



# Disseminated Intravascular Coagulation

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Oncologic emergencies, such as disseminated intravascular coagulation (DIC), can cause significant morbidity and mortality. DIC is a frequent thrombotic event that arises in the cancer population; in fact, patients with acute promyelocytic leukemia (APL) have an 85% risk of developing DIC (Ezzone, 2000; Holmes-Gobel, 2000). DIC is a coagulation disorder that can be caused by, for example, cancer or infection. DIC prompts overstimulation of the normal clotting cascade and results in simultaneous thrombosis and hemorrhage (Ezzone).

Intrinsic and extrinsic pathways are responsible for clotting. The intrinsic pathway is initiated by endothelial cell damage. Sepsis, a phenomena that disrupts the endothelial cell membrane, is one type of intrinsic-triggering event. The extrinsic pathway is initiated during tissue injury, and triggering events include malignancy, trauma, or obstetric complications (Ezzone, 2000; Tan, 2002). Both the extrinsic and intrinsic pathways lead to the common pathway where fibrinogen is converted to fibrin; blood clotting, which balances homeostasis, subsequently occurs (Ezzone).

DIC develops when the clotting cascade becomes disrupted from the normal homeostatic balance. Activation of the clotting cascade causes plasmin and thrombin to circulate, which, in turn, simultaneously induces bleeding and clot formation. DIC results from abnormal procoagulant activation and consumption of clotting factors. Thrombosis and organ damage occur because of excess platelet aggregation. Excess fibrin clots capture the platelets and produce thrombosis with impaired organ perfusion. Platelet aggregation leads to thrombocytopenia and additional consumption of clotting factors (Holmes-Gobel, 2000).

Bleeding can occur in patients with DIC because excess thrombin facilitates the conversion of plasminogen to plasmin. The conversion of plasmin produces fibrinolysis, which leads to a rise in fibrin split products

(FSPs). FSPs have strong procoagulant activity, thus causing hemorrhage (Holmes-Gobel, 2000).

## Case Study

Mr. M is a 53-year-old male admitted to the oncology unit with newly diagnosed APL. He had a central venous catheter placed and began idarubicin and cytarabine yesterday. He offers no complaints at the beginning of the 12-hour shift. His vital signs are stable, and his laboratory values, drawn this morning, are hemoglobin 10 g/dl, platelet count 50,000/mm<sup>3</sup>, fibrinogen 160 mg/dl, FSPs 50 ug/ml, and international normalization ratio (INR) 1.6. A complete blood count and coagulation panel, including fibrinogen and FSP, are ordered to be drawn at 5 pm. Upon entering the patient's room to measure his 12 pm vital signs, the nurse found Mr. M to be disoriented and lethargic. A large pool of blood is on his bed and shirt. Mr. M also has epistaxis, and a large ecchymotic area the size of an orange is present on his right thigh. After reporting these findings, "stat" laboratory studies reveal hemoglobin 8 g/dl, platelet count 22,000/mm<sup>3</sup>, prothrombin time (PT) 15.8 seconds, fibrinogen 96 mg/dl, FSP 100 ug/ml, and INR 2.0. Mr. M's vital signs reveal a temperature of 100°F, blood pressure of 90/50 mm/Hg, pulse 120 beats per minute, respiration rate 22 breaths per minute, and oxygen saturation, as measured by pulse oximetry, 90%.

1. What event most likely precipitated DIC in Mr. M?
  - a. Nutritional deficiencies
  - b. Sepsis
  - c. Placement of a new central venous catheter
  - d. Initiation of chemotherapy
2. All of the following are causes of DIC except
  - a. Trauma.
  - b. Sepsis.

- c. Renal failure.
  - d. Malignancy.
3. All of the following are signs and symptoms of DIC except
    - a. Ecchymosis.
    - b. Hemoptysis.
    - c. Slow, irregular heart rhythm.
    - d. Scleral hemorrhage.
  4. After a thorough assessment and oxygen therapy, a care plan is developed in collaboration with Mr. M's oncologist. Which of the following plans is most appropriate for this patient?
    - a. Discontinue the chemotherapy, initiate a workup of the fever to determine its cause, and administer blood products.
    - b. Continue chemotherapy and begin a normal saline solution at 200 ml per hour, work up the fever and administer antibiotics if indicated, administer blood products, and monitor the patient's complete blood count, metabolic profile, coagulation studies, and liver panel.
    - c. Initiate heparin at 2,200 units per hour and monitor coagulation studies, including PT, partial thromboplastin time (PTT), INR, FSP, and fibrinogen, every six hours.
    - d. Administer furosemide 80 mg IV bolus (push) now, monitor complete blood count and coagulation panels daily, initiate thrombocytopenic precautions, and closely monitor intake and output.
  5. Which series of blood tests can indicate the presence of DIC?
    - a. Elevated PT and fibrinogen and presence of schistocytes.

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