Disseminated Intravascular Coagulation (DIC) Assessment

Disseminated Intravascular Coagulation (DIC) is an abnormal initiation of the clotting cascade due to an underlying disease or event. This results in the formation of a large number of small clots throughout the body. These clots then become trapped in smaller blood vessels, obstructing blood flow. As this condition progresses, it consumes clotting factors, fibrinogen, and platelets at a faster rate than the body can produce. This lack of clotting factors and after effects of the disorder is what we commonly find during the patient assessment. Assessment and quick identification of the patient at risk for DIC is key, because DIC has a high mortality rate. Some causes of DIC include any infection of the blood causing sepsis, amniotic fluid or fat emboli, transfusion and transplant reactions, severe burns, and toxins like snake or spider venoms.



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Mechanism

Improper Initiation of Clotting Cascade

Initiated Clogs on Cascading-steps

DIC is not a disease, but rather the result of another disease process or disorder that causes the improper initiation of the clotting cascade. Activation of the coagulation cascade yields thrombin that converts fibrinogen to fibrin, which leads to microvascular thrombi in various organs. To combat this, the fibrinolytic system is activated to break down fibrinogen and fibrin, resulting in the widespread clotting with resultant bleeding that is characteristic of DIC.

Signs and Symptoms

Systemic Bleeding

Bleeding all over

Patients classically bleed from every opening. Typically one will find bleeding around IV sites first when quickly reassessing patients. Patients may also present with hematuria, hematemesis, persistent bleeding from gums and from previous wounds. Be sure to assess for bleeding that cannot be immediately seen like gastrointestinal and genitourinary bleeding.

Petechiae, Purpura, Ecchymosis

Tiki-mask, Purple-cat, Eggy-moose

As small capillaries burst, they typically clot and heal, but these patients will present with varying degrees of small pinpoint purple spots (petechiae), large areas of bruise like discoloration (purpura), and large easy bruising areas of skin (ecchymosis).

Change in LOC

Delta Halo

Patients often lose a large amount of blood and that decreases the number of circulating red blood cells available to properly oxygenate the tissues. Changes in mental status, lethargy, dizziness, headache occur, because the brain is poorly perfused.

Increased PT and PTT

Up-arrow PT and PTT hourglass

As clotting factors are consumed the PT and PTT lab times are prolonged. These labs measure the propensity for the activation of the clotting factors in seconds. As the times are prolonged (or higher), the longer the activation takes, meaning more bleeding will be noted.

Cyanosis

Cyan-crayon

Patients who have hypovolemia associated with this condition may present with bluish colored skin, which is termed cyanosis or pallor. This may present with associated dyspnea, tachycardia, or orthopnea.

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Increased Fibrin Degradation Products (FDP)

Up-arrow Fiber Degradation Products

As the fibrin and fibrinogen that formed the clot are broken down the remnants are called fibrin degradation products (FDP) or fibrin split products. The process of breaking down fibrin in a clot is termed fibrinolysis. In DIC there is excessive clot formation and as they break down, these FDP/FSP’s can be measured in increased levels in the serum.

Decreased Platelets and Fibrinogen

Down-arrow Plate and Fiber-jam

As DIC progresses, platelets are consumed in microthrombi all over the body. As such, low platelet counts (thrombocytopenia) will be seen on labs. Also, fibrinogen levels will be decreased because they have been activated to fibrin by thrombin in order to form meshworks around weak platelet plugs.

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