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Disseminated Intravascular Coagulation: What's New?

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Systemic activation of coagulation may occur in a variety of disorders, most of which are: associated with inflammatory activation. This coagulation activation may only be detectable by measuring sensitive molecular markers for activation of coagulation factors and pathways, but can become clinically manifest as changes in routine coagulation tests may occur [1]. The spectrum of clinically manifest coagulation activation ranges from a small decrease in platelet count and sub-clinical prolongation of global clotting times, to fulminant disseminated intravascular coagulation (DIC), which is characterized by simultaneous widespread microvascular thrombosis and profuse bleeding from various sites.

DIC is caused by widespread and ongoing activation of coagulation, leading to vascular or microvascular fibrin deposition; thereby compromising an adequate blood supply to various organs, which can contribute to organ failure [2]. Because of ongoing activation of the coagulation system and other factors (eg, impaired synthesis and increased degradation), exhaustion of coagulation, protease inhibitors, and platelets may occur. This situation could result in serious bleeding, particularly in patients who are at risk for major blood loss, such as perioperative patients or trauma patients. Simultaneous thrombosis and bleeding could occur, resulting in a difficult problem for the clinician.

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Clinical setting

DIC is not a disease in itself, but is always secondary to an underlying disorder. The underlying disorders most commonly known to be associated with DIC are listed in Box 1.

Bacterial infection, in particular septicemia, is often associated with DIC [3,4]. However, systemic infections with other microorganisms, such as viruses and parasites, may lead to DIC as well. Endotoxins (eg, from Gram negative bacteria) or exotoxins (eg, staphylococcal alpha toxin) may contribute to the development of DIC in patients with infections. These components may cause a generalized inflammatory response, characterized by the systemic occurrence of cytokines. Cytokines are mainly produced by activated mononuclear cells and endothelial cells and are responsible for the derangement of the coagulation system in DIC [5].

Severe trauma is another clinical condition frequently associated with DIC [6]. A combination of mechanisms—including release of tissue material (fat, phospholipids) into the circulation, hemolysis, and endothelial damage—may contribute to the systemic activation of coagulation. There is solid evidence that cytokines play a pivotal role in the occurrence of DIC in trauma patients as well. In fact, systemic cytokine patterns have been shown to be virtually identical in trauma patients and septic patients [7].

Box 1. Clinical conditions that may be associated with disseminated intravascular coagulation (DIC)

- Sepsis/severe infection (any microorganism)
- Trauma (eg, polytrauma, neurotrauma, fat embolism)
- Organ destruction (eg, severe pancreatitis)
- Malignancy
  - –solid tumors
  - –myeloproliferative/lymphoproliferative malignancies
- Obstetrical calamities
  - –amniotic fluid embolism
  - –abruptio placentae
- Vascular abnormalities
  - –Kasabach-Merritt Syndrome
  - –large vascular aneurysms
- Severe hepatic failure
- Severe toxic or immunologic reactions
  - –snake bites
  - –recreational drugs
  - –transfusion reactions
  - –transplant rejection

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