Case Conference

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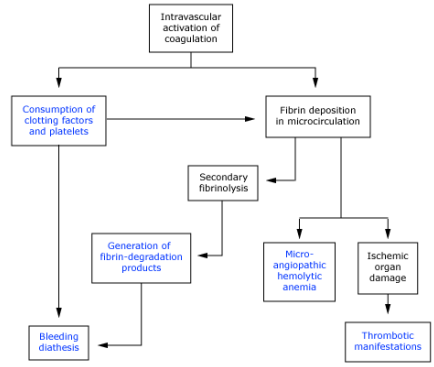
Discussion

Disseminated intravascular coagulation

Disseminated intravascular coagulation

- Disseminated intravascular coagulation is a systemic process producing both thrombosis and hemorrhage.
  - Exposure of blood to procoagulants
  - Formation of fibrin in the circulation
  - Fibrinolysis: release of FDP
  - Depletion of clotting factors
  - End-organ damage

Pathophysiology of the clinical manifestations of disseminated intravascular coagulation



Pathogenesis

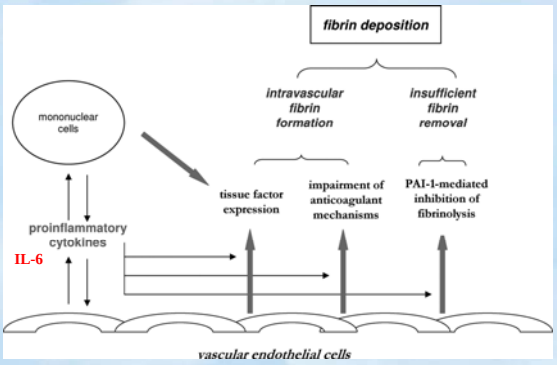


Table 1. Clinical conditions that may be associated with disseminated intravascular coagulation

Sepsis/severe infection (any microorganism)
Malignancy
Myeloproliferative/lymphoproliferative malignancies
Solid tumors
Trauma (e.g., polytrauma, neurotrauma, fat embolism)
Obstetrical calamities
Amniotic fluid embolism
Abruptio placentae
Organ destruction (e.g., severe pancreatitis)
Severe toxic or immunologic reactions
Snake bites
Recreational drugs
Transfusion reactions
Transplant rejection
Vascular abnormalities
Kasabach-Merritt syndrome
Large vascular aneurysms
Severe hepatic failure

It is important to stress that DIC is not a disease in itself but is always secondary to an underlying disorder that causes the activation of coagulation.

Clinical condition associated with DIC

- Severe sepsis may be complicated by DIC in about 35% of cases.
  - Gram-negative microorganisms → endotoxin
  - Gram-positive microorganisms → exotoxin
  - Viruses
  - parasites

Clinical condition associated with DIC

- Both solid tumors and hematologic malignancies may be complicated by DIC.
  - tissue factor → activates coagulation
  - Solid tumor → express procoagulant molecules (factor X- activating properties)

Clinical condiation associated with DIC

- Severe trauma:
  - release of tissue thromboplastin (in particular in patients with head trauma) into the circulation and endothelial damage may contribute to the systemic activation of coagulation.
  - DDx: coagulopathy due to massive blood loss → 發生於前幾個小時

Diagnosis

Table 2. Algorithm for the diagnosis of disseminated intravascular coagulation (DIC) (38)

Score global coagulation test results
1. Platelet count (>100 × 10^9/L = 0, <100 × 10^9/L = 1, <50 × 10^9/L = 2)
2. Elevated fibrin-related marker (e.g., fibrin degradation products or D-dimer) (no increase, 0; moderate increase, 2; strong increase, 3)[a]
3. Prolonged prothrombin time (<3 secs = 0, >3 but <6 secs = 1, >6 secs = 2)
4. Fibrinogen level (>1.0 g/L = 0, <1.0 g/L = 1)

Calculate score
If ≥5: compatible with overt DIC
If <5: no overt DIC; repeat next 1–2 days

Acute DIC

- Bleeding (64 percent)
- Renal dysfunction (25 percent)
- Hepatic dysfunction (19 percent)
- Respiratory dysfunction (16 percent)
- Shock (14 percent)
- Thromboembolism (7 percent)
- Central nervous system involvement (2 percent)

Chronic DIC

- Chronic DIC develops when blood is continuously or intermittently exposed to small amounts of tissue factor.
- Compensatory mechanisms in the liver and bone marrow are largely able to replenish the depleted coagulation proteins and platelets, respectively.
- Malignancy, particularly solid tumors, is the most common cause of chronic DIC.

Acute vs. chronic DIC

Coagulation parameters in acute and chronic disseminated intravascular coagulation

Parameter	Acute (decompensated) DIC	Chronic (compensated) DIC
Platelet count	Reduced	Variable
Prothrombin time	Prolonged	Normal
Activated partial thromboplastin time	Prolonged	Normal
Thrombin time	Prolonged	Normal
Plasma fibrinogen	Reduced	Normal-elevated
Plasma factor V	Reduced	Normal
Plasma factor VIII	Reduced	Normal
Fibrin degradation products	Elevated	Elevated
D-dimer	Elevated	Elevated

Management – Replacement Therapy

- Platelet transfusions should be considered to maintain the count greater than 20 to 30 $x10^9$/L in a bleeding patient.
  - f/u 10 ~ 60 mins after transfusion and q6h
- Fresh frozen plasma is given if significant DIC-associated bleeding is accompanied by a prolonged PT and PTT.

Management – Replacement Therapy

- Cryoprecipitate administration is considered in a symptomatic patient to maintain plasma fibrinogen more than 100 mg/dl.
  - each cryoprecipitate unit varies from 100 to 250 mg
  - 1 to 4 units/10 kg
  - f/u fibrinogen 30 to 60 minutes after transfusion and q6h

Management – Heparin

- chronic DIC (eg, secondary to solid tumor), the picture is more likely to be complicated by thromboembolic phenomena rather than bleeding
- 80 units/kg intravenously can be given, followed by an 18 units/kg/h

Thank you for your attention !