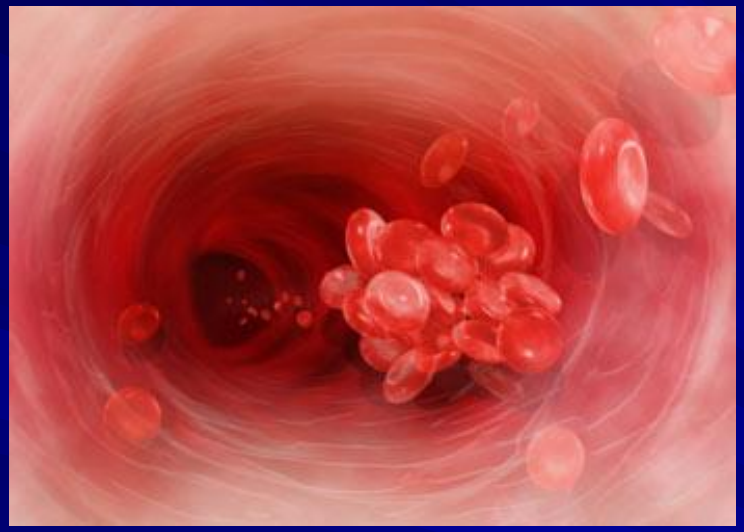
# Disseminated Intravascular Coagulation (DIC) Seminar

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- 8.3.2012

The Chaim Sheba Medical Center
at Tel Hashomer - Est. 1948
The Hospital of Israel

# Our plan:

- Understand the pathophysiology
- Identify risk factors and etiology
- Understand Acute vs Chronic DIC
- Signs and symptoms
- Diagnosing DIC (lab interpretations)
- Treatment modalities
- Case presentation

# What is DIC?

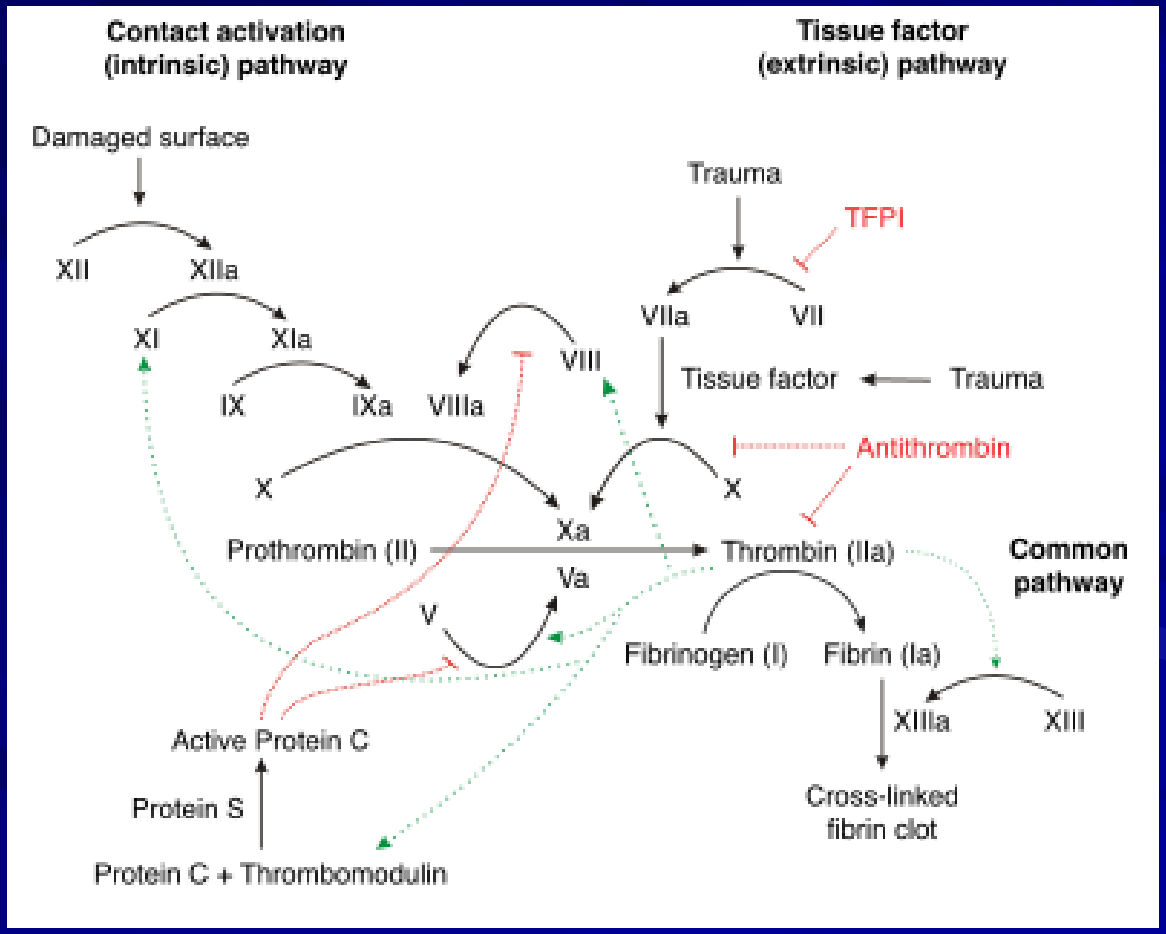
- Is considered an “acquired bleeding disorder”
- Is not a disease entity but an event that can accompany various disease processes
- Is an alteration in the blood clotting mechanism: abnormal acceleration of the *coagulation cascade*, resulting in thrombosis
- As a result of the depletion of clotting factors, hemorrhage occurs simultaneously
- Is a *Paradoxical Clinical Presentation* “clotting and hemorrhage”

(Porth, C.M. (2004) Essentials of Pathophysiology) & (Otto, S. (2001). Oncology Nursing)

# Pathophysiology

In DIC, a systemic activation of the coagulation system simultaneously leads to thrombus formation (compromising blood supply to various organs) and exhaustion of platelets and coagulation factors (results in hemorrhage). This is a disruption of body homeostasis.

# A quick reminder:



# Pathophysiology

**Thrombosis**-brief period of hypercoagulability

1) Coagulation cascade is initiated, causing widespread fibrin formation
2) Microthrombi are deposited throughout he microcirculatory
3) Fibrin deposits result in tissue ischemia, hypoxia, necrosis
4) Leads to multi organ dysfunction

**Fibrinolysis**-period of hypocoagulability (the hemorrhagic phase)

1) Activates the complement system
2) Byproducts of fibrinolysis (fibrin/fibrin degradation products(FDP)) further enhance bleeding by interfering with platelet aggregation, fibrin polymerization, & thrombin activity
3) Leads to Hemorrhage

# Pathophysiology

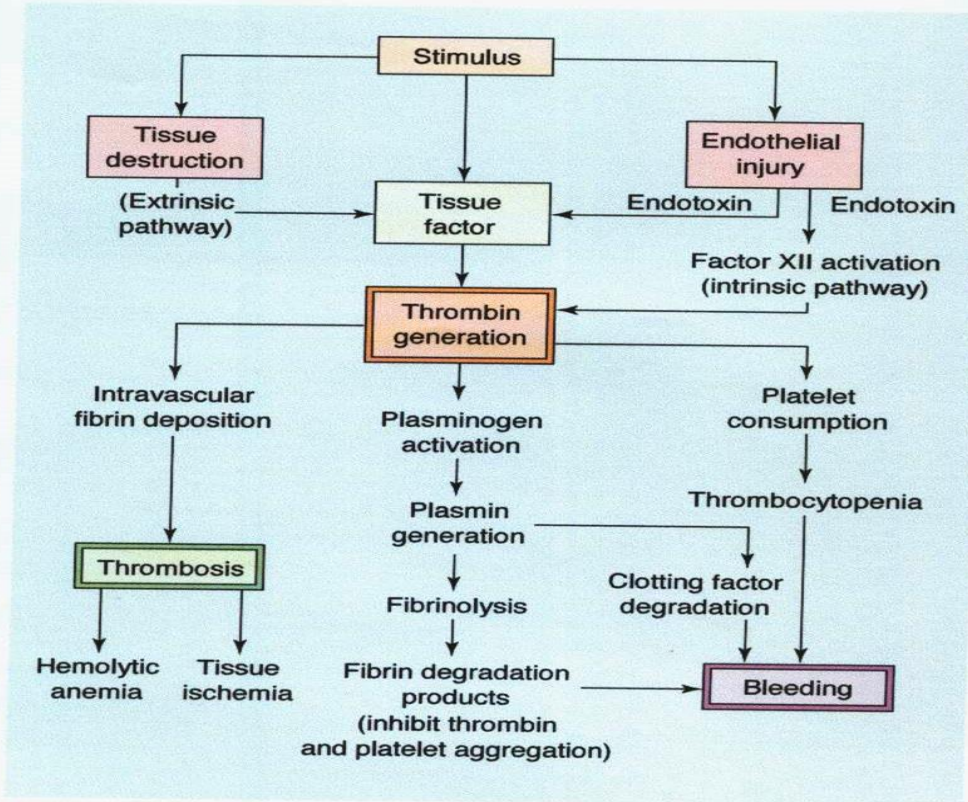



Figure 15-6 Pathophysiology of disseminated intravascular coagulation.

# Pathologic Pathways

- **Extrinsic** (endothelial)
  - Shock or trauma
  - Infections ( Endotoxin, gram positive and gram negative sepsis, aspergillosis)
  - Obstetric complications (eclampsia, placenta abruptio, fetal death syndrome)
  - Malignancies: APML, AML, cancers of the lung, colon, breast, prostate (adenocarcinomas)
- **Intrinsic** (blood vessel)
  - Infectious vasculitis (certain viral infections, rickettsial)
  - Vascular disorders (giant hemangioma, aortic aneurysm)
  - Intravascular hemolysis (hemolytic transfusion reactions)
  - Miscellaneous: snakebite, pancreatitis, liver disease (cirrhosis, hepatic failure)

# Clinical Features

- Onset maybe Acute or Chronic
  - Acute DIC
    - Develops rapidly over a period of hours
    - Presents with sudden bleeding from multiple sites
    - Treated as a medical emergency
  - Chronic DIC
    - Develops over a period of months
    - Maybe subclinical
    - Eventually evolves into an acute DIC pattern

# Signs and Symptoms

Most common sign of DIC is bleeding

-manifested by ecchymosis, petechiae,and purpura

-bleeding from multiple sites either oozing or frank bleeding

-cool and or mottled extremities

-dyspnea and chest pain if pleura and pericardium involvement

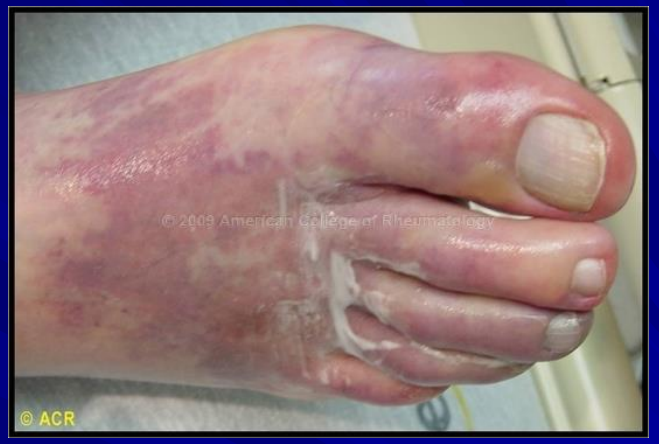
-hematuria

# Diagnosis/Lab Findings

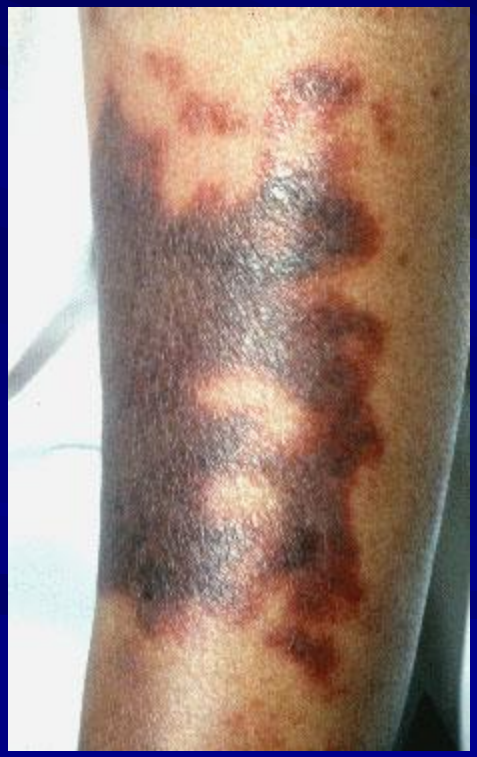
| *Test* | *Abnormality* |
| --- | --- |
| Platelet count | Decreased |
| **Fibrin degradation product (FDP)** | Increased |
| Prothrombin time (PT) | Prolonged |
| Activated PTT | Prolonged |
| Thrombin time | Prolonged |
| Fibrinogen | Decreased |
| **D-dimer** | Increased |
| Antithrombin | Decreased |

# Clinical Manifestations:

Purpura fulminans




Ecchymosis



# Treatment Modalities

- **Treat the underlying cause!!!**
- Provide supportive management of complications
- Support organ function
- Stop abnormal coagulation and control bleeding by replacement of depleted blood and clotting components (FFP,Platelets,PRBC)
- Low doses of Heparin may be effective for low-grade DIC, not for severe cases.

# Case Presentation

- On his 50th birthday, a patient with known advanced hepatitis C was forcibly taken by his best friends to an oyster raw bar to eat raw oysters. Despite his pleas that he hated raw oysters, he swallowed a few. After a few hours of partying, he felt ill, developed chills, fevers, and confusions and was taken to the Emergency Room where staff considered him inebriated and placed him in a quiet, dark room.

**Thrombocytopenia and thrombosis in disseminated intravascular coagulation (DIC)**
**Craig S. Kitchens**1, Hematology Am Soc Hematol Educ Program. 2009:240-6

- In fact his ethanol level returned at 180 mg/dL.
- An hour later he developed large ecchymoses all over his body.
- His PT was 16.7 seconds; his PTT 68 seconds; platelet count 48,000/μL; pH was 7.08, and analysis for D-dimer tests returned markedly positive.

- The ecchymoses worsened, epistaxis began, lactic acidosis worsened, and the results of all the prior blood tests deteriorated.
- What is going on? What can be expected? What can we do for this patient?

- Blood cultures were drawn and broad spectrum antibiotic therapy started.
- The patient had acquired *Vibrio vulnificus* septicemia from ingestion of raw seafood.
- Because this skin necrosis was rapidly advancing, it was elected to administer heparin by infusion
- He expired from cardiovascular collapse. Autopsy showed microvascular changes consistent with DIC, advanced hepatic cirrhosis, and adrenal infarction/hemorrhage.

# Take home message:

- a *Paradoxical Clinical Presentation* "clotting and hemorrhage"
- Patient bleeding, thrombosing, or both.
- An underlying illness or process
- Test: thrombin time (TT), prothrombin time (PT), partial thromboplastin time (PTT), fibrin degradation products (FDP), D-dimer, or platelet count.
- **Treat the underlying cause!!!**

*Qustions?*