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INFLAMMATORY MECHANISMS in response to injury

VASCULAR INJURY: blood vessel damaged

Ruptured epithelium releases **ENDOTHELIN; Von Willebrandt's Factor (VWF);** and **TISSUE FACTOR** at the damaged membrane

VWF stored in easily ruptured vesicles on the endothelial cell membrane

Blood-floating **PLATELETS** are now **EXPOSED TO COLLAGEN;** **VWF** bridges connect platelets to collagen; therefore **PLATELETS ADHERE to COLLAGEN** **Tough as glue to resist shear forces**

ENDOTHELIN causes **TRANSIENT VASOCONSTRICTION to reduce blood loss**

Membrane-bound **TISSUE FACTOR Activates the COAGULATION CASCADE:** Results in **ACTIVATION OF THROMBIN**

THROMBIN induces DEGRANULATION

Physical and chemical barriers to infection are BREACHED

DEGRANULATION AND SHAPE CHANGE:
Platelets FLATTEN and spill granular contents:

FROM α-granules:
- Fibronectin
- **Fibrinogen**
- Factor 5
- Platelet-Derived Growth factor (**PDGF**)
- Transforming Growth Factor β (**TGF- β**)

FROM δ-granules (dense body):
- Adenosine Diphosphate (**ADP**)
- **HISTAMINE**
- **SEROTONIN**
- **ADRENALINE**
- **THROMBOXANE A2 (TXA2)**

THROMBIN also induces **AGGREGATION** **-and so does ADP and TXA2** **-this forms the PRIMARY (temporary) HEMOSTATIC PLUG** (with FIBRINOGEN linking platelets together)

Aggregated **PLATELETS CONTRACT: A fused lump is formed**

THROMBIN converts FIBRINOGEN into FIBRIN (insoluble) **this forms the SECONDARY (permanent) HEMOSTATIC PLUG:** **Fibrin *forms the scaffold* for FIBROBLASTS**

PDGF and **TGF- β** **Stimulate FIBROBLASTS**

BACTERIA enter the body

Hydrostatic pressure pushes water out into the tissue; thus, OEDEMA occurs

HISTAMINE and SEROTONIN INCREASE VASCULAR PERMEABILITY And DILATE VESSELS

Dilated vessels Have **LOWER BLOOD PRESSURE** therefore its easy for leucocytes to attach: less shear forces

Langerhans cells detect pathogen, migrate to lymph nodes, → specific immune response

FIBRINOGEN BREAKDOWN PRODUCTS and complement breakdown products C3a and C5a Are CHEMOTAXINS = attract **Neutrophils + Macrophages**

COMPLEMENT PROTEINS PENETRATE INTO TISSUE: COMPLEMENT CASCADE ACTIVATED BY *ALTERNATIVE PATHWAY* (directly by bacterial wall components)

C3a, c5a ativate **BASOPHIL and MAST CELL DEGRANULATION**: more **HISTAMINE** and **PROSTAGLANDIN**; thus, increased **VASCULAR PERMEABILITY**

NEUTROPHILS / MACROPHAGES **ROLL (via selectins)** **ADHERE (via integrins)** **DIAPEDESIS (penetrate endothelium)** **And MIGRATE DOWN CHEMOTAXIS GRADIENT TOWARDS THE SITE OF INJURY (via chemotaxins)**

C3b complement product OPSONISES antigens

MAC perforates bacterial walls, causing lysis

MACROPHAGES devour **C3b-** opsonised antigens **secrete TNF-α** to increase expression of **selectins and Integrins** **therefore, increased NEUTROPHIL MIGRATION** **+ exude IL-1** to activate more **MACROPHAGES** **+ remove cellular debris, eg. dead neutrophils**

EVENTUAL REMOVAL OF PATHOGEN, FORMATION OF COLLAGEN SCAR ON FIBRIN SCAFFOLD AND REGENERATION OF TISSUE