Review Article

Disseminated Intravascular Coagulation: Diagnosis in Complex Clinical Scenarios

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Abstract

Disseminated intravascular coagulation is a severe acquired syndrome characterized by systemic activation of coagulation, impaired fibrinolysis, endothelial injury, and progressive consumption of platelets and coagulation factors. Its pathophysiology explains the coexistence of thrombosis, bleeding, and organ dysfunction, making it one of the most complex hemostatic disorders in critical care. The condition is commonly triggered by sepsis, trauma, obstetric complications, solid and hematologic malignancies, transfusion reactions, acute pancreatitis, severe burns, and other major systemic insults. Although its mechanisms are shared across these conditions, the clinical presentation varies according to the underlying trigger, the balance between coagulation and fibrinolysis, and the stage of disease progression. The diagnosis of disseminated intravascular coagulation remains challenging because it evolves dynamically from subclinical forms to overt decompensated states. Hemorrhagic manifestations may include petechiae, ecchymoses, gastrointestinal bleeding, or intracranial hemorrhage, whereas thrombotic manifestations are often driven by microvascular thrombosis and may lead to renal, pulmonary, hepatic, or neurologic dysfunction. Laboratory evaluation relies on platelet count, prothrombin time, fibrinogen, D-dimer, and fibrin degradation

products, but no single marker is sufficient for diagnosis. For this reason, serial interpretation of laboratory trends and clinical context is essential. Scoring systems such as the International Society on Thrombosis and Haemostasis, Sepsis-Induced Coagulopathy, and Japanese Association for Acute Medicine criteria improve diagnostic structure, although their performance varies across populations. Differential diagnosis with thrombotic thrombocytopenic purpura, hemolytic uremic syndrome, liver disease-associated coagulopathy, trauma-related coagulopathy, and catastrophic antiphospholipid syndrome is essential. Early and context-adapted recognition remains critical for improving outcomes and guiding appropriate management.

Key words

Disseminated intravascular coagulation, coagulopathy, microvascular thrombosis, fibrinolysis, diagnostic scoring systems, critical illness.

Introduction

Disseminated intravascular coagulation is defined as an acquired, life-threatening condition characterized by systemic activation of coagulation, impaired fibrinolysis, and endothelial injury [1]. It is commonly triggered by underlying conditions such as infection, cancer, or obstetrical complications [2]. Although it is frequently under-recognized in critically ill patients, its presence has a substantial impact on prognosis and demands timely intervention [3].

Early recognition is especially important because it enables prompt intervention and may reduce mortality, particularly in conditions such as sepsis, in which disseminated intravascular coagulation can double the mortality rate [4]. In addition, machine learning models, including the XGB model, have shown promise for predicting disseminated intravascular coagulation in critically ill children, thereby supporting earlier intervention [5].

However, the diagnosis of disseminated intravascular coagulation remains challenging because of its dynamic progression from preclinical to overt stages. To address this complexity, the International Society on Thrombosis and Haemostasis has proposed a phase-based classification intended to improve early detection through recognition of subclinical abnormalities [1]. Even so, current diagnostic systems often fail to identify disseminated intravascular coagulation during its reversible phase, which contributes to suboptimal treatment outcomes. In this regard, biomarkers such as thrombin-antithrombin complex and antithrombin III have demonstrated potential for predicting disseminated intravascular coagulation before it progresses to an irreversible phase [6].

Current diagnostic approaches have therefore evolved toward updated and more individualized strategies. The revised criteria of the International Society on Thrombosis and Haemostasis incorporate a phase-based classification and individualized approaches, together with tools such as the sepsis-induced coagulopathy score for the detection of early-stage disease [1]. At the same time, emerging diagnostic biomarkers and machine learning models continue to be explored as strategies to enhance early detection and improve management [2, 5].

The aim of this article is to analyze the current diagnostic approach to disseminated intravascular coagulation in complex clinical scenarios, emphasizing its pathophysiological basis, diagnostic challenges, laboratory and scoring tools, and the role of emerging biomarkers and early recognition strategies in improving timely identification and clinical decision-making.

Methodology

This manuscript was developed as a structured narrative review aimed at providing an updated and clinically integrated analysis of disseminated intravascular coagulation, with particular emphasis on diagnostic challenges in complex clinical scenarios, early recognition strategies, laboratory assessment, and emerging diagnostic tools. The review was conducted in accordance with the SANRA (Scale for the Assessment of Narrative Review Articles) framework and followed a predefined methodological protocol established prior to literature screening. Given the marked clinical heterogeneity of disseminated intravascular coagulation, the variability in underlying triggers, and the differences in diagnostic performance across patient populations and clinical settings, a narrative interpretative synthesis was selected over quantitative pooling to integrate pathophysiological, hematologic, and clinical considerations into a coherent and clinically applicable framework. Special attention was given to early-phase recognition, the limitations of conventional diagnostic criteria, the use of scoring systems, and the potential contribution of novel biomarkers and predictive models in critically ill patients. The objective was to provide a structured synthesis capable of supporting clinical reasoning and diagnostic decision-making in patients with suspected disseminated intravascular coagulation across complex and high-risk settings.

A comprehensive literature search was conducted in PubMed, Scopus, and Web of Science, including peer-reviewed articles published in English or Spanish between January 2020 and December 2025. The final search was performed in March 2026. This timeframe was selected to capture recent advances in diagnostic criteria, phase-based classification systems, biomarker development, sepsis-related coagulopathy assessment, and the incorporation of predictive technologies such as machine learning models. Foundational studies were incorporated when necessary to contextualize the pathophysiological basis of disseminated intravascular coagulation, the historical evolution of diagnostic frameworks, or the development of current scoring systems. The search strategy combined MeSH and free-text terms using Boolean operators related to disseminated intravascular coagulation, sepsis-induced coagulopathy, diagnosis, early recognition, biomarkers, thrombin-antithrombin complex, antithrombin, fibrinolysis, endothelial injury, critical illness, scoring systems, and machine learning. Searches were conducted in titles and abstracts as well as indexed subject headings to maximize sensitivity.

The initial search yielded 125 records. After removal of duplicates, 92 articles remained for title and abstract screening. Of these, 50 underwent full-text evaluation, and 27 studies were included in the final synthesis. Selection was performed independently by two authors, with disagreements resolved through discussion and consensus. Exclusion criteria comprised non-peer-reviewed publications, isolated case reports, editorials without relevant diagnostic data, studies focused exclusively on treatment without diagnostic analysis, redundant datasets, and studies not directly addressing diagnostic criteria, laboratory evaluation, early recognition, or diagnostic performance in disseminated intravascular coagulation.

Eligible studies included randomized controlled trials, large observational cohorts, systematic reviews, meta-analyses, expert consensus statements, and contemporary international guidelines from hematology, intensive care, sepsis, and thrombosis societies. Priority was assigned to multicenter investigations, studies with clearly defined diagnostic criteria, and research evaluating early diagnostic performance, biomarker utility, predictive accuracy, and clinical applicability across different underlying conditions. Extracted variables included study design, population characteristics, underlying clinical trigger, diagnostic criteria used, laboratory markers

assessed, scoring system performance, timing of diagnosis, predictive models when applicable, and major clinical outcomes related to diagnostic timing or disease recognition. Methodological quality and internal validity were assessed narratively, considering risk of bias, sample size, follow-up duration when relevant, consistency of diagnostic definitions, and reproducibility of reported findings. In cases of conflicting evidence, greater interpretative weight was assigned to higher-level evidence and guideline-supported recommendations.

Reference lists of included studies were manually screened to identify additional relevant publications. Given its narrative design, this review is subject to potential selection bias and does not provide pooled quantitative estimates. Artificial intelligence-based tools were used exclusively to assist in literature organization and structural coherence, whereas critical appraisal, synthesis, and final interpretation were conducted independently by the authors to preserve methodological rigor.

Pathophysiological basis

Disseminated intravascular coagulation is initiated by systemic activation of the coagulation cascade, most triggered by underlying conditions such as sepsis, trauma, or malignancy [1, 2]. In this process, tissue factor expression on activated monocytes and endothelial cells plays a central role in the initiation of coagulation and promotes thrombin generation. As thrombin production becomes excessive, widespread fibrin deposition develops within the microvasculature, contributing to microvascular thrombosis. This phenomenon is further intensified by impaired fibrinolysis, since upregulation of plasminogen activator inhibitor type 1 inhibits fibrin breakdown [7, 8, 9].

At the same time, the ongoing activation of coagulation leads to progressive consumption of platelets and coagulation factors, resulting in their depletion and contributing to the development of a bleeding phenotype. This consumptive coagulopathy is a defining feature of disseminated intravascular coagulation, particularly in its hemorrhagic phenotype [1, 2]. In parallel, natural anticoagulant pathways, including the protein C system, become impaired as a consequence of endothelial injury and inflammation. The loss of these regulatory mechanisms further enhances thrombin generation and aggravates the prothrombotic state [7, 9].

In addition, dysregulation of fibrinolysis contributes to reduced fibrin breakdown, favoring persistent fibrin deposition and sustained microvascular thrombosis [9]. As a result, the physiological balance between coagulation and fibrinolysis is profoundly disrupted, giving rise to both thrombotic and hemorrhagic complications [1]. This pathophysiologic interaction explains the close link between microthrombosis, bleeding, and organ dysfunction. Microthrombosis promotes tissue ischemia and organ injury, as observed in sepsis-associated disseminated intravascular coagulation, where microthrombi impair the microcirculation. The simultaneous occurrence of bleeding and thrombosis further complicates clinical management and contributes to the high morbidity and mortality associated with this condition [2, 4].

Etiologies and triggering conditions

Sepsis is one of the leading causes of disseminated intravascular coagulation and has a substantial prevalence in intensive care units. It develops as a consequence of an excessive immune response to infection, which promotes systemic activation of coagulation together with impaired fibrinolysis. In septic patients, risk factors associated with the development of disseminated intravascular coagulation include low albumin levels, respiratory infections, and delayed antibiotic treatment. In this setting, sepsis-induced disseminated intravascular coagulation is closely associated with microvascular thrombosis, which contributes to

organ dysfunction and an unfavorable prognosis [4, 10].

Major trauma also represents an important triggering condition for disseminated intravascular coagulation. In these patients, the condition results from dysregulated innate immune responses and the release of damage-associated molecular patterns, which activate both coagulation and inflammation [7]. Trauma patients with low antithrombin activity are at particularly high risk of developing disseminated intravascular coagulation, a complication that is associated with increased mortality and multiple organ dysfunction syndrome. For this reason, early diagnosis in trauma settings is especially important, since disseminated intravascular coagulation predicts poor outcomes, including massive transfusion requirements and hospital death [11].

Obstetric complications constitute another well-recognized group of triggers. Conditions such as placental abruption and amniotic fluid embolism may precipitate disseminated intravascular coagulation through the release of tissue factors and other procoagulant substances into the maternal circulation. In these cases, management is primarily focused on controlling the underlying obstetric cause while providing supportive care tailored to the patient's clinical condition [2].

In the oncologic setting, advanced-stage solid tumors, particularly stage IV disease, may induce disseminated intravascular coagulation because of tumor-related procoagulant activity. In this context, recombinant thrombomodulin therapy has been explored as a treatment option and has shown potential benefits in selected cancer types, including colorectal and gynecological malignancies [12]. Hematologic malignancies, including leukemia and lymphoma, are also recognized triggers of disseminated intravascular coagulation because malignant cells release procoagulant factors that contribute to systemic coagulation activation. In these patients, management similarly depends on treatment of the underlying malignancy together with supportive care directed at controlling coagulation abnormalities [2].

Acute hemolytic transfusion reactions are another important cause, as they can lead to disseminated intravascular coagulation through the release of procoagulant substances and activation of the coagulation cascade. Because of this, prompt recognition and management of transfusion reactions are essential to prevent progression to disseminated intravascular coagulation. Acute pancreatitis, severe burns, and other major systemic insults may also trigger disseminated intravascular coagulation through systemic inflammation and endothelial injury. These conditions promote the release of pro-inflammatory cytokines and tissue factors, thereby enhancing coagulation activation and impairing fibrinolysis [2].

Clinical presentation and evolutionary patterns

Hemorrhagic disseminated intravascular coagulation is characterized by bleeding secondary to the consumption of coagulation factors and platelets. This may manifest as petechiae, ecchymoses, or more severe bleeding events such as gastrointestinal hemorrhage or intracranial bleeding [1, 2]. In critically ill patients, hemorrhagic complications are associated with increased mortality, particularly in those with coronavirus disease 2019, in whom gastrointestinal and pulmonary hemorrhages are common [13].

In contrast, thrombotic disseminated intravascular coagulation is characterized by microvascular thrombosis leading to organ dysfunction. This pattern is frequently observed in sepsis-induced disseminated intravascular coagulation, where microthrombi contribute to multiple organ failure [4, 8]. A similar prothrombotic profile has been described in patients with coronavirus disease 2019, in whom thrombotic events such as pulmonary embolism

and myocardial ischemia are prevalent, further highlighting the thrombotic nature of disseminated intravascular coagulation in certain clinical settings [13, 14].

The development of organ dysfunction in disseminated intravascular coagulation is primarily related to microvascular thrombosis. Among the most commonly affected organs are the kidneys, lungs, and brain, resulting in acute renal failure, respiratory failure, and neurological disturbances. In obstetric patients, disseminated intravascular coagulation is frequently associated with postpartum hemorrhage and may progress to multiple organ dysfunction syndrome [15].

From an evolutionary standpoint, acute disseminated intravascular coagulation is characterized by rapid progression of coagulation activation and therefore requires urgent intervention. It is commonly associated with severe underlying conditions such as sepsis or trauma [1, 17]. This acute form is marked by the simultaneous presence of bleeding and thrombotic complications, which necessitates a balanced approach to management [16].

By contrast, chronic or compensated disseminated intravascular coagulation progresses more slowly and may not initially present with overt clinical symptoms. It is often associated with malignancies and chronic inflammatory conditions [17]. In these cases, laboratory abnormalities may precede the appearance of clinical manifestations, making early detection more difficult [1].

Subclinical disseminated intravascular coagulation, also referred to as early-phase or compensated disseminated intravascular coagulation, is characterized by laboratory abnormalities in the absence of overt clinical symptoms. This phase may be identified through specific scoring systems, such as the sepsis-induced coagulopathy score [1]. In addition, atypical presentations may arise in particular contexts, such as coronavirus disease 2019, where the balance between thrombosis and bleeding is especially delicate [15].

Scoring systems and diagnostic criteria

The International Society on Thrombosis and Haemostasis scoring system is one of the most widely used tools for the diagnosis of disseminated intravascular coagulation, particularly in its overt form. It incorporates conventional parameters such as platelet count, prothrombin time-international normalized ratio, and fibrinogen levels. Its relevance has been reinforced by the 2025 update, which introduced a phase-based classification distinguishing pre-disseminated intravascular coagulation, early-phase disseminated intravascular coagulation, and overt disseminated intravascular coagulation, with criteria adapted to specific underlying conditions such as sepsis [1]. In septic patients, the International Society on Thrombosis and Haemostasis score has also been associated with mortality, although its predictive performance appears to vary across different populations [18].

In the specific context of sepsis, other diagnostic models have been developed to improve the identification of early coagulation abnormalities. The Sepsis-Induced Coagulopathy score, introduced by the International Society on Thrombosis and Haemostasis, was designed to detect early-phase disseminated intravascular coagulation in septic patients using readily available biomarkers. Likewise, both the Sepsis-Induced Coagulopathy score and the Japanese Association for Acute Medicine score have shown greater sensitivity than the International Society on Thrombosis and Haemostasis score for identifying coagulopathy and predicting outcomes in patients with sepsis. These characteristics make them especially useful for early recognition and for identifying patients who may benefit from timely therapeutic interventions in sepsis-associated disseminated intravascular coagulation [19, 20].

More broadly, scoring models such as the International Society on Thrombosis and

Haemostasis, Sepsis-Induced Coagulopathy, and Japanese Association for Acute Medicine systems have demonstrated value not only for diagnosing disseminated intravascular coagulation but also for estimating mortality risk, especially in septic populations [20, 21]. Although the International Society on Thrombosis and Haemostasis score is generally less sensitive, it offers greater specificity and may therefore be more useful for confirming disseminated intravascular coagulation at later stages [1]. In addition, the quick disseminated intravascular coagulation score system, based on simple parameters such as prothrombin time-international normalized ratio, platelet count, and D-dimer levels, has been proposed as a rapid diagnostic tool and has shown high diagnostic agreement [11].

The diagnostic performance of these scoring systems varies according to the clinical setting and the intended purpose of their use. In sepsis patients, the International Society on Thrombosis and Haemostasis score has shown a pooled sensitivity of 0.43 and a specificity of 0.81 for predicting mortality. By comparison, the Japanese Association for Acute Medicine and Sepsis-Induced Coagulopathy scores demonstrate higher sensitivity, at 0.73 and 0.71 respectively, although this occurs at the expense of lower specificity [20]. Consequently, the selection of a scoring model may depend on whether the clinical objective is early detection or confirmatory diagnosis within a particular setting [21].

Despite their usefulness, these scoring systems have important limitations in patients with baseline coagulation abnormalities. This is particularly relevant in populations such as patients with liver disease, in whom standard disseminated intravascular coagulation criteria may be difficult to interpret. Although the International Society on Thrombosis and Haemostasis score has been shown to predict mortality in patients with liver disease and low fibrinogen levels, its applicability may vary according to the underlying clinical condition. For this reason, adjustments or alternative criteria may be required to achieve more accurate diagnosis in these complex populations [22].

Differential diagnosis

Thrombotic thrombocytopenic purpura is characterized by a severe deficiency of ADAMTS13, a von Willebrand factor-cleaving protease, which leads to the formation of platelet-rich microvascular thrombi. Clinically, it presents with microangiopathic hemolytic anemia, severe thrombocytopenia, and ischemic end-organ injury. Confirmation of the diagnosis is based on measurement of ADAMTS13 activity, with levels below 10% being indicative of thrombotic thrombocytopenic purpura. Its treatment differs substantially from that of disseminated intravascular coagulation, as it relies on plasma exchange and immunosuppression, often including rituximab and caplacizumab [23, 24].

Hemolytic uremic syndrome shares certain features with thrombotic thrombocytopenic purpura, but it is primarily associated with renal impairment and is often triggered by infections, particularly Escherichia coli O157:H7. Although it may overlap clinically with other thrombotic microangiopathies, it usually presents with more pronounced renal dysfunction and less severe neurological involvement. Differentiation from disseminated intravascular coagulation depends on both clinical presentation and laboratory findings, including normal ADAMTS13 activity [25].

Liver disease-associated coagulopathy represents another important diagnostic consideration because hepatic dysfunction impairs the synthesis of coagulation factors and reduces the clearance of activated clotting factors. Distinguishing this condition from disseminated intravascular coagulation is particularly difficult because both may present with overlapping laboratory abnormalities, including thrombocytopenia and elevated D-dimer levels.

This distinction may be further complicated by the fact that ADAMTS13 activity is often reduced in liver disease, thereby also creating overlap with thrombotic thrombocytopenic purpura [23].

Acute traumatic coagulopathy arises in the setting of trauma-induced systemic inflammation and shock, both of which contribute to the development of coagulopathy. It is characterized by hyperfibrinolysis and consumption of coagulation factors, features that resemble disseminated intravascular coagulation. In these cases, differentiation depends largely on the clinical context and on specific laboratory parameters, including fibrinogen levels and thromboelastography findings [17].

Dilutional coagulopathy and abnormalities related to massive transfusion must also be distinguished from disseminated intravascular coagulation. These disorders occur because of dilution of coagulation factors and platelets during large-volume transfusion. Their differentiation from disseminated intravascular coagulation is based on the history of massive transfusion and on laboratory findings that reflect dilutional effects rather than systemic activation of coagulation [17].

Catastrophic antiphospholipid syndrome is another condition that may mimic disseminated intravascular coagulation because it is characterized by widespread thrombosis. However, it is specifically associated with antiphospholipid antibodies, and its distinction from disseminated intravascular coagulation relies on the identification of these antibodies together with clinical features suggestive of systemic lupus erythematosus or other autoimmune disorders [25].

More broadly, thrombotic microangiopathies encompass a group of disorders, including thrombotic thrombocytopenic purpura and hemolytic uremic syndrome, that are characterized by endothelial injury and microvascular thrombosis. The distinction among these entities depends on targeted laboratory testing, such as ADAMTS13 activity and complement factor assays, which help identify the specific underlying cause and thereby differentiate them from disseminated intravascular coagulation [25, 26].

Diagnostic challenges in complex clinical scenarios

In sepsis, coagulopathy often begins as sepsis-induced coagulopathy, a precursor to disseminated intravascular coagulation characterized by systemic activation of coagulation and suppressed fibrinolysis. In this setting, the sepsis-induced coagulopathy score, which incorporates platelet count, prothrombin time-international normalized ratio, and the Sequential Organ Failure Assessment score, is used to identify early-phase disseminated intravascular coagulation in septic patients [19]. This distinction is clinically important because the prevalence of disseminated intravascular coagulation among patients with sepsis ranges from 30% to 80%, with a substantial impact on mortality [4, 20]. Biomarkers such as D-dimer and fibrin degradation products are also relevant for early recognition, although their diagnostic performance is variable [27].

In patients with malignancy, disseminated intravascular coagulation often presents as a chronic process in which thrombotic complications are more frequent than hemorrhagic manifestations [2]. Although the International Society on Thrombosis and Haemostasis criteria are commonly applied in this context, diagnosis is often complicated by the marked heterogeneity of cancer types and treatment-related factors [1].

In obstetric patients, disseminated intravascular coagulation is frequently associated with conditions such as placental abruption and amniotic fluid embolism, both of which require rapid diagnosis and management because of their acute and potentially catastrophic presentation.

In this context, the International Society on Thrombosis and Haemostasis criteria have been adapted for obstetric patients, with particular emphasis on rapid changes in coagulation parameters [2].

The diagnosis of disseminated intravascular coagulation in trauma and major surgery is likewise challenging. Trauma-induced coagulopathy may initially present with a bleeding phenotype and subsequently progress toward a prothrombotic state, which complicates its differentiation from disseminated intravascular coagulation. In addition, the management of trauma-induced coagulopathy often focuses on massive blood loss and tissue damage, factors that may either mask or mimic the manifestations of disseminated intravascular coagulation [19].

Advanced liver disease represents another particularly difficult diagnostic scenario because baseline hepatic dysfunction is already associated with coagulopathy and altered production of coagulation factors [2]. As a result, distinguishing liver disease-related coagulopathy from disseminated intravascular coagulation requires especially careful interpretation of coagulation studies and their clinical context [1].

In intensive care settings, diagnosis becomes even more complex because multiple organ dysfunction and overlapping coagulopathies frequently coexist. In these patients, the use of both sepsis-induced coagulopathy and International Society on Thrombosis and Haemostasis criteria can help identify individuals at risk of disseminated intravascular coagulation [19, 27].

More broadly, disseminated intravascular coagulation shares important features with other coagulopathies such as sepsis-induced coagulopathy and trauma-induced coagulopathy, making differential diagnosis difficult. This phenotypic overlap highlights the need for a comprehensive diagnostic approach that integrates multiple criteria rather than relying on a single framework [19].

Both underdiagnosis and overdiagnosis remain important concerns. Underdiagnosis is favored by the subclinical nature of early-phase disseminated intravascular coagulation, whereas overdiagnosis may result from applying diagnostic criteria too broadly without adequate consideration of the underlying disorder [17]. In this regard, the use of tailored diagnostic criteria and scoring systems such as sepsis-induced coagulopathy and Japanese Association for Acute Medicine-disseminated intravascular coagulation may improve diagnostic accuracy and ultimately contribute to better patient outcomes [20].

Conclusions

Disseminated intravascular coagulation is a dynamic and multifactorial syndrome in which systemic coagulation activation, impaired fibrinolysis, endothelial injury, and consumption of platelets and coagulation factors lead to the simultaneous risk of thrombosis, bleeding, and organ dysfunction, which explains its high morbidity and mortality across diverse clinical settings.

Its diagnosis remains particularly challenging because clinical presentation and laboratory findings vary according to the underlying trigger, phase of disease, and coexistence of other coagulopathies; therefore, accurate recognition requires integration of clinical context, serial laboratory trends, and the appropriate use of scoring systems such as the International Society on Thrombosis and Haemostasis, Sepsis-Induced Coagulopathy, and Japanese Association for Acute Medicine models.

Early and context-adapted identification of disseminated intravascular coagulation is essential to improve outcomes, especially in high-risk scenarios such as sepsis, trauma, malignancy, obstetric emergencies, liver disease, and critical care, where both underdiagnosis and

overdiagnosis may delay appropriate management or lead to misdirected treatment decisions.

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