DIC

Disseminated intravascular coagulation

by

Venkatesan Abinesh

Group-163(2)

WHAT IS DIC ?

- Disseminated intravascular coagulation (DIC) is an acquired syndrome characterized by the intravascular activation of coagulation with loss of localization arising from different causes.It can originate from and cause damage to microvasculature, which if sufficiently severe , can produce organ dysfunction.

- Normal Pregnancy – Hypercoagulable state.
- After the 1st trimester there occurs a marked increase in plasma fibrinogen(more than double the non pregnant level).
- Plasma fibrinolytic activity is decreased during pregnancy and returns to normal within one hour of delivery of placenta.

TYPES OF DIC ?

- ACUTE DIC – the physical findings are those of underlying or inciting etiology .
- Patients with acute DIC have petechiae on the soft palate and legs from thrombocytopenia and ecchymosis at venipuncture sites.
- Acute DIC occurs in obstetric calamities such as placental abruption
- and amniotic fluid emboli.

- Amniotic fluid has been shown to be able to activate coagulation in vitro, and the degree of placental seperation correlates with the extent of DIC , suggesting that leakage of thromboplastin like material from the placental system is responsible for the occurrence of DIC.

- Coagulation system may also be activated in patients with pre
- eclampsia and HELLP syndrome.

- CHRONIC DIC- manifestation is thrombosis from excess thrombin formation , the symptoms and signs of venous thromboembolism may be present.

COMMON CAUSES OF DIC

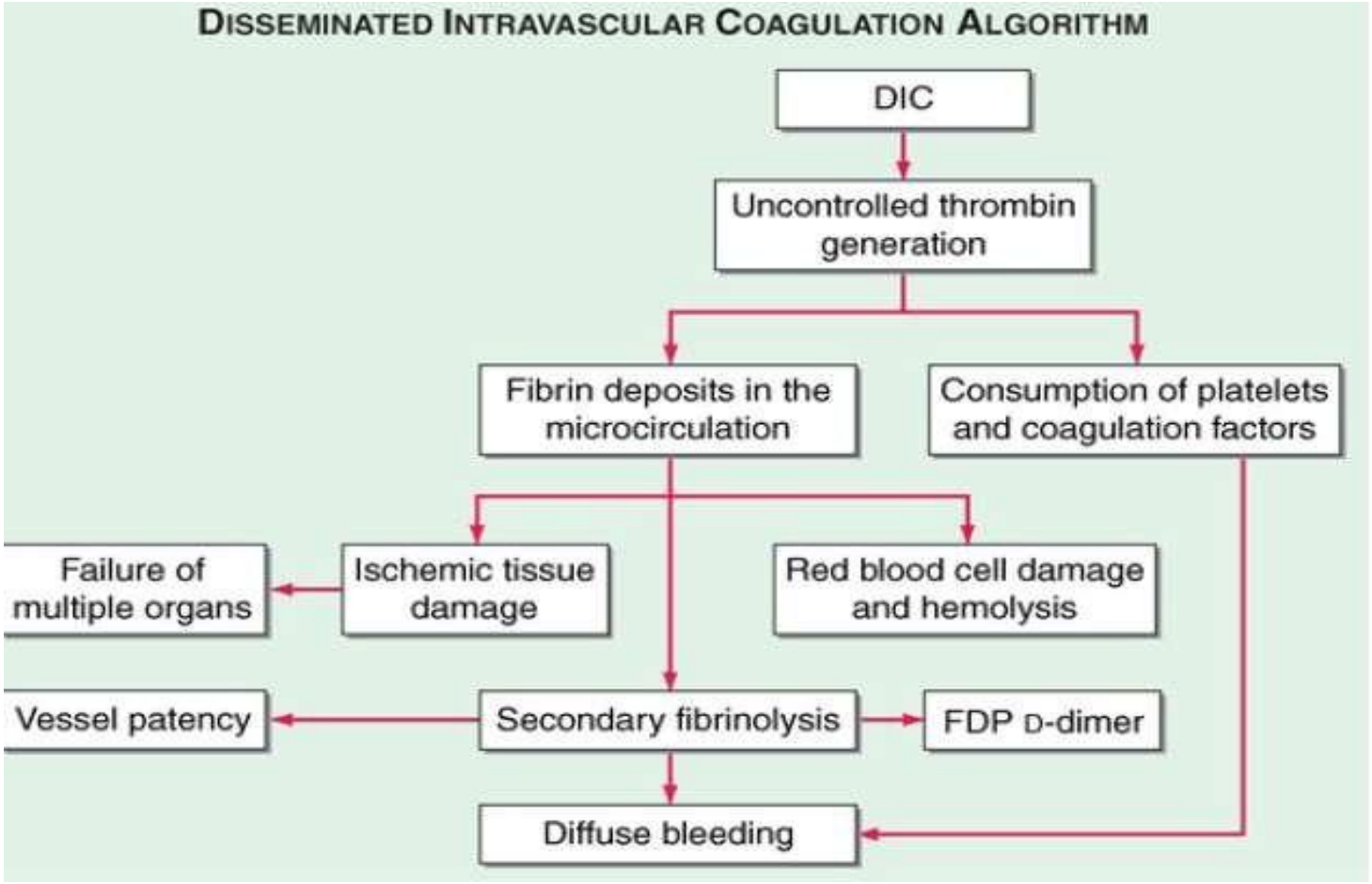
ACUTE DIC

- Abruptio Placentae
- Endotoxemia- septic abortions, chorioamnionitis, pyelonephritis of pregnancy.
- Amniotic Fluid Embolism
- Severe pregnancy induced hypertension
- Intra-amniotic hypertonic saline

- Vesicular mole
- Dextran Infusion
- Hemorrhagic shock due to –PPH , Cs
- **CHRONIC DIC** – IUD (prolonged retension of dead fetus).

PATHOPHYSIOLOGY

- DIC is diagnosed in almost one-half of pregnant women with abruptio placentae, or with amniotic fluid embolism. Trauma, particularly to the brain, can also result in DIC.


DISSEMINATED INTRAVASCULAR COAGULATION ALGORITHM
DIC
Uncontrolled thrombin generation
Fibrin deposits in the microcirculation
Consumption of platelets and coagulation factors
Failure of multiple organs
Ischemic tissue damage
Red blood cell damage and hemolysis
Vessel patency
Secondary fibrinolysis
FDP D-dimer
Diffuse bleeding

CLINICAL MANIFESTATION

- Clinical manifestations of DIC are related to the magnitude of the imbalance of hemostasis, to the underlying disease, or to both.
- The most common findings are bleeding ranging from oozing from venipuncture sites, petechiae, and ecchymoses to severe hemorrhage from the gastrointestinal tract, lung, or into the CNS.
- In chronic DIC, the bleeding symptoms are discrete and restricted to
- skin or mucosal surfaces.

- The hypercoagulability of DIC manifests as the occlusion of vessels in the microcirculation and resulting organ failure.
- Thrombosis of large vessels and cerebral embolism can also
- occur.
- Hemodynamic complications and shock are common among patients with acute DIC. The mortality ranges from 30 to >80% depending on the underlying disease, the severity of the DIC, and the age of the patient.

- Bleeding from at 3 unrelated sites is particularly suggestive of DIC.
- Brain- altered state of consiousness , seizures
- Lungs- respiratory distress
- Heart- hypotension, cardiac arrest
- Kidney – Oliguria,Anuria, Acidosis

LAB INVESTIGATIONS

- The laboratory investigation should include coagulation tests [aPTT, PT, thrombin time (TT)] and markers of fibrin degradation products (FDPs), in addition to platelet and red cell count and analysis of the blood smear.

- These tests should be repeated over a period of 6–8 hours because an initially mild abnormality can change dramatically in patients with severe DIC.

- A reduction in platelet count at subsequent tests is a sensitive sign of DIC.

LAB RESULTS

- prolongation of PT and/or aPTT
- platelet counts less than 100,000, or a rapid decline in platelet numbers.
- the presence of schistocytes (fragmented red cells) in the blood smear; and elevated levels of FDP.
- The most sensitive test for DIC is the FDP level..

- DIC is an unlikely diagnosis in the presence of normal levels of FDP. The D-dimer test is more specific for detection of fibrin—but not fibrinogen degradation products and indicates that the cross-linked fibrin has been digested by plasmin. Because fibrinogen has a prolonged half-life, plasma levels diminish acutely only in severe cases of DIC.

RISK ASSESSMENT

- Does the patient have an underlying disorder compatible with DIC?
- Lab coagulation tests- Platelet counts, D-dimer ,

  Fibrinogen , PT and aPTT.
- Platelet count >1 lac = 0 points , 50,000 to 1 lac = 1 point

  , <50,000 = 2 point.
- Elevated fibrin marker – No elevation = 0 point , moderate increase=
- 2 point , strong inc = 3 points.

- Prolonged PT <3sec = 0 point , 3 to 6 sec = 1 point ,

  >6 sec = 2 point.

- Fibrinogen level > 1 gm/l = 0 points , < 1 = 1 point
- Calculate Score- > or = 5 compatible with overt DIC ,

  repeat scoring daily.

Japanese Association for Acute Medicine Scoring System for Disseminated Intravascular Coagulation	
Criteria	**Score**
Systemic inflammatory response syndrome criteria	
• ≥ 3	1
• 0-2	0
Platelet count (cells/μL)	
• < 80,000 or > 50% decrease within 24 hr	3
• ≥ 80,000 and < 120,000 or > 30% decrease within 24 hr	1
• ≥ 120,000	0
Prothrombin time (patient's value/normal value)	
• ≥ 1.2	1
• < 1.2	0
Fibrin/fibrinogen degradation products (mg/L)	
• ≥ 25	3
• ≥ 10 and < 25	1
• < 10	0
A score of ≥ 4 indicates a diagnosis of DIC.	
Adapted from Gando S, Iba T, Eguchi Y et al; Japanese Association for Acute Medicine Disseminated Intravascular Coagulation	

TREATMENT

- The morbidity and mortality associated with DIC are primarily related to the underlying disease rather than the complications of the DIC. The control or elimination of the underlying cause should therefore be the primary concern.
- Patients with severe DIC require control of hemodynamic parameters, respiratory support, and sometimes invasive surgical procedures.
- Attempts to treat DIC without accompanying treatment of the
- causative disease are likely to fail.

MANAGEMENT OF HEMORRHAGIC SYMPTOMS

- The control of bleeding in DIC patients with marked thrombocytopenia (platelet counts <10,000–20,000/ L3) and low levels of coagulation factors will require replacement therapy.

- The PT (>1.5 times the normal) provides a good indicator of the severity of the clotting factor consumption.

- Replacement with FFP is indicated (1 unit of FFP increases most coagulation factors by 3% in an adult without DIC).

- Low levels of fibrinogen (<100 mg/dL) or brisk hyperfibrinolysis will require infusion of cryoprecipitate (plasma fraction enriched for fibrinogen, FVIII, and vWF).

- The replacement of 10 U of cryoprecipitate for every 2–3 U of FFP is sufficient to correct the hemostasis.

- The transfusion must be adjusted according to the patient's clinical and laboratory evolution.
- Platelet concentrates at a dose of 1–2 U/10 kg body weight are
- sufficient for most DIC patients with severe thrombocytopenia.
- Clotting factor concentrates are not recommended for control of bleeding in DIC because of the limited efficacy afforded by replacement of single factors (FVIII or FIX concentrates), and the high risk of
- products containing traces of aPCCs that further aggravate the disease.

REPLACEM ENT O F CO AG ULATI O N O R FI BRI NO LYSI S I NHI BI TO RS

- Drugs to control coagulation such as heparin, ATIII concentrates, or antifibrinolytic drugs have all been tried in the treatment of DIC.
- In acute DIC, the use of heparin is likely to aggravate bleeding. To date, the use of heparin in patients with severe DIC has no proven survival benefit.

- The use of antifibrinolytic drugs, EACA, or tranexamic acid, to prevent fibrin degradation by plasmin may reduce bleeding episodes in patients with DIC and confirmed hyperfibrinolysis. However, these drugs can increase the risk of thrombosis and concomitant use of heparin is indicated.

Blood Component Replacement Therapy		
Blood Component	**Suggested Dose**	**Indication**
Cryoprecipitate (10–15 mL/unit)	1 unit/10 kg body weight	Symptomatic bleeding with fibrinogen < 100 mg/dL
Fresh frozen plasma (175–250 mL/ unit)	15–20 mL/kg body weight	Symptomatic bleeding with prolonged PT or aPTT
Platelet concentrates	1–2 units/10 kg body weight	Symptomatic bleeding with platelet count < 50,000 cells/µL or < 10,000–20,000 cells/µL without bleeding

THANK YOU!!