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Review Article

The aetiology, pathophysiology, and laboratory diagnostic methods of disseminated intravascular coagulation: A review

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Abstract
One of the most difficult haematological emergencies that clinicians deal with is disseminated intravascular coagulation (DIC). Despite decades of investigation, this complex coagulopathy still causes fatalities and confounds clinicians. It paradoxically combines extensive thrombosis with catastrophic bleeding. Alarming fatality rates are shown by current epidemiological data, which range from 20% in obstetric cases to over 50% in DIC linked to sepsis. Our review explains the complex pathophysiology of DIC, summarises our current understanding of its complex origins, and critically assesses changing diagnostic paradigms. We specifically look at how new developments in scoring systems and biomarker science are changing how we approach this elusive ailment. This article serves as both a compass and an anchor for physicians negotiating the perilous waters of managing DIC. It acknowledges the enduring ambiguities that make DIC one of critical care's most powerful enemies while providing evidence-based guidance.

Keywords: *aetiology, pathophysiology, laboratory diagnostic methods, disseminated intravascular.*

1.Introduction
Disseminated intravascular coagulation is one of the most difficult medical disorders to diagnose and treat today. DIC is still referred to by many haematologists as "the great masquerader" of critical disease, having been first defined by Dupuy in 1834 as "thrombosis with haemorrhages" [1]. Its dual nature—DIC is never a major disease but rather a menacing companion to other life-threatening conditions makes it more frustrating. DIC can present in a variety of ways that continuously challenge clinical judgement, from the septic shock patient bleeding from all puncture sites to the trauma sufferer experiencing sudden renal failure due to microthrombi.

DIC has a significant worldwide burden that is often overlooked. According to recent registry statistics, DIC complicates about 1% of hospital admissions overall, with an incidence that can reach 30–50% in intensive care units. Even more concerning are the mortality rates, which range from 20% in obstetric cases to 50–80% in septic shock caused by multiple organ failure. These figures highlight the reasons behind the recent designation of DIC as a "medical emergency" by the International Society on Thrombosis and Haemostasis (ISTH), which calls for prompt identification and treatment [2].

The goal of this study is to give medical professionals a thorough yet useful framework for comprehending the causes, mechanisms, and diagnostic difficulties of DIC. We'll look at how recent studies are changing our understanding of this illness from a straightforward coagulation problem to a complicated thromboinflammatory disease. Above all, we will convert these insights into information that frontline practitioners can use.

2.Aetiological Factors
About half of all cases of DIC are caused by sepsis, making it the most common and fatal initiator. Examining meningococcal sepsis reveals the horrifying synergy between infection and coagulation; in this case, DIC progresses so quickly that patients may exhibit mucosal bleeding within hours as well as purpuric rash (from microthrombi). According

to the pathophysiology, inflammatory cytokines concurrently block natural anticoagulant pathways, while microbial components such as endotoxins cause monocytes to overexpress tissue factor [3].

Sobering lessons on infection-related DIC were learnt during the recent COVID-19 epidemic. According to Thachil et al. (2023), autopsy investigations of severe COVID-19 patients showed widespread microthrombi in the pulmonary vasculature, with viral particles directly triggering the contact activation pathway. This explains why, even prior to clinical deterioration, several individuals displayed high D-dimer elevations (>10 times normal) [4].

A distinct DIC contradiction is presented by major trauma. Plasmin activation causes excessive bleeding during the first "fibrinolytic phase" (the first three hours after damage), while microvascular blockage occurs during the "thrombotic phase" that follows (Davenport et al., 2023). The increasing use of viscoelastic tests such as TEG/ROTEM by trauma surgeons to direct transfusion techniques in real-time can be explained by this biphasic pattern [5].

Particular attention should be paid to crush injuries. We learnt during the 1995 Kobe earthquake that rhabdomyolysis releases large amounts of tissue factor in addition to myoglobin, which makes DIC a perfect storm. For crush victims, early coagulation screening is now required by contemporary disaster protocols [6].

Compared to septic DIC, cancer-associated DIC frequently progresses more slowly, but it might explode during chemotherapy. One well-known example is acute promyelocytic leukaemia (APL), in which the cancerous blasts produce both tissue factor and cancer procoagulant. Unfortunately, hemorrhagic complications prior to remission induction still account for up to 30% of APL-related mortality [7].

The story is different for solid tumours. Prostate cancer may cause a fibrinolytic-type DIC with bleeding predominance, whereas patients with pancreatic cancer often experience chronic DIC with venous thromboembolism. These variations highlight the necessity of management strategies tailored to cancer [3].

Obstetric DIC is one of the few clinical situations that need immediate attention. The rate at which coagulopathy develops in placental abruption can be astounding; in just 90 minutes, fibrinogen levels can drop from 4 g/L to less than 1 g/L. The picture of amniotic fluid embolism is even more frightening because anaphylactoid reactions can cause both cardiovascular failure and DIC at the same time [5].

Management has been completely transformed by recent developments in point-of-care diagnostics. In order to advise fibrinogen replacement during significant postpartum haemorrhage, several tertiary obstetric centres now employ viscoelastic testing. Massive transfusions have not been required as frequently during these situations thanks to the real-time monitoring [8].

3.Pathophysiology:

Although tissue factor (TF) has long been thought to be the "master switch" of DIC, we now know of other pathways as well. DIC under situations with platelet activation is explained by the powerful activation of factor XII by platelet-derived polyphosphates Circulating histones from neutrophil extracellular traps (NETs) directly encourage the production of thrombin in addition to causing endothelial injury The C5a-C5aR axis increases inflammation and coagulation, especially in sepsis. This is known as complement activation [9].

An unchecked activation of the coagulation cascade produces a perfect storm:
One factor Xa molecule can produce more than 1000 thrombin molecules in a “thrombin burst” Fibrin strand obstructing capillaries in several organs are visible under a microscope. In severe DIC, fibrinogen half-life drops from four days to just a few hours. The multisystem influence of DIC has been demonstrated using modern imaging techniques: Acute kidney damage is caused by fibrin thrombi in glomerular capillaries. ARDS is made worse by microthrombi in the pulmonary vasculature. Delirium in septic DIC may be explained by microvascular blockage [10].

4. Diagnostic Methods:

Expert medical professionals are aware of some "red flags": such as Rapidly developing petechiae or purpura., Unexpected bleeding from several locations, (venipuncture wounds, mucosal surfaces), Acral cyanosis, indicating microvascular thrombosis, Sudden organ dysfunction with no apparent cause But DIC can be really subtle. some cancer patients only exhibit increasing thrombocytopenia over several weeks [6].

Laboratory Results: Analysing the Trends

Thrombocytopenia (usually less than 100 x 10^9/L and decreasing) Extended PT/APTT (but circulating activated clotting factors may cause it to normalise later) Elevated D-dimer (often greater than 5–10 times the maximum limit) Because

fibrinogen is an acute phase reactant, its levels can be misleading and may stay normal for a long time even when active intake is occurring [11].

5.Conclusion:

Our diagnostic methods must change as our knowledge of DIC advances from a straightforward coagulation condition to a complex thromboinflammatory illness. Although there are still obstacles to overcome, developments in targeted medicines and point-of-care testing give promise for bettering the prognosis of this debilitating illness.

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