regulatory proteins interact in a coordinated manner to produce stable clot formation at sites of injury. When this system becomes dysregulated, the consequences can range from severe bleeding to life-threatening thrombosis. Coagulation cascade dysregulation represents a significant concern in modern medicine because it contributes to numerous acute and chronic disorders affecting patients of all ages.

The coagulation cascade consists of multiple clotting factors that become activated in a stepwise sequence. Traditionally, this process has been divided into intrinsic, extrinsic, and common pathways. Although modern understanding emphasizes a more integrated cellular model, the traditional framework remains useful for understanding how abnormalities arise. Activation begins when tissue injury exposes sub endothelial structures and tissue factor to circulating blood. This event initiates a chain reaction that eventually converts fibrinogen into fibrin, creating a mesh that stabilizes the platelet plug and limits blood loss.

Dysregulation occurs when the equilibrium between clot formation and clot limitation becomes disturbed. Such disturbances may involve excessive activation of coagulation factors, inadequate production of regulatory proteins, genetic abnormalities, inflammatory responses, infections, malignancies, autoimmune diseases, or metabolic disorders. The resulting imbalance alters normal hemostatic function and can produce diverse clinical manifestations.

One of the most recognized examples of coagulation dysregulation is disseminated intravascular coagulation. This condition involves widespread activation of clotting pathways throughout the circulation. Instead of remaining confined to a localized injury, clotting mechanisms become activated in multiple vascular beds simultaneously. As clotting factors and platelets are consumed, patients experience both thrombosis and bleeding. Small vessel occlusion can impair blood supply to organs, while depletion of coagulation components increases susceptibility to hemorrhage.

Inflammation plays a substantial role in coagulation abnormalities. The immune and coagulation systems maintain close communication through numerous signaling pathways. During inflammatory responses, cytokines stimulate tissue factor expression and promote activation of clotting factors. Simultaneously, natural anticoagulant systems may become less effective. This interaction creates a postthrombotic environment that can persist long after the initial inflammatory trigger. Chronic inflammatory disorders such as rheumatoid arthritis, inflammatory bowel disease, and systemic lupus erythematosus have been associated with elevated thrombotic risk due to these mechanisms.

Liver disease frequently affects coagulation because the liver synthesizes many clotting factors and anticoagulant proteins. Conditions such as cirrhosis can reduce production of essential coagulation components, leading to bleeding tendencies. At the same time, reduced synthesis of anticoagulant factors may increase thrombotic risk. This dual effect illustrates the complexity of coagulation regulation and demonstrates why laboratory findings do not always predict clinical outcomes.

Cancer-associated coagulation abnormalities represent a significant challenge in clinical practice. Malignant cells can release substances that activate coagulation pathways and stimulate platelet aggregation. Tumours may also promote inflammation, endothelial dysfunction, and vascular injury. As a result, individuals with cancer face a substantially increased risk of venous thromboembolism. Certain malignancies, including pancreatic, gastric, and lung cancers, demonstrate particularly strong associations with thrombotic complications. Anticoagulant therapy often becomes an important aspect of comprehensive cancer management.

Correspondence to: Chris Reeder, Department of Hematology and Transfusion Medicine, Southern Pacific University, Wellington, New Zealand, Email: chris.reeder@sputm.ac.nz

Received: 27-Jan-2026, Manuscript No. JBDT-26-31741; **Editor assigned:** 30-Jan-2026, PreQC No JBDT-26-31741 (PQ); **Reviewed:** 13-Feb-2026, QC No. JBDT-26-31740; **Revised:** 20-Feb-2026, Manuscript No. JBDT-26-31741 (R); **Published:** 27-Feb-2026, DOI: 10.4172/2155-9864.25.17.641

Citation: Reeder C (2026). Coagulation Cascade Dysregulation: Biological Disturbances in Hemostatic Control. J Blood Disord Transfus.17:641.

CONCLUSION

Coagulation cascade dysregulation represents a complex biological phenomenon involving interactions among clotting factors, platelets, vascular structures, immune mediators, and genetic influences. Disturbances within this network can produce severe clinical consequences, ranging from uncontrolled bleeding to widespread thrombosis. Continued investigation into molecular mechanisms, diagnostic technologies, and targeted therapeutic strategies is enhancing the ability of healthcare professionals to identify and manage these disorders. As scientific knowledge expands, improved understanding of hemostatic regulation will continue to support safer and more effective approaches for patients affected by coagulation abnormalities.