Disseminated Intravascular Coagulation

Disseminated intravascular coagulation is characterized by the widespread activation of coagulation, which results in the intravascular formation of fibrin and ultimately thrombotic occlusion of small and midsize vessels which can compromise the blood supply to organs and, in conjunction with hemodynamic and metabolic derangements, may contribute to multi-organ failure; at the same time, the use and subsequent depletion of platelets and coagulation proteins may induce severe bleeding

ANESTHETIC CONSIDERATIONS:

- A condition carrying a high mortality rate – requires urgent evaluation & management
- There is a need to search for, and aggressively treat, underlying cause
    - Sepsis, trauma (massive tissue injury, burns, head injury, fat embolism), malignancy, obstetrical (AFE, abruption, fetal death in utero, PIH), vascular disorders, toxins, immunologic
- Potentially **massive hemorrhage** and **thrombosis** requiring resuscitation and **transfusion**
- Perioperative complications:
    - Acute hemorrhage (GI, hemothorax, cardiac tamponade, ICH & surgical site)
    - Microthrombotic / ischemic end-organ injury (ARF, progressive hepatocellular injury)
- Post-op disposition: ICU

ANESTHETIC GOALS:

- Identify and aggressively treat underlying cause
- Replace consumed coagulation factors and platelets
- Support multisystem organ failure

HISTORY

- Basic anesthetic history (AMPLE)
- Underlying condition

PHYSICAL

- Primary ATLS survey
    - **VITALs**
    - Ease of intubation
    - Airway bleeding / engorged & friable mucous membranes
    - Hypotension / shock / signs of hypoperfusion
    - Respiratory distress / tachypnea
- Secondary ATLS survey
    - Evidence of bleeding
    - Petechiae

INVESTIGATIONS

- **Labs**
    - No single diagnostic test
    - CBC and differential
        - Thrombocytopenia - sensitive early marker, initial PLT count < 100 or rapid decline
        - Schistocytes on peripheral smear
    - Coagulation profile (INR & PTT increased)
    - FDPs increased
    - Inhibitors of coagulation (e.g. antithrombin III, protein C) are decreased
    - Fibrinogen (acute phase reactant) may remain normal until late (not very sensitive)
    - LFTs (hepatic involvement)
    - Electrolytes, BUN / creatinine (renal perfusion – ARF d/t ATN)
    - Stool for occult blood PRN
- **Imaging**
    - EKG - ST-T changes / myocardial ischemia
    - ECHO PRN
- **Special**
    - Consult blood bank PRN
    - Consult hematology PRN
    - Consult ICU PRN

OPTIMIZATION

- Consider canceling if patient is unstable
- Correction of underlying problem **most important!**
- ABCs
    - General support of vascular volume and oxygen transport is essential
        - ETT and PPV usually required
    - O_2, IV access and fluid resuscitation
    - Avoid hypothermia
- Specific treatment of DIC only if:
    - Uncontrolled bleeding
    - End-organ damage from microthrombi

- Anticoagulation with **heparin** is controversial and no longer routinely done
  - Small uncontrolled trials showed benefit but controlled clinical trials did not
  - If heparin is used, low doses are given (300 to 500 U per hour) as a continuous infusion
  - Low-molecular weight heparin may also be used as an alternative to unfractionated heparin
  - Novel, antithrombin III-independent inhibitors of thrombin, such as **desirudin** and related compounds, might be more effective than heparin, and experimental studies have had promising results
- Antifibrinolytics not generally recommended
  - A clear exception might be made in the case of patients with primary or secondary hyperfibrinolysis, such as those with the coagulopathy associated with acute promyelocytic leukemia and some patients with DIC in association with cancer
- Blood products
  - No evidence supporting administration if not bleeding
  - pRBCs as required if significant hemorrhage
  - PLT concentrates to keep PLT > 20 (some recommend > 50)
  - FFP +/- cryoprecipitate if hypofibrinogenemic
  - Consider AT III concentrates
  - Factor VIIa for massive hemorrhage
- Medicine / Hematology / ICU Consult

ANESTHETIC OPTIONS
- Usually requires GETA
- Neuraxial techniques contraindicated

ANESTHETIC SETUP
- **Drugs**
  - Standard emergency drugs
  - Inotropes / vasopressors available
- **Equipment**
  - Standard CAS monitors
  - Multiple large bore IVs
  - Temperature probe
  - Arterial line + CVC +/- PAC / TEE
  - Foley catheter
  - Hotline, body warmer, rapid infuser

MANAGEMENT OF ANESTHESIA
- **Induction**
  - Consider full stomach
  - Septic patients likely hypovolemic and may have some degree of myocardial depression
- **Maintenance**
  - Coagulation monitoring frequently
  - Transfusion of blood components according to clinical situation and coagulation status
- **Emergence**
  - Cardiovascular and pulmonary insufficiency may be present if acute DIC
  - Consider postoperative mechanical ventilation

DISPOSITION & MONITORING
- ICU

COMPLICATIONS
- Uncontrolled hemorrhage: patients may bleed from areas of even minor tissue trauma
- Complications of massive transfusion
- End-organ damage from microthrombosis
- Concomitant medical problems may present life-threatening situations

PATHOPHYSIOLOGY
- DIC is a consumptive coagulopathy
- DIC is triggered by the appearance of tissue factor (thromboplastin) activity in the circulation in amounts sufficient to overwhelm the mechanisms that normally restrain and localize clot formation
- The accelerated process of clot formation causes both **tissue ischemia** and, ultimately, **critical depletion of platelets and factors leading to bleeding**

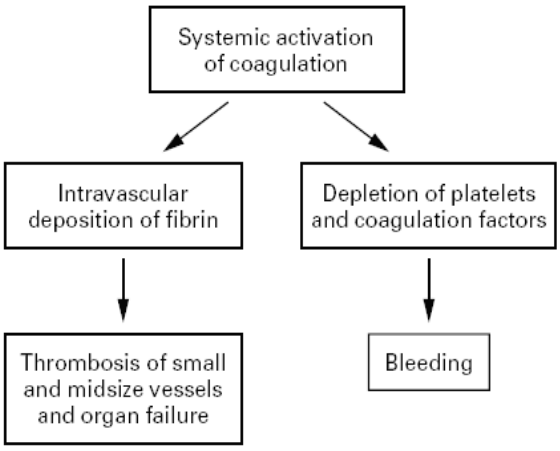

Figure 1. The Mechanism of Disseminated Intravascular Coagulation.

Systemic activation of coagulation leads to widespread intravascular deposition of fibrin and depletion of platelets and coagulation factors. As a result, thrombosis of small and midsize vessels may occur, contributing to organ failure, and there may be severe bleeding.

TABLE 1. COMMON CLINICAL CONDITIONS ASSOCIATED WITH DISSEMINATED INTRAVASCULAR COAGULATION.

Sepsis
Trauma
Serious tissue injury
Head injury
Fat embolism
Cancer
Myeloproliferative diseases
Solid tumors (e.g., pancreatic carcinoma, prostatic carcinoma)
Obstetrical complications
Amniotic-fluid embolism
Abruptio placentae
Vascular disorders
Giant hemangioma (Kasabach–Merritt syndrome)
Aortic aneurysm
Reactions to toxins (e.g., snake venom, drugs, amphetamines)
Immunologic disorders
Severe allergic reaction
Hemolytic transfusion reaction
Transplant rejection

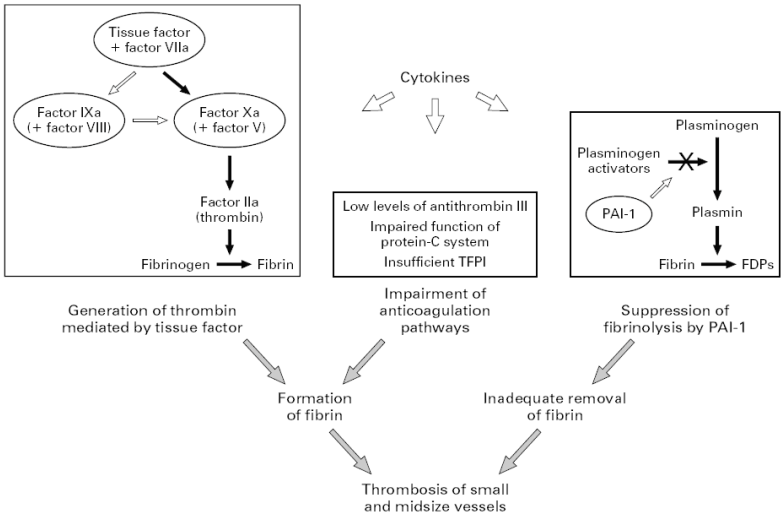

Figure 2. Pathogenetic Pathways Involved in Disseminated Intravascular Coagulation.

In patients with disseminated intravascular coagulation, fibrin is formed as a result of the generation of thrombin mediated by tissue factor. Tissue factor, expressed on the surface of activated mononuclear cells and endothelial cells, binds and activates factor VII. The complex of tissue factor and factor VIIa can activate factor X directly (black arrows) or indirectly (white arrows) by means of activated factor IX and factor VIII. Activated factor X, in combination with factor V, can convert prothrombin (factor II) to thrombin (factor IIa). Simultaneously, all three physiologic means of anticoagulation — antithrombin III, protein C, and tissue factor–pathway inhibitor (TFPI) — are impaired. The resulting intravascular formation of fibrin is not balanced by adequate removal of fibrin because endogenous fibrinolysis is suppressed by high plasma levels of plasminogen-activator inhibitor type 1 (PAI-1). The high levels of PAI-1 inhibit plasminogen-activator activity and consequently reduce the rate of formation of plasmin. The combination of increased formation of fibrin and inadequate removal of fibrin results in disseminated intravascular thrombosis. FDPs denotes fibrin-degradation products.

REFERENCES

- Barash chapter 10, pp. 230-231
- Stoelting chapter 25, pp. 496-497
- Roizen & Fleisher p. 120
- Levi M, Ten Cate H: Disseminated intravascular coagulation. N Engl J Med 1999; 341(8):586-92.