SCHOLIX RESEARCH SIMPLIFIED

Journal of Medical and Health Sciences

Open access | ISSN :1234-5678

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Disseminated Intravascular Coagulation is a Serious Blood Clotting Disorder

Volume 1, Issue 1

Date of Submission : 03-07-2026

Date of Acceptance : 04-07-2026

Date of Publication : 11-07-2026

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Citation: Siniša Franjić. (2026) "Disseminated Intravascular Coagulation is a Serious Blood Clotting Disorder", Journal of Medical and Health Sciences, 1(1); 10.61148/JIMHS /001.

Abstract

Siniša Franjić. (2026) "Disseminated Intravascular Coagulation is a Serious Blood Clotting Disorder", Journal of Medical and Health Sciences, 1(1); 10.61148/JIMHS /001.vessels. This condition simultaneously causes the depletion of platelets and clotting factors, leading to excessive bleeding. It arises as a result of various critical illnesses and is characterized by the occurrence of both blood clotting and bleeding at the same time.

Keywords: DIC, Blood, Thrombosis, Pathology, Health

Introduction

DIC, or disseminated intravascular coagulation, represents a syndrome marked by the uncontrollable activation of coagulation within the blood vessels [1]. The key feature of DIC is the extensive activation of thrombin, which causes thrombosis leading to a significant reduction in clotting factors and platelets, subsequently resulting in severe bleeding. The coexistence of thrombosis and bleeding, coupled with the triggering of the inflammatory response, results in extensive damage to organs and increases mortality rates.

Platelets

Platelets are critical components in the process of blood coagulation and are tiny, nucleated cell fragments that derive from substantial precursor cells known as megakaryocytes found in the bone marrow [2]. Their average lifespan in the bloodstream is around ten days, after which they are cleared away by macrophages in the spleen. Platelets contain proteins that can contract and have two different types of granules that include various bioactive mediators necessary for proper platelet functionality. When a blood vessel gets injured, platelets quickly adhere to the damaged area, forming a temporary seal, a process significantly aided by von Willebrand Factor (vWF). This adhesion triggers platelet activation and the release of substances resulting in the secretion of several active elements. Serotonin, resembling histamine, causes blood vessels to constrict. Furthermore, certain platelet activators, such as ADP and thromboxane A2, which is a substance similar to a lipid-derived prostaglandin, are also released. These agents encourage platelet aggregation by partially binding to fibrinogen in the plasma, creating links between platelets and attracting more platelets from the bloodstream. Moreover, platelets undergo substantial shape changes that significantly increase their

surface area. This group of clustered platelets is referred to as the primary hemostatic plug, which initially manages bleeding but can be dislodged or washed away. The establishment of this primary hemostatic plug defines what is known as primary hemostasis, the aspect of the hemostatic mechanism that does not rely on blood coagulation factors found in the serum.

Aggregated platelets present phospholipids with a negative charge on their exterior. These phospholipids are essential for the processes involved in secondary hemostasis. This mechanism includes plasma coagulation factors, which are a sequence of inactive enzymes and cofactors that gather in the presence of the phospholipid surface and calcium ions, transforming into active enzymes. At the initial stages, thrombin is generated from its precursor, prothrombin. Thrombin is vital for secondary hemostasis, commonly referred to as humoral coagulation. A portion of the initially generated thrombin attaches to a particular receptor on the clustered platelets, leading to the contraction of the platelets and resulting in an irreversibly merged assembly of platelets and fibrinogen. This conglomeration creates a strong secondary hemostatic plug, with fibrin, derived from fibrinogen through thrombin's activity, being a major constituent of the developed thrombus. However, it is important to understand that the creation of the mature thrombus necessitates the collaboration of humoral coagulation factors.

Thrombosis

This highly critical disruption of the clotting mechanisms within the blood vessels arises when localized areas of thrombosis are concurrently activated across the entire vascular system [3]. Numerous triggers exist, but primarily, they function by increasing tissue factor expression on monocytes/macrophages and endothelial cells—cells that usually do not have this on their surface—thereby initiating the clotting process. In cases caused by infections, this occurs through the creation of a lipopolysaccharide/lipopolysaccharide-binding protein complex, which attaches to CD14 on macrophages/monocytes, resulting in activation and expression of tissue factor.

The process of forming and removing several thrombi in the smaller blood vessels and capillary network rapidly depletes the plasma reserves of both coagulation and antithrombotic factors. Depending on which factor dominates, the resulting clinical presentation can manifest as either thrombosis or bleeding.

In hemorrhagic diseases, the damage to endothelial cells often coincides with the same triggers causing disseminated intravascular coagulation (DIC), leading to bleeding and oozing. The overwhelmed coagulation cascade fails to close the minor injuries caused by the loss of endothelial cells. This results in significant blood volume loss and the onset of multiorgan failure. Failure of the kidneys and lungs tends to be first. If more than three organ systems fail, death is almost unavoidable.

DIC is clinically visible as widespread petechial hemorrhages on the skin and mucous membranes; during autopsy, these spots can be found throughout the gastrointestinal tract, in the mucous membranes of the bronchial tree, and on the surface of the pericardium. The early detection of fibrin degradation products in the bloodstream signifies the beginning of DIC. All clotting assessments—international normalized ratio (INR), activated partial thromboplastin time (APTT), and thrombin time (TT)—show abnormal results in DIC cases, accompanied by thrombocytopenia.

Management includes eliminating the underlying cause (e. g., septicemia), replenishing the lost blood and clotting factors, and providing support for failing organs.

Multiple-Organ Failure

Many individuals who succumb to septic shock experience either persistent low blood pressure or failure of multiple organs [4]. Persistent low blood pressure can arise due to two primary processes. Initially, some individuals may be unable to maintain a substantial cardiac output in response to the septic condition, leading to an advancing form of high-

output cardiac failure. Additionally, circulatory collapse might occur as a result of significant blood vessel relaxation and low blood pressure that do not respond to intravenous fluids or medications aimed at increasing blood pressure.

The onset of multiple-organ failure marks the final stage of an excessive metabolic reaction that initiates in the early moments of shock. Organ dysfunction arises from damage to the microvasculature caused by both local and systemic inflammatory reactions to infections. An uneven distribution of blood flow is worsened by the compromised ability of red blood cells to change shape, leading to blockages in small blood vessels. Clumping of neutrophils and platelets may further impede blood circulation. The release of neutrophils from the blood vessel walls leads to greater liberation of inflammatory substances and the movement of neutrophils into surrounding tissues. The activation of components from the complement system draws in additional neutrophils while releasing active substances like prostaglandins and leukotrienes in the area. The cumulative effect of these alterations is the collapse of the microvasculature and, eventually, organ failure.

The prognosis for sepsis is linked to the extent of organ failure: the death rate among individuals with failure of multiple organs (three or more systems) tends to be around 70%. About 18% of individuals suffering from sepsis experience respiratory failure. The most acute condition on this spectrum is acute respiratory distress syndrome, which is marked by persistent low oxygen levels, reduced lung flexibility, pulmonary swelling not caused by heart issues, and elevated blood pressure in the lungs. Kidney failure, present in 15% of cases, is typically multifactorial, resulting from a combination of reduced blood flow to the kidneys, intra-renal shunting, and the use of substances that can harm the kidneys, such as certain antibiotics and dyes used in imaging. Other affected organs due to sepsis can include the central nervous system (leading to altered consciousness and coma) and blood issues (such as disseminated intravascular coagulation).

Conditions

Damage to organs may trigger issues in disseminated intravascular coagulation (DIC): kidney failure can hinder the function of platelets, liver dysfunction can reduce the production of clotting factors, and issues with blood vessel lining can diminish nitric oxide levels in the blood, causing continuous activation of platelets [1]. Several conditions are linked to DIC:

- Sepsis
- Trauma (such as head injuries, burns, or fat emboli)
- Cancer
- Complications during pregnancy (like placental abruption or pre-eclampsia)
- Pancreatitis

In hematological cancers such as leukemia, intracranial bleeding often occurs either at diagnosis or during relapse [5]. In these cases, thrombocytopenia frequently accompanies intracranial bleeding. In some instances, this is due to the consumption of platelets resulting from DIC rather than a decline in platelet production. DIC is marked by a tendency for both the accumulation of fibrin and increased breakdown of fibrin. In the absence of any additional clotting abnormalities, intracranial bleeding is relatively rare if the platelet count exceeds 20,000/mm3. Typically, the bleeds are multiple and can vary from small pinpoint bleeds to large, widespread ones. The extensive nature of these bleeds can lead to quick neurological deterioration. If the hemorrhage is localized and not widespread, the resulting stroke syndromes depend on the vascular regions affected. Both acute and chronic subdural hematomas can occur.

Coagulation System

Disseminated intravascular coagulation arises from the abnormal activation of the coagulation system and can be triggered by multiple disease states [6]. Damage to endothelial cells and the release of tissue factor can instigate this activation, resulting in the presence of both thrombin and plasmin in circulation. Thrombin facilitates the conversion of

fibrinogen to fibrin, which accumulates in small blood vessels, leading to thrombosis in the microvasculature. This process results in the depletion of coagulation factors, which manifests as extended prothrombin time, an extended activated partial thromboplastin time, lower levels of fibrinogen, and diminished platelet counts. This leads to an increased risk of bleeding. Enhanced levels of circulating plasmin break down both fibrinogen and fibrin, causing elevated fibrin degradation products and D-dimers. These factors interfere with the polymerization of fibrin and the functionality of platelets, contributing further to the risk of bleeding. In pediatric cases, DIC is typically acute and associated with an unwell child; it may be secondary to infections, injuries (like crush injuries or burns), and cancers (such as leukemia and liver conditions). There can also be a more chronic, compensated version characterized by: (1) milder alterations in coagulation parameters, with the possibility of only a moderate decrease in platelet levels; (2) plasma fibrinogen levels that are usually normal or slightly elevated; and (3) normal ranges for both prothrombin time and activated partial thromboplastin time. For such individuals, diagnosis is based on evidence of microangiopathy seen in blood films (including fragments of red blood cells and lower platelet numbers) alongside increased levels of fibrin degradation products and/or D-dimers. A persistent localized variant of DIC is observed in cases of giant hemangiomas, known as Kasabach-Merritt syndrome.

Abnormal Activation

The significance and prevalence of this condition have become increasingly acknowledged in the past few years, as it can arise following a diverse array of traumatic, infectious, and other acute incidents [7]. Commonly referred to as DIC, this condition has multiple alternate names reflecting its nature as a consumption coagulopathy related to the blood-clotting mechanism.

In DIC, there is atypical activation of the coagulation cascade within blood vessels due to various factors associated with the blood itself, the walls of blood vessels, and the flow of blood. Tissue damage from trauma and burns can initiate coagulation driven by thromboplastin: the introduction of cellular components from tissues, particularly ones from erythrocytes, brain matter, and placenta, is especially potent. Particulate substances such as microorganisms or types of micro emboli (including fat and air emboli, particularly those arising from decompression) can trigger coagulation through factor XII activation. Damage to the vascular endothelium and slowed blood flow may produce similar outcomes.

Regardless of the underlying causes—which are exceptionally intricate—fibrinogen is consumed and fibrin is formed within blood vessels, causing both blockage effects in the vasculature and a bleeding diathesis due to the depletion of the coagulation apparatus. Platelets are also depleted, adhering to the formed fibrin thrombi. Fibrinolysis is initiated, resulting in a continuous struggle between intravascular coagulation and its clearance.

After death, post-mortem fibrinolysis may occur, making it more challenging to detect fibrin deposits. Staining methods for visualizing fibrin are also not entirely reliable, thus necessitating a thorough examination of autopsy histology to locate any remnants of fibrin. The organs most likely to provide positive findings include the lungs, kidneys, liver, and adrenal glands.

Microvascular blockages, which can occasionally lead to significant infarctions and functional impairments, alongside bleeding, are the main threats posed by DIC within a forensic framework.

Tests

For individuals exhibiting clinical signs of a bleeding condition, specific screening tests are recommended [8]. The physiological trigger for coagulation primarily originates from tissue factor, with small amounts of thrombin enhancing this process. For the sake of convenience, the terminology of extrinsic, intrinsic, and common pathways continues to be utilized.

Coagulation assessments evaluate the duration until clot formation occurs in vitro using a platelet-poor plasma sample after activating agents and calcium are introduced. The results from the test sample are then compared against normal control values. The assessment of the tissue factor ("extrinsic") pathway is conducted using the prothrombin time (PT), while the "intrinsic" pathway is evaluated via the activated partial thromboplastin time (APTT), which is sometimes referred to as the partial thromboplastin time with kaolin (PTTK). Delays in coagulation can arise from a lack of coagulation factors or the presence of inhibitors, like heparin and lupus anticoagulants. If both PT and APTT values are extended, it suggests either a deficiency or inhibition within the final common pathway, which encompasses factors X, V, prothrombin, and fibrinogen, or a global deficiency affecting multiple factors, as seen in disseminated intravascular coagulation (DIC). Additional specific tests might be conducted depending on the clinical context and the outcomes of the initial screening tests. A mixing test involving normal plasma provides a way to distinguish between a factor deficiency (where the prolonged time normalizes) and the presence of an inhibitor (where the prolonged time remains unchanged). The latter may occur either through a chemical agent, like heparin, or an antibody (commonly a lupus anticoagulant, though it can also involve a specific inhibitor targeting a coagulation factor, typically factor VIII).

Historically, platelet function was evaluated through bleeding time, determined as the duration required for bleeding to cease following a standardized incision. However, most laboratories have moved away from this method. In vitro assessments of platelet function can be conducted by analyzing aggregation responses to various agonists such as adrenaline (epinephrine), collagen, thrombin, arachidonic acid, and ADP, as well as measuring agglutination in response to ristocetin or by evaluating the levels of intracellular granule components such as adenosine triphosphate, adenosine diphosphate, and their respective ratios (ATP/ADP).

In disseminated intravascular coagulation (DIC), there is a depletion of platelets and coagulation factors, leading to thrombocytopenia along with extended PT and APTT values. Furthermore, signs of active coagulation are present with the consumption of fibrinogen and the production of fibrin degradation products (D-dimers). It is important to note that fibrinogen is an acute phase protein that may also see an increase in inflammatory diseases.

Diagnosis

Significant disseminated intravascular coagulation can occur even when standard coagulation tests appear normal [9]. The primary diagnostic tests are those that show excessive conversion of fibrinogen into fibrin and its subsequent breakdown. Platelet and fibrin clots form a structure in the microcirculation, which can injure passing red blood cells, leading to fragmentation and hemolysis. A blood smear may reveal red cell fragmentation (microangiopathic hemolytic anemia), though this is often seen more frequently in chronic disseminated intravascular coagulation, particularly in cases related to cancer.

Diagnosis typically relies on a blend of the correct clinical presentation and lab assessments of the hemostatic system. Indicators of disseminated intravascular coagulation include thrombocytopenia, low fibrinogen levels, and prolonged activated partial thromboplastin time, prothrombin time, and thrombin clotting time, paired with high levels of fibrin degradation products from the D-dimer test. More specific tests can further support the diagnosis, such as increased fibrinopeptide A and decreased antithrombin levels.

In chronic disseminated intravascular coagulation, laboratory results differ from those in acute cases, frequently showing normal or nearly normal hemostatic testing outcomes. Chronic disseminated intravascular coagulation represents a balanced state where there is heightened turnover of the hemostatic factors. D-dimer levels are elevated in this context, and fragmentation of red blood cells is often visible in the peripheral blood smear.

Management

The most effective approach for managing disseminated intravascular coagulation is to address the underlying cause [1]. Additionally, supportive treatment with blood products is essential:

• Platelet transfusions are necessary to maintain a platelet count above 50 x 10^9/L if there is a significant bleeding risk.
• Fresh frozen plasma serves as a source of clotting factors, excluding fibrinogen, with an adult dosage of 15 mL/kg, which is roughly equal to four units of fresh frozen plasma.
• Cryoprecipitate or fibrinogen concentrate should be administered to ensure fibrinogen levels stay above 1. 5.
• The use of prothrombin complex concentrate may be contemplated, and in cases of severe bleeding, recombinant factor VIIa might be recommended; however, these specialized products require prior discussion with the hematology team before use.

Extensive clotting within the microcirculation of organs can lead to ischemia and potential irreversible harm to the kidneys, liver, spleen, lungs, and brain.

Conclusion

Disseminated intravascular coagulation is a severe disorder resulting from a malfunctioning process of fibrin and thrombin generation within the blood. This abnormal production causes platelets to aggregate and deplete coagulation factors. Consequently, small blood clots form and travel through the bloodstream, leading to blockages in blood vessels.

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