Coagulation Disorders

Importance in surgical practice

Surgical procedures

Trauma

Additionally,

Emboli

Thrombosis

DVT

- Events
- Vascular constriction
- Platelet plug formation
- Fibrin formation
- Fibrinolysis

- Vascular constriction:
- Thrombaxane A2
- via release of Arachidonic acid
- conversion to PG2
- Serotonin release from injured endothelium

- Coagulation:
- Platelet adhesion(this require vWB factor)recruites other platelets
- Principal mediators
- ADP
- Serotonin

- Coagulation cascades:
- Intrinsic (activation of factor X11)
- Subsequent activation of X1 1X V111

- Extrinsic
- Tisuue Factor(TF)
- Binds to V11 activation to V11a
- Then activation of X to Xa
- Conversion of prothrombin(11) to thrombine and factor 1 (Fibrinogen)
- To fibrin.
- Fibrin monomers are cross-linked to polymers with the assistance of V111

- Degeneration:
- 1 Feedback inhibition
- 2 Activation of protein C that leads to release of TPA
- protein S
- Fibrin clot degeneration is accomplished by Plasmin.

- Congenital Factor Deficiencies:
- V111 Hemophilia A and vWB disease
  1X Hemophilia B (Christmas disease)
  X1 Hemophilia C

- vWB disease,
- The most common congenital bleeding disorder
- Quantitative and qualitative defect in vWB factor responsible for carrying V111 and platelet adhesion.

- Approach:

  History

  PE

  Investigations:

    Pre OP clinics

    Guidelines, protocols and policies.

These are done for etiological Factors in bleeding

- DIC:
- Disseminated Intravascular Coagulation, or Acquired Hypofibrinogenemia
- Systemic activation of coagulation pathways
- Excessive Thrombine generation
- Diffuse microthrombi
- Consumption and depletion of Platelets and coagulation factors
- Classic picture of diffuse bleeding

- DIC contd.
- Causes:
- Emboli
- Organ injuries
- Massive transfusion
- Malignancy
- Sepsis
- Transplant rejection
- Snake bites

- DIC Diagnoses:
- The presence of inciting underlying pathology
- Low Platelets
- Prolonged PT
- Low Fibrinogen levels
- high FDPs

- DIC Treatment:
- Relieving inciting factor
- Adequate perfusion
- FFP
- Cryoprecipitate,Platelets and/or Factor supplements

- Coagulopathy of liver disease:
- Prolonged PT
- Thromboses(protein C and S deficiency)

- Coagulopathy of trauma:
- Dilutional
- Acidosis
- Hypothermia
-

- Local Hemostases:
- Digital
- Tourniquet
- Packing
- Biological agents
-

- Blood Transfusion:
- Major blood groups
- Indications
- Principles:
- Serologic compatibility
- Cross matching
- Rh –ve should be transfused with Rh-ve only
- In emergency, O-ve can be transfused to all recipients
- Typing and cross matching are difficult inpatients with hemolytic anemia and those with multiple transfusions

- Hazards of transfusion:
- 10% of all transfusions
- Less than 0.5% are serious

- Non hemolytic reactions
- 1%
- Rise in Temp. more than 1C
- Bacterial contamination
- Allergic reactions
- Respiratory(TACO)
- TRALI
- Hemolytic reactions

- Hemolytic reactions Contd.

1 Acute

ABO incompatibility

Fatal in 6% of cases

Result from Lab. Or clerical errors

Basic problem is destruction of RBCs

Hemoglobinemia and Hemoglobinuria

Deaths occur from DIC and acute renal failure

2 Delayed, 2 to 10 days later

Extravascular hemolyses leading to anemia and hyper bilirubinemia

- What to look for?
- Pain at the site of transfusion
- Facial flushing
- Back and chest pain
- Associated symptoms(Tachycardia,Hypotension orrespiratory distress)

- What to do?
- Stop transfusion
- Suspected unit and a sample of recipients blood to be sent to blood bank
- Adequate hydration
- Monitoring urinary output
- Supportive messures

- Blood Components:
- Whole Blood
- RBCs
- WBCs
- Platelets
- FFP
- Cryoprecipitate
- Factor components