

DIC (Disseminated Intravascular Coagulation aka Coagulation Consumption Coagulopathy)

DIC – or coagulation consumption coagulopathy is an abnormal response of the normal cascade caused by an underlying disease or condition. It is not a disease but rather a complication resulting from an underlying disease or disease process.

Etiology and Pathophysiology:

Causes include: Infection-especially gram negative, bacterial, viral, fungal (all leading to endotoxins), obstetric complications, heat stroke, shock-especially septic, cirrhosis, fat embolism, cardiac arrest, incompatible blood transfusion, transplant rejection, neoplastic disease (cancer-necrosis factor), poisonous snake bite, trauma-especially crush or burns, organ necrosis, hypothermia

Regardless of the cause, DIC is a serious bleeding disorder resulting from initiated and accelerated clotting (**thrombosis**) causing generalized activation of **prothrombin** and therefore an excess of **thrombin** (the most powerful coagulant catalizer in conversion of **fibrinogen** to **fibrin**). The excess **thrombin** converts **fibrinogen** (a sticky protein) to **fibrin** (a matrix material resembling a spider web) producing fibrin clots in the microcirculation. The sticky fibrin matrix promotes platelet disposition in capillaries and arterioles leading to **thrombosis** and ultimately causing small vessel occlusion. Clots or thrombosis within blood vessels lead to decreased perfusion thus decreased supply of oxygen and other vital nutrients to tissues distal to the clot. The decreased perfusion leads to ischemia, injury and without treatment, loss of cell function. The ischemic cells revert to anaerobic metabolism with resulting increase in lactic acid causing metabolic acidosis and eventually necrosis/cell death. This excessive clotting occurring within the microcirculation usually affects the kidneys and extremities but can also affect the lungs, brain, GI tract, adrenal and pituitary glands.

The excessive clotting process in turn leads to decreased available clotting factors (**hypoprothrombinemia**), platelets (**thrombocytopenia**) and deficiencies in factors V and VIII resulting in hemorrhage. The **thrombin** circulating in the blood vessels also activates the **fibrinolytic system** (trying to maintain homeostasis) which lyses or dissolves the newly formed clots breaking them down into **fibrin degradation products** or **FDP's also known as FSP's- fibrin split products (anticoagulant byproducts)** which actually inhibit clotting.

Concisely stated, **DIC** is a paradox of profuse bleeding resulting from depletion of platelets and clotting factors.

***Anatomy Overview*-See Handout**

Clinical manifestations-signs and symptoms: – bleeding or oozing from multiple sites, petechiae, ecchymoses, hematomas, gingival bleeding, bleeding from sites of surgical or invasive procedures, hematuria, intracranial bleeding, rapid onset of unexplained bruising, micro-thrombosis leading to bluish discoloration of fingers/toes (acrocyanosis). Other related symptoms include dyspnea, oliguria, seizures, coma, severe muscle, back and abdominal pain and failure of major organ systems.

Complications: Hemorrhage, Shock, MODS-multi system organ dysfunction syndrome

Diagnostic Studies: Lab – increased PT, PTT, bleeding time, FSP's/FDP's, **D-dimer** (most specific marker for the degree of DIC as it is a specific polymer resulting from the breakdown of fibrin and not fibrinogen), platelets and fibrinogen levels decreased (these are the raw materials used for clotting) Increased BUN and serum creatinine.

Treatment Modalities – aimed at treating the cause and replenishing factors needed for clotting. We must administer the specific agents necessary to replenish supplies:

Platelets – are given to correct thrombocytopenia

Cryoprecipitate – replaces factor VIII and fibrinogen

FFP or fresh frozen plasma- replaces all clotting factors except platelets and provides a source of antithrombin

Packed RBC's-provide oxygen carrying capacity of hemoglobin to cells

*May need to infuse heparin to prevent further micro occlusion of small blood vessels

Nursing Diagnosis and related interventions:

Deficient fluid volume: Hemorrhage R/T depletion of clotting factors

Fear R/T threat to well-being

Ineffective protection R/T abnormal clotting mechanisms

Risk for ineffective tissue perfusion: peripheral: Risk factors: hypovolemia from profuse bleeding, formation of microemboli in vascular system

- Monitor patient's cardiac, respiratory, and neurological status frequently
- Assess breath sounds, vital signs, cardiac rhythm
- Assess for signs of hemorrhage and hypovolemic shock
- Inspect skin and mucous membranes for signs of bleeding-observe color and capillary refill
- Check IV and puncture sites frequently, apply pressure to injections sites for at least 15 minutes
- Administer supplemental oxygen-assess ABG's and O2 sats.
- Minimize oxygen demands, keep patient calm and quiet in semi-Fowler's position to maximize lung expansion
- Monitor diagnostic lab (especially PT, PTT, FDP's. platelets, D-Dimer, BUN/creatinine) and administer blood, FFP or platelets as ordered-watch closely for transfusion reactions and signs of fluid overload

- Monitor I & O hourly
- Monitor for potential complications including pulmonary emboli, acute tubular necrosis or multiple organ dysfunction syndrome
- Provide emotional support to patient and his family