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LITERATURE REVIEW: DIC SYNDROME – CURRENT ISSUES IN DIAGNOSIS AND TREATMENT

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Abstract: *Disseminated intravascular coagulation (DIC) is a complex pathological condition characterized by uncontrolled activation of the hemostatic system, leading to the depletion of coagulation factors, widespread microthrombus formation, and severe hemorrhagic complications. The pathogenesis of DIC involves a dysregulated interplay between coagulation and fibrinolysis, with key contributors including excessive tissue factor release, endothelial dysfunction, and inflammatory activation. This review explores the fundamental mechanisms driving DIC progression, highlighting the roles of thrombin generation, platelet consumption, and fibrinolytic disturbances. Additionally, modern diagnostic approaches, including multi-biomarker panels, are discussed alongside emerging therapeutic strategies aimed at restoring hemostatic balance. Understanding these mechanisms is crucial for improving early detection, risk stratification, and patient outcomes in DIC.*

Keywords*: DIC syndrome, coagulopathy, anticoagulants, diagnosis, hemostasis.*

I. INTRODUCTION

Disseminated intravascular coagulation (DIC) syndrome is a severe pathological condition characterized by systemic activation of the coagulation system, leading to widespread thrombus formation, depletion of clotting factors, and a high risk of hemorrhagic complications. This dysregulated process results in the simultaneous development of microvascular and macrovascular thromboses, which impair organ perfusion and contribute to multiple organ dysfunction syndrome (MODS). At the same time, consumption of platelets and coagulation factors, combined with excessive fibrinolysis, predisposes patients to life-threatening bleeding.

DIC can develop in response to various underlying conditions, including sepsis, severe trauma, obstetric complications (such as placental abruption or amniotic fluid embolism), ischemic strokes, malignant tumors, and systemic inflammatory diseases [1]. The condition is particularly common in critically ill patients, where persistent inflammatory and prothrombotic stimuli drive

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coagulation activation and endothelial injury. The release of tissue factor (TF) from damaged tissues and activated monocytes plays a central role in initiating the coagulation cascade, while the loss of natural anticoagulant mechanisms, such as antithrombin III (ATIII), protein C, and protein S, exacerbates thrombotic risk.

Endothelial dysfunction is another key contributor to DIC pathogenesis. Under normal conditions, the endothelium regulates hemostasis by balancing procoagulant and anticoagulant factors. However, in DIC, endothelial cells become activated and lose their protective function, releasing von Willebrand factor (vWF), pro-inflammatory cytokines (such as interleukin-6 and tumor necrosis factor-alpha), and plasminogen activator inhibitor-1 (PAI-1), further amplifying coagulation and inhibiting fibrinolysis. This dysregulation leads to a vicious cycle of clot formation, ischemic damage, and hemorrhagic complications.

The clinical manifestations of DIC vary widely, ranging from subclinical forms detected only by laboratory abnormalities to fulminant cases with severe thrombosis and profuse bleeding. The variability of presentations, combined with the absence of a single definitive diagnostic test, makes DIC a challenging condition to identify and manage. Laboratory markers such as D-dimer, fibrin degradation products (FDPs), prolonged prothrombin time (PT), activated partial thromboplastin time (APTT), low fibrinogen, and thrombocytopenia are commonly used to assess disease severity. Additionally, scoring systems such as the International Society on Thrombosis and Haemostasis (ISTH) DIC score help clinicians evaluate the risk and progression of the disorder.

Given the complex pathophysiology of DIC, treatment strategies must be tailored to the underlying cause while addressing both thrombotic and hemorrhagic risks. Supportive therapy, including anticoagulation with heparin in cases of overt thrombosis, administration of fresh frozen plasma, fibrinogen concentrates, or platelet transfusions for bleeding complications, and correction of underlying conditions, remains the cornerstone of management. Additionally, emerging research on novel anticoagulants, fibrinolysis modulators, and endothelial protective agents offers new avenues for therapeutic intervention.

Understanding the intricate mechanisms of DIC is crucial for improving early diagnosis, optimizing treatment approaches, and reducing the high morbidity and mortality associated with this life-threatening syndrome.

II. Relevance of the Problem

Despite significant progress in understanding the pathogenesis and developing treatment approaches for disseminated intravascular coagulation (DIC) syndrome, its diagnosis remains challenging due to the wide variability in clinical manifestations and the lack of universal biomarkers. This literature review examines the key aspects of DIC pathogenesis,

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emphasizing the roles of coagulation activation, endothelial dysfunction, and fibrinolytic imbalance. Additionally, modern diagnostic approaches, including emerging multi-biomarker panels, are explored alongside current treatment strategies aimed at restoring hemostatic equilibrium. The review also highlights ongoing challenges in early detection and management, as well as potential research directions that may lead to improved diagnostic precision and targeted therapeutic interventions.

III. Pathophysiology of DIC Syndrome: Mechanisms and Consequences

Disseminated Intravascular Coagulation (DIC) is a serious condition where the body's blood clotting system becomes overactive, leading to both excessive clotting and severe bleeding. This results in widespread tiny blood clots (microthrombi) that block small blood vessels, cutting off oxygen to organs and causing multiple organ failure.

DIC is often triggered by infections (such as sepsis), major injuries, burns, or cancer. The main cause is the massive release of tissue factor (TF) into the blood from damaged cells, which starts an uncontrolled clotting process. This leads to excessive thrombin production, which converts fibrinogen into fibrin—forming the basis of clots. The result is a hypercoagulable state, where microclots form everywhere, impairing circulation.

At the same time, the body's natural anticoagulants (antithrombin III, protein C, and protein S) get used up. Normally, these substances help keep clotting under control, but in DIC, their depletion worsens the problem. Additionally, the inner lining of blood vessels (the endothelium) stops working properly, further promoting clot formation. It also releases inflammatory molecules like TNF-α and IL-6, which make the condition worse.

Eventually, the body tries to break down the excessive clots through fibrinolysis. However, this process becomes overactive, leading to an increase in fibrin degradation products (FDPs), which prevent normal clotting. This shift causes the body to bleed uncontrollably, marking the later stages of DIC.

DIC is a dangerous cycle: first, excessive clotting leads to organ failure, and then uncontrolled bleeding occurs due to the depletion of clotting factors. This makes the condition life-threatening and difficult to manage.

IV. Clinical Manifestations and Diagnosis

Disseminated Intravascular Coagulation (DIC) syndrome presents with a wide spectrum of clinical manifestations, ranging from latent forms with minimal symptoms to severe cases characterized by profuse hemorrhages and multi-organ failure. The syndrome progresses through three distinct phases. Initially, the hypercoagulable phase is marked by excessive thrombus formation in small blood vessels, leading to impaired organ perfusion and dysfunction. As clotting

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factors and platelets become depleted, the condition transitions into the consumption coagulopathy phase, where the body's ability to maintain hemostasis deteriorates. Eventually, in the fibrinolytic phase, the body attempts to break down the widespread clots, leading to an overproduction of fibrin degradation products (FDPs). These FDPs interfere with normal coagulation, resulting in uncontrolled bleeding and worsening the hemorrhagic complications of DIC [5].

Diagnosis of DIC relies on specific laboratory markers that reflect both hypercoagulation and fibrinolysis. Elevated levels of D-dimer and FDPs indicate excessive clot breakdown, while decreased fibrinogen and platelet counts suggest ongoing consumption of clotting factors. Additionally, prolongation of prothrombin time (PT) and activated partial thromboplastin time (APTT) confirms the presence of coagulation abnormalities, further supporting the diagnosis [6].

To assess the severity of DIC, the International Society on Thrombosis and Haemostasis (ISTH) has established a standardized scoring system based on four key laboratory parameters (Table 1). This system is widely implemented in clinical practice for diagnosing and monitoring DIC progression. However, despite its usefulness, the ISTH criteria have limited sensitivity in the early stages of the disease, which can pose challenges in early detection and intervention.

TABLEI. ISTH DIAGNOSTIC CRITERIA (2001) [6]

Parameter	Points
Platelets < 100 × 10^9/L	1
Platelets < 50 × 10^9/L	2
Moderately elevated D-dimer	2
Markedly elevated D-dimer	3
Fibrinogen < 1 g/L	1
Prothrombin time (PT) prolonged by 3-6 seconds	1
PT prolonged by > 6 seconds	2

Diagnostic Criteria:

- ≥5 points – high probability of DIC syndrome.
- <5 points – possible latent DIC, requires monitoring.

The ISTH (International Society on Thrombosis and Haemostasis) criteria offer a structured and standardized approach to diagnosing disseminated intravascular coagulation (DIC), balancing both strengths and limitations. One of their key advantages is their high specificity for severe cases, ensuring that significant coagulation abnormalities are reliably identified.

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Additionally, the scoring system is straightforward and user-friendly, making it a practical tool for clinicians working in critical care and hematology settings.

Despite these benefits, the ISTH criteria also have notable drawbacks. Their low sensitivity in the early stages of DIC means that mild or evolving cases may go undiagnosed until the condition becomes more advanced. Furthermore, the system is not always well-suited for chronic forms of DIC, such as those associated with malignancies, where coagulation abnormalities tend to be less pronounced and fluctuate over time [7].

While the ISTH criteria remain a widely used tool for diagnosing and monitoring DIC, they are most effective when supplemented with clinical judgment and additional laboratory assessments. Given the complex and multifactorial nature of DIC, there is an increasing need for more precise diagnostic methods that can detect early pathological changes and improve risk stratification. Recent research has focused on identifying novel biomarkers that reflect different pathophysiological mechanisms, including coagulation activation, endothelial dysfunction, systemic inflammation, and fibrinolysis regulation. These emerging biomarkers hold promise for enabling earlier and more accurate detection of DIC, potentially improving patient outcomes.

A. Biomarkers of Coagulation Activation

Since disseminated intravascular coagulation (DIC) is characterized by widespread thrombin generation and dysregulated coagulation, several biomarkers have been identified to evaluate the intensity of thrombosis. One such marker is prothrombin fragment F1+2, which is produced during the conversion of prothrombin to thrombin and serves as an indicator of thrombin activity [5]. Another key biomarker is the thrombin-antithrombin (TAT) complex, which correlates with disease severity and is particularly elevated in cases of DIC associated with septic shock [6].

In addition to markers of thrombin activity, the protein C system plays a crucial role in regulating coagulation. A significant decline in protein C and its activated form (aPC) reflects depletion of the body's natural anticoagulant mechanisms, increasing the risk of microvascular thrombosis [7]. The reduction in aPC activity not only signifies a loss of anticoagulant control but also contributes to endothelial dysfunction and inflammatory responses, further exacerbating the pathological process of DIC.

B. Biomarkers of Endothelial Dysfunction

Endothelial activation is a central factor in the pathogenesis of disseminated intravascular coagulation (DIC), making markers of endothelial injury valuable tools for early diagnosis and disease monitoring. One such marker is vascular endothelial growth factor (VEGF), which increases in response to endothelial dysfunction and serves as an indicator of vascular damage [8].

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Similarly, von Willebrand factor (vWF), a key regulator of platelet adhesion, is significantly elevated in severe DIC, reflecting extensive endothelial injury and heightened thrombotic activity [9].

Another important marker of endothelial dysfunction is hyaluronic acid, which signals degradation of the endothelial glycocalyx—a protective barrier lining the vasculature. Increased levels of hyaluronic acid provide insights into inflammatory vascular damage and microvascular dysfunction, which contribute to the progression of DIC [10]. The evaluation of these endothelial biomarkers, alongside traditional coagulation markers, may enhance early detection and risk stratification in patients with DIC.

C. Biomarkers of Inflammation

Given the strong association between disseminated intravascular coagulation (DIC) and systemic inflammation, particularly in septic conditions, inflammatory biomarkers play a crucial role in predicting disease progression. High-sensitivity C-reactive protein (hsCRP) is widely used to assess systemic inflammatory activity and has been shown to correlate with the severity of coagulopathy in DIC [11]. Elevated interleukin-6 (IL-6) levels further contribute to disease progression by promoting coagulation activation while simultaneously inhibiting fibrinolysis, thereby exacerbating the prothrombotic state [12].

Another important biomarker is soluble CD40 ligand (sCD40L), which serves as a marker of platelet activation. Increased levels of sCD40L are associated with a heightened risk of thrombosis and may have prognostic significance in patients with DIC [13]. The interplay between these inflammatory markers and the coagulation system highlights the complex pathophysiology of DIC, emphasizing the need for comprehensive biomarker profiling to improve early diagnosis and risk stratification.

D. Biomarkers of Fibrinolysis Activation

Fibrinolysis regulation plays a crucial role in disseminated intravascular coagulation (DIC), as an imbalance in this process can lead to either severe hemorrhagic complications or an increased risk of thrombosis. One of the key biomarkers in this context is soluble γ-thrombomodulin (sTM), which serves as an indicator of severe endothelial activation and has been closely linked to advanced stages of DIC [14].

In cases of excessive fibrinolysis, α2-antiplasmin, the primary inhibitor of plasmin-mediated fibrinolysis, becomes significantly depleted. This reduction weakens the body's ability to control plasmin activity, increasing the risk of severe bleeding [15]. On the other hand, when fibrinolysis is suppressed, levels of plasminogen activator inhibitor-1 (PAI-1) rise, preventing clot breakdown and predisposing patients to thrombotic complications [16]. These fibrinolytic markers provide valuable insights into the dynamic balance between clot formation and dissolution in DIC, helping to refine risk assessment and therapeutic strategies.

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E. Modern Multi-Biomarker Panels

Given the complexity of disseminated intravascular coagulation (DIC), relying on a single biomarker often fails to provide a comprehensive assessment of disease status. To address this limitation, multi-biomarker panels integrating various diagnostic indicators are being actively developed. A combination of thrombin-antithrombin (TAT) complexes, D-dimer, and plasminogen activator inhibitor-1 (PAI-1) has shown promise in evaluating hypercoagulation and thrombosis risk. Additionally, the use of von Willebrand factor (vWF) and hyaluronic acid provides valuable insights into endothelial dysfunction, while interleukin-6 (IL-6) and high-sensitivity C-reactive protein (hsCRP) help identify inflammatory triggers that contribute to DIC progression.

The implementation of such combined biomarker strategies significantly enhances diagnostic accuracy, offering a more detailed understanding of the underlying pathophysiological mechanisms. Compared to traditional coagulation tests, these multi-marker panels allow for a more precise evaluation of disease severity and progression. By incorporating these emerging biomarkers into clinical practice, physicians may be able to achieve earlier detection, improved risk stratification, and more personalized treatment approaches for patients with DIC. This shift toward biomarker-driven diagnostics represents a promising step in optimizing patient outcomes and guiding targeted therapeutic interventions.

V. Modern Approaches to Treatment

The treatment approach focuses on eliminating the underlying trigger, ensuring adequate microcirculation, and correcting coagulopathy. One of the primary strategies involves addressing the root cause, such as administering antibacterial therapy in cases of sepsis or performing surgical debridement to remove the source of infection [7]. In thrombotic forms of disseminated intravascular coagulation (DIC), anticoagulant therapy is employed, utilizing either unfractionated or low-molecular-weight heparin to prevent excessive clot formation and reduce the risk of microvascular thrombosis, which can lead to organ dysfunction 8. Careful monitoring of coagulation parameters is essential to balance the benefits of anticoagulation with the potential risk of bleeding complications.

Replacement therapy plays a crucial role in managing hypocoagulable forms of the syndrome, involving the administration of fresh frozen plasma or coagulation factor concentrates to restore hemostatic balance [9]. In severe cases of bleeding, platelet transfusions may be required to prevent life-threatening hemorrhagic complications. Additionally, antifibrinolytic therapy is indicated in cases of hyperfibrinolysis, where fibrinolysis inhibitors such as ε-aminocaproic acid are used to control excessive clot breakdown and stabilize clot formation [10]. However, antifibrinolytic agents must be used

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cautiously, as their inappropriate use in patients with an ongoing thrombotic process may exacerbate complications.

Supportive care is also a fundamental aspect of DIC management, with a focus on maintaining hemodynamic stability, ensuring adequate oxygenation, and optimizing organ perfusion. Intravenous fluid resuscitation and vasopressor support may be necessary in cases of shock, while mechanical ventilation may be required for patients with respiratory failure. Close monitoring of renal function is crucial, as DIC can lead to acute kidney injury, necessitating interventions such as renal replacement therapy in severe cases.

Despite these established strategies, there is no universal treatment algorithm for DIC syndrome, highlighting the necessity of an individualized approach tailored to each patient's condition. The complexity of DIC requires a multidisciplinary effort involving intensivists, hematologists, and other specialists to continuously assess the patient's response to therapy and adjust treatment accordingly. Future research is needed to refine therapeutic protocols and identify novel targeted therapies that can improve outcomes in patients with this challenging and life-threatening condition.

VI. Current Challenges and Research Perspectives

Despite extensive research, DIC syndrome remains a major issue in intensive care, requiring further investigation in several key areas.

Major Challenges:

1. Early diagnosis – existing laboratory tests lack specificity, and a unified screening algorithm for high-risk patients has not yet been developed.

2. Optimization of anticoagulant therapy – the role of heparin therapy in different forms of DIC remains unclear, and the development of new anticoagulants that minimize bleeding risk is increasingly important.

3. Investigation of anti-inflammatory therapy – cytokine modulation, including IL-6 and IL-1 inhibitors, may become a promising strategy in treating septic DIC syndrome [11].

4. Personalized medicine – creating individualized treatment protocols based on biomarkers and using artificial intelligence for DIC prediction could significantly improve patient outcomes [12].

VII. Conclusion

DIC syndrome remains one of the most complex medical conditions requiring a multidisciplinary approach.

Modern research is focused on improving diagnosis and treatment, but several issues remain unresolved.

The development of new therapeutic strategies, refinement of laboratory criteria, and implementation of personalized medicine are the key directions for future research.

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