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Renal morphologic lesions in disseminated intravascular coagulation; a letter to the editor on recent findings

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Abstract
Disseminated intravascular coagulation (DIC)-associated renal lesions may resemble those seen in thrombotic microangiopathy, which includes conditions like hemolytic-uremic syndrome and thrombotic thrombocytopenic purpura. Thrombotic microangiopathy is characterized by widespread microthrombi formation in small blood vessels, leading to organ damage, including the kidneys. Bleeding tendencies in DIC can manifest in the renal interstitium, leading to hemorrhage and edema within the kidney tissue. DIC can also cause damage to the renal tubules, impairing their ability to reabsorb electrolytes and maintain fluid balance. This can result in electrolyte imbalances and further exacerbate kidney dysfunction. In severe cases, DIC can cause renal infarction.

Keywords: Disseminated intravascular coagulation, Microthrombi, Microangiopathic hemolysis, Hemolytic-uremic syndrome
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Introduction

Disseminated intravascular coagulation (DIC) is a serious disorder considered by systemic activation of blood coagulation, leading to the generation and deposition of fibrin. This results in the formation of microvascular thrombi in several organs, interposing to multiple organ dysfunction syndrome (1). Disseminated intravascular coagulation can be caused by various factors, including inflammation, infection, cancer, major trauma, severe necrotizing pancreatitis, and sepsis. The underlying mechanism involves the over-activation of proteins that control blood clotting, leading to the formation of small blood clots that can block normal blood supply to organs such as the liver, brain, or kidneys, causing damage and injury (2,3). Disseminated intravascular coagulation can manifest as both acute and chronic forms, with acute DIC developing when there is abrupt exposure of blood to procoagulants, overwhelming the compensatory hemostatic mechanisms and resulting in severe consumptive coagulopathy and hemorrhage (4).

Search strategy

For this review, we searched PubMed, Web of Science, EBSCO, Scopus, Google Scholar, Directory of Open Access Journals (DOAJ) and Embase, using different keywords including disseminated intravascular coagulation, microthrombi, microangiopathic hemolysis and hemolytic-uremic syndrome.

Mechanistic impact of DIC

The mechanistic impact of DIC involves the overactivation of proteins that control blood clotting, leading to the formation of small blood clots that can block normal blood supply to organs such as the liver, brain, or kidneys, causing damage and injury (5).

The pathophysiology of DIC involves the consumption of clotting agents and platelets, which may develop in life-threatening hemorrhage. Disseminated intravascular coagulation can be initiated by various factors, including inflammation, infection, cancer, major trauma, severe necrotizing pancreatitis, and sepsis. The fibrinolytic pathway is also activated in this syndrome. Additionally, triggering of endothelial cells by the cytokines and also disturbed microvascular blood flow leads to the release of tissue plasminogen activator (tPA) by the endothelial cells (1). The delayed dissolution of fibrin polymers by fibrinolysis could lead to the mechanical disruption of RBCs (red blood cells), and production of schistocytes and mild intravascular hemolysis. Inflammation plays a significant role in the progress of DIC. During DIC, both blood cells and vascular endothelial cells, together with coagulation factors, play key roles in the underlying pathophysiology (4,6).

Renal histopathology features of DIC

Renal pathological lesions in DIC include thrombosis, microangiopathic hemolysis, glomerular capillary

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Implication for health policy/practice/research/ medical education

Kidney pathologic lesions in disseminated intravascular coagulation can be of prognostic significance, but their impact on prognosis is intertwined with various factors, including the extent of kidney damage, the underlying cause of disseminated intravascular coagulation (DIC), the timeliness of diagnosis and treatment, and the presence of complications. Management should be tailored to address both the coagulation abnormalities and the underlying condition, with the goal of minimizing further kidney damage and improving overall patient outcomes. Close monitoring of renal function and timely intervention are essential in managing DIC-related kidney lesions and optimizing prognosis.

thrombi, and interstitial fibrosis/tissue injury. Thrombosis can occur throughout the kidney, including arteries, veins, and small vessels within the parenchyma, leading to ischemia and necrosis of tissues (7,8). Microangiopathic hemolysis refers to damage to red blood cells and platelets due to activation of the clotting cascade, resulting in intracellular hemoglobin release that can cause renal inflammation and tubulointerstitial injury. Glomerular thrombi are rare but may form when large amounts of fibrin and platelet-rich clots obstruct glomeruli, causing acute kidney injury. Interstitial fibrosis/tissue injury occurs as a result of chronic inflammation and scarring from repeated cycles of ischemia and reperfusion, leading to progressive loss of functional nephron mass over time. These lesions contribute to the progression of DIC and its associated complications such as acute renal failure, chronic renal failure, and end-stage kidney failure (9,10)

Prognostic significance of kidney pathology in DIC

The prognostic significance of kidney pathologic lesions in DIC can vary depending on the seriousness of renal involvement and the underlying cause of DIC. Here are some key points to consider. The extent of kidney damage and the specific lesions present can have a significant impact on prognosis (7). Mild renal involvement with reversible changes may not carry as poor a prognosis as severe, irreversible kidney damage such as renal infarction. The prognosis of DIC is often closely tied to the underlying condition that triggered it. For example, if DIC is secondary to a severe infection (sepsis) and the infection is successfully treated, the prognosis may be more favorable. Conversely, if DIC is associated with advanced malignancy, the prognosis may be more guarded. Kidney pathologic lesions can lead to complications such as acute kidney injury, electrolyte imbalances, and fluid overload (10-14). The development of these complications can further worsen the prognosis and impact overall patient health. Early diagnosis and prompt treatment of both the underlying cause and the coagulation abnormalities in DIC are crucial for improving outcomes. Delayed diagnosis and treatment can worsen kidney damage and overall prognosis (15,16).

Treatment of renal involvement in DIC

The treatment approach should be individualized based on the patient's clinical status, the severity of renal involvement, and the underlying cause of DIC. Timely and comprehensive management of both the coagulation abnormalities and kidney dysfunction is essential to improve outcomes for patients with DIC-associated renal involvement (11,17).

Conclusion

Disseminated intravascular coagulation is a serious disorder in which the proteins that control blood clotting become overactive, leading to widespread microvascular fibrin thrombi, which can result in multi-organ dysfunction syndrome from tissue ischemia. Thus, condition is not itself a specific illness, but rather a complication or an effect of the progression of other disease, and is continuously secondary to an underlying illness.

Conflicts of interest

The authors declare that she has no competing interests.

Disclosing the use of generative AI

The authors used Perplexity and ChatGPT-3 during the preparation of the manuscript for editing and improving the English language. After utilizing of Perplexity and ChatGPT-3, we reviewed and edited the content, taking full responsibility for the content of the publication.

Ethical issues

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

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