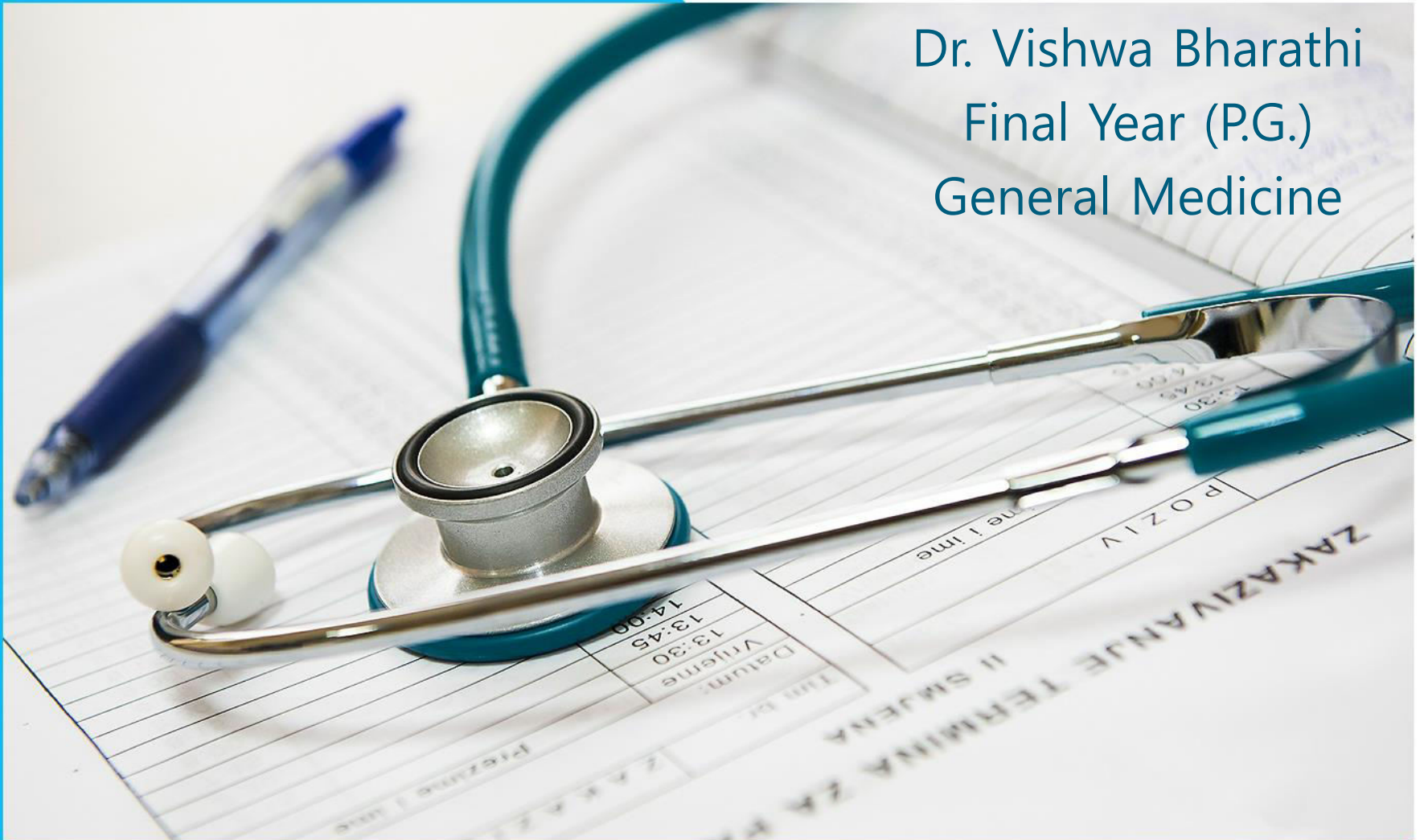


# DISSEMINATED INTRAVASCULAR COAGULATION (DIC)

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Final Year (P.G.)  
General Medicine



# Introduction



## According to International Society of Thrombosis & Hemostasis

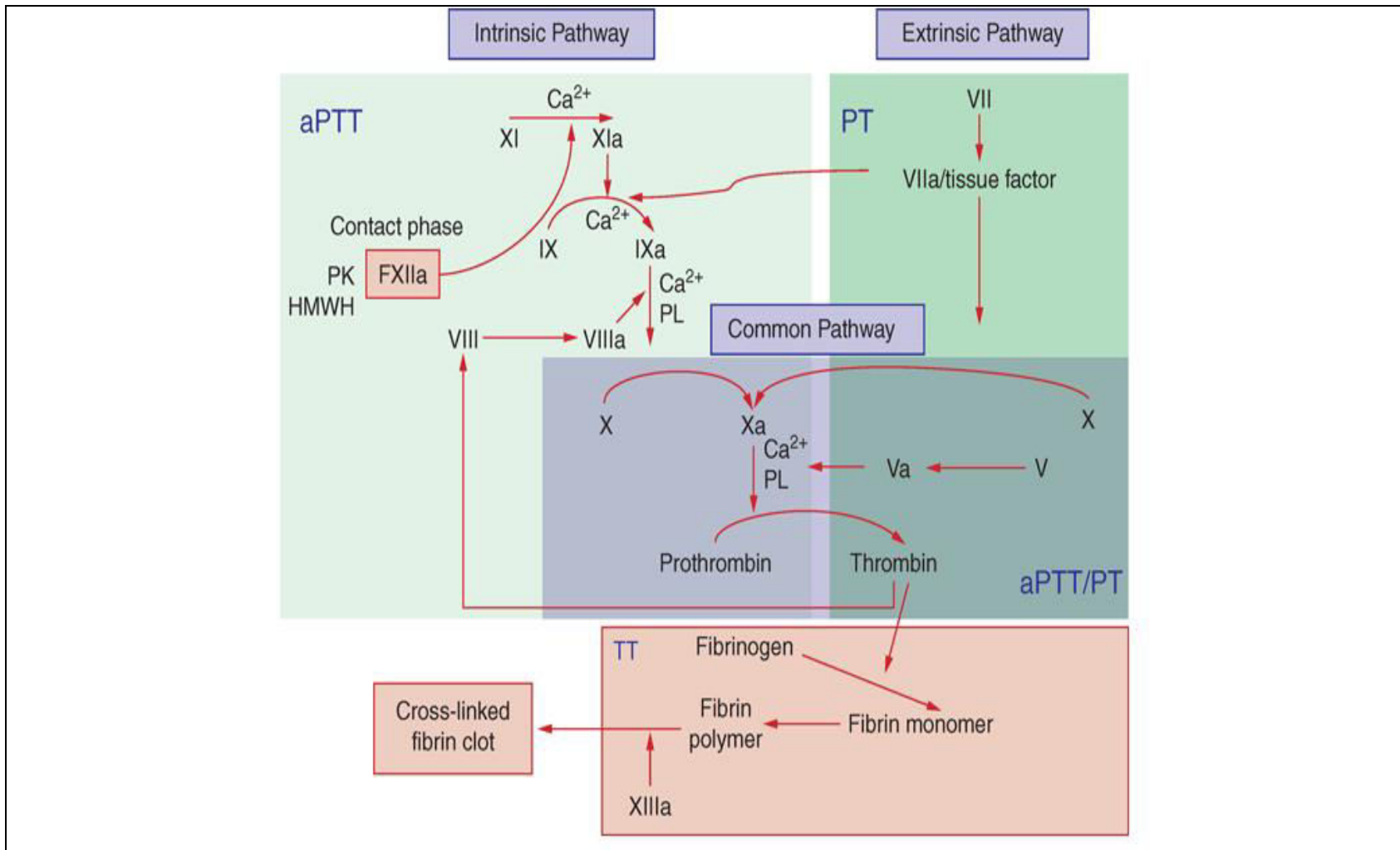
- ❖ Disseminated intravascular coagulation (DIC) is an acquired syndrome characterized by intravascular activation of coagulation with loss of localization arising from different causes.
- ❖ It can originate from and also cause damage to the microvasculature, which if sufficiently severe ,can produce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

# Frequency of DIC

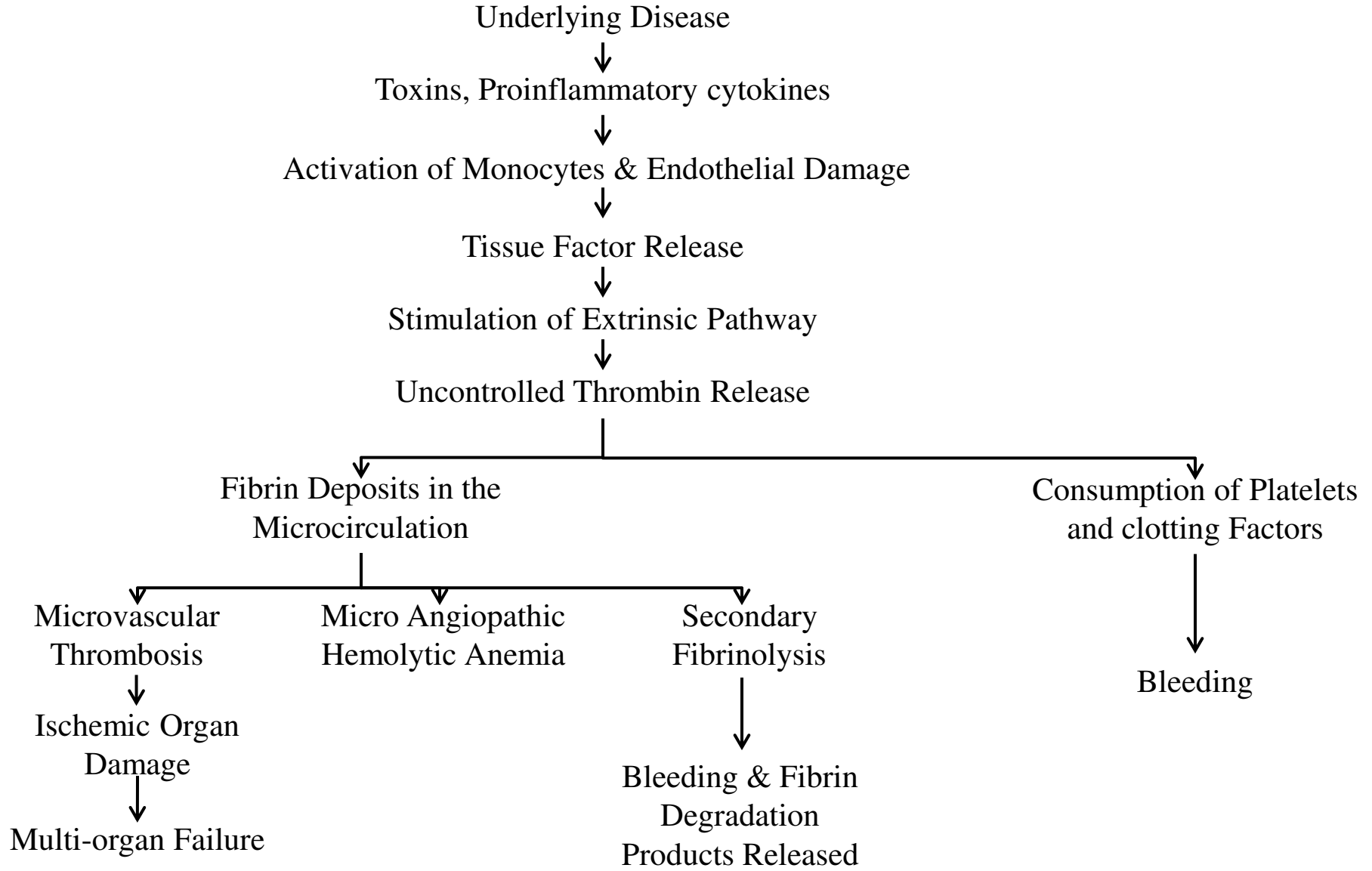


- ❖ DIC occurs in 30 – 50% of patients with sepsis, with equal frequency in gram positive and gram negative bacterial infections.
- ❖ It can occur in more than 50% patients with abruptio placentae, septic abortions and amniotic fluid embolism.
- ❖ In about 30% patients with **HELLP** syndrome and severe pre eclampsia
- ❖ In 10 -15% of Cancer patients with metastasis
- ❖ 50 -70% pts with neurotrauma

# Summary Of Coagulation Pathways



# Pathophysiology



# Clinical Features of DIC



- ❖ Skin ecchymosis
- ❖ Bleeding from i/v sites, endotracheal tubes and urinary catheters.
- ❖ Gingival bleeding, epistaxis, malena
- ❖ Cough, dyspnoea
- ❖ Confusion, disorientation
- ❖ Fever

In addition, symptoms related to underlying disease

# Common Clinical Causes Of DIC



## Sepsis

- ❖ Bacterial: staphylococci, streptococci, pneumococci, meningococci, gram negative bacilli
- ❖ Viral
- ❖ Mycotic
- ❖ Parasitic
- ❖ Rickettsial

## Trauma and tissue injury

- ❖ Brain injury, extensive burns, fat embolism, rhabdomyolysis

# Common Clinical Causes Of DIC



## Drugs

- ❖ Fibrinolytic agents, aprotinin, warfarin, prothrombin complex concentrates, amphetamine

## Vascular disorders

- ❖ Giant hemangiomas, aortic aneurysms

## Obstetrical complications

- ❖ Abruption placentae, amniotic fluid embolism, septic abortion, dead fetus, **HELLP** syndrome, severe pre eclampsia

## Cancer

- ❖ Adenocarcinoma of pancreas, prostate, hematologic malignancies



# Common Clinical Causes Of DIC



## **Immunologic disorders**

- ❖ Acute hemolytic transfusion reaction, organ or tissue transplant rejection, Graft versus host disease

## **Envenomation**

- ❖ Snake, insects

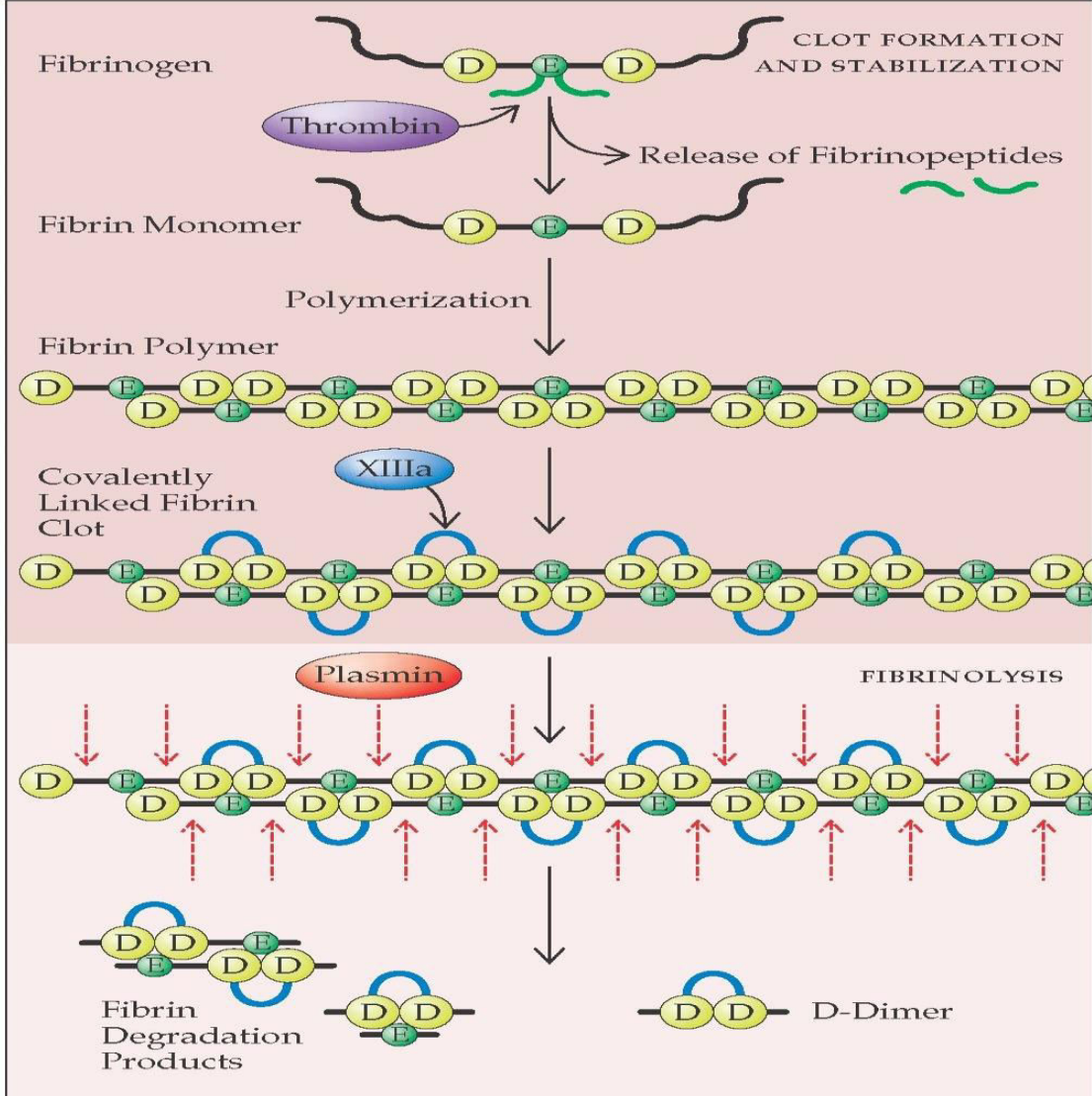
## **Liver disease**

- ❖ Fulminant hepatic failure, cirrhosis, fatty liver of pregnancy

## **Miscellaneous**

- ❖ Shock, acute respiratory distress syndrome, massive transfusion

# Fibrinolytic System



# Laboratory Studies



## Screening assays

- ❖ Prothrombin time(PT)
- ❖ Activated partial thromboplastin time (APTT)
- ❖ Platelet count,RBC count and blood smear analysis
- ❖ Fibrinogen levels

## Lab markers of thrombin generation

- ❖ D–dimer
- ❖ Protamine paracoagulation assay for fibrin monomer
- ❖ Ethanol gel assay for fibrin monomers
- ❖ Thrombin anti-thrombin complex

## Ancillary tests

- ❖ Fibrin degradation products
- ❖ Antithrombin levels
- ❖ Anti plasmin levels
- ❖ Factor V levels

# Laboratory Studies (Contd...)



## D-dimer test

- ❖ It is a very sensitive test for the diagnosis of DIC
- ❖ The test has a negative predictive value of  $> 90\%$
- ❖ Normal value is  $0.2 - 0.5$  mg/ml

## False positive D-dimer test

- ❖ Recent surgery
- ❖ Trauma
- ❖ Renal, liver and cardiac failure

# Laboratory Studies (Contd...)



## Fibrin degradation products

- ❖ FDPs are a measure of plasmin cleaved fibrinogen or fibrin
- ❖ FDPs do not distinguish between plasmin degradation by product of either fibrin or fibrinogen
- ❖ FDPs have a sensitivity of 85% & specificity 50%
- ❖ Normal value < 10 mg/ml
- ❖ **A combination of FDP and D dimer has 100% specificity and sensitivity**



# Diagnostic Criteria for DIC

## International Society for Thrombosis and Hemostasis Disseminated Intravascular Coagulation Scoring System

Use only in patients with an underlying condition known to be associated with DIC

	0	1	2
Thrombocytopenia	$> 100,000 / \text{mm}^3$	$\leq 100,000 / \text{mm}^3$	$\leq 50,000 / \text{mm}^3$
D-dimer	Normal	$\leq 10$ times ULN	$\geq 10$ times ULN
PT prolongation	$< 3$ sec	3-6 sec	$> 6$ sec
Fibrinogen	$> 100$ mg/dl	$\leq 100$ mg/dl	

Overt DIC  $\geq 5$  points

Non overt DIC  $< 5$  points

# Acute and Chronic DIC



Parameters	Acute (decompensated DIC)	Chronic (compensated DIC)
Platelet count	Reduced	Variable
Prothrombin time	Prolonged	Normal
APTT	Prolonged	Normal
Thrombin time	Prolonged	Normal
Plasma fibrinogen	Reduced	Normal – elevated
Plasma factor 5	Reduced	Normal
Plasma factor 8	Reduced	Normal
Fibrin degradation products	Elevated	Elevated
D -dimer	Elevated	Elevated

Reference: Medicine Update 2015

# Differential Diagnosis of DIC



- ❖ Advanced liver disease
- ❖ Thrombotic micro angiopathy
- ❖ Fibrinogenolysis
- ❖ Disorders of hemostasis (correctable factor deficiency, vit k deficiency)
- ❖ Thrombocytopenia



# Management of DIC



- ❖ Management of DIC involves following three important steps, which should be initiated timely and sequentially.
- ❖ **I. Vigorous therapy underlying disorder.**
- ❖ **II. Energetic treatment of life threatening complication e.g. shock, hypoxaemia, and acidosis.**
- ❖ **III. Therapy of DIC per se.**
- ❖ When there is no bleeding or venous thromboembolism with only lab parameters deranged - observation without any replacement therapy
- ❖ Frequent monitoring of blood counts and clotting factors
- ❖ Monitoring the CVP, gas exchange and electrolyte balance

# Management of DIC (Contd...)



## **Prompt treatment of the underlying cause**

- ❖ Optimal antibiotics in sepsis syndrome
- ❖ Uterine evacuation for abruptio placenta
- ❖ Restoration of hemodynamic stability for hypovolemic shock
- ❖ Anti snake venom for snake bite

## **ANTIBIOTICS**

- ❖ Always i.v antibiotics are given
- ❖ started after taking blood cultures
- ❖ Outcomes are worse if the organism is insensitive to the initial regimen

# Management of DIC (Contd...)



## COMPONENT SUPPORT

### PLATELET TRANSFUSION

- ❖ Platelet count should be maintained around 20,000-30,000/mm<sup>3</sup> in a bleeding patient
- ❖ single donor platelet transfusion

### CRYOPRECIPITATE

- ❖ Is rich in factor VIII, Fibrinogen and VWF
- ❖ 1-2 units/ 10 kg can be given
- ❖ Maintain fibrinogen level > 100 mg/dl

# Management of DIC (Contd...)



## **FRESH FROZEN PLASMA**

- ❖ Provides all clotting factors and corrects PT and APTT
- ❖ Dose 10-15ml/kg every 8-12 hrly
- ❖ Transfusion can be stopped once lab parameters improve

## **ANTITHROMBIN CONCENTRATES**

- ❖ More effective in the presence of hepatic insufficiency

## **ACTIVATED PROTEIN C CONCENTRATES (Drotrecogin alfa)**

## **PROTHROMBIN COMPLEX CONCENTRATES**

# Management of DIC (Contd...)



## HEPARIN

❖ Is a naturally occurring anticoagulant

In acute DIC heparin can aggravate bleeding

To date use of heparin in acute DIC has no proven survival ben

## Indications of heparin in DIC

❖ Chronic DIC of malignancy

❖ Clinical thrombosis: dermal necrosis, purpura fulminans, acral ische mia, VTE

❖ Retained dead fetus with hypofibrinognemia

❖ AML M3 prior to conventional chemotherapy

# Management of DIC (Contd...)



## **SYNTHETIC INHIBITORS OF THROMBIN**

- ❖ Hirudin, desirudin, bivalirudin, argatroban, melagatran, ximelgatran, dabigatran
- ❖ Can be used in heparin induced thrombocytopenia and when heparin is ineffective in the presence of antithrombin deficiency

## **FIBRINOLYSIS INHIBITORS**

- ❖ They block the secondary fibrinolysis that accompanies DIC  
Tranexamic acid and Epsilon Amino Caproic Acid (EACA) prevent Fibrin degradation by plasmin and reduce bleeding episodes in patients with confirmed fibrinolysis

# Prognosis



- ❖ Prognosis depends on the underlying disorder
- ❖ If the condition is self limiting, prognosis is good
- ❖ Appropriate and early initiation of antibiotic therapy has a positive impact on the outcome
- ❖ Prognosis is poor if there is
  - Failure to recognize the underlying etiology and in case of sepsis if the organism is insensitive to the initial empirical antibiotic regimen

# References



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- ❖ Furlong MA,Furlong BR.Disseminated intravascular coagulation.jan 2009
- ❖ Disseminated intravascular coagulation in obstetric disorders and its acute hematological management,jecko thachil,school of clinical sciences,university of liverpool,UK,2009



**Thank You !**

