Inflammation

Def.: The host’s protective response to eliminate the initial cause of cell injury as well as the necrotic cells/tissues resulting from that original injury.

Inflammation 1: Acute inflammation: vascular and cellular pathways

Inflammation 2: Chemical mediators and chronic inflammation

Nomenclature of Inflammation

• Acute: hrs—days
• Chronic: weeks—mos.—yrs.

“…itis”: inflammation of

Heart: myocarditis (myocardium) endocarditis (valves)
Appendix: appendicitis
Colon: colitis

5 Cardinal Signs of Acute Inflammation

• Rubor
• Tumor
• Calor
• Dolor
• Functio laesa

Key Players in Inflammation

Leukocytes (and their mediators)
Blood vessel
Injurious agent
- Microbe
- Toxin
- Radiation
- Burn

Damaged cell
Extracellular matrix (ECM)
Plasma proteins
Endothelial cell
Resident WBC’s
Macrophage
Vascular Phase of Acute Inflammation

Cellular phase of Acute Inflammation
MARGINATION of WBC’s

Blood vessel lumen

ADHESION (firm)

Rolling with transient adhesions

PECAM-1 (CD31)

Collagenases

Transmigration (diapedesis)

Opsonization

Receptors

Opsonins

IgG

Microbe (e.g., bacterium)

C3

C1q

Collectins

Plasma CHO-binding lectins
Toll-like receptors (TLR)
- Homology to Drosophila
- Family of PPR’s (pattern recognition receptors)
- TLR 4: LPS (endotoxin)

Modified from Robbins Basic Pathology 8/e Fig. 1-20

DEFECTS IN LEUKOCYTE FUNCTION

Acute Appendicitis
- fecalith
- mucus
- transmural inflammation
- impaired venous drainage
- fibrinous exudate
Streptococcus pneumoniae

Acute Pneumonia
Acute Cell / Tissue Injury

Vascular phase

Cellular phase

Acute Inflammation
Chronic inflammation

De novo chronic disease
autoimmune, etc.

 Exceptions:
allergy/drugs/
parasites

eos

lymphocytes

viral infections

Abscess
Resolution

Fibrosis
(scar)

PMN

PL
mac

Hours—days
Weeks—mos—years