NURS 821
Vascular and Cardiac Disorders

Ischemic Heart Disorders
Lecture 6, Part 7

Angina

- Definition: Symptomatic paroxysmal pain or pressure associated with transient myocardial ischemia **lasting less than 5 minutes**
- Types
  - Classic-exertional angina-metabolic demands
  - Variant or Prinzmetal angina-CA spasm at rest
    - Etiology-?hyperactive SNS
  - Unstable-changing anginal pattern-preinfarction-danger sign; lasts longer

Myocardial Infarction

- **Definition**: Necrotic heart disease; necrosis of myocardium
- Predominant initial clinical presentation in men
- **Character**: prolonged myocardial ischemia longer than 30-45 minutes
- **Result**: irreversible cellular damage and muscle death

Myocardial Cell Regeneration

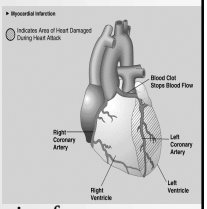
- 6/7/01 Reported by NHLBI that a landmark study has found evidence that human heart muscle cells may regenerate after MI.
- Changes longstanding ideas
- Further investigation warranted

Left Ventricular Vulnerability

- High O2 demand
  - Large muscle
  - High systemic vascular resistance
- Phasic coronary flow due to deep artery embedment in myocardium
  - CA compression during systole
  - Coronary flow during diastole

Myocardial Infarction Causes

- Atherosclerotic narrowing
- Thromboembolus occlusion
  - Morning peril
- Diminished systemic blood flow
- Infectious process-C pneumoniae from lungs cultured from plaques; irritable focus



Pathophysiology of Myocardial Ischemia

- Myocardium-bruised and cyanotic due to stagnation
  - Within 24h-cellular edema and inflammation; enzymes released
  - 2-3 days-tissue degradation and foreign debris removal; wall thins. ***Vulnerable period***.
  - 3weeks+-scar formation producing fibrous connective tissue, replacing necrosis
  - 6weeks+-scar well-established

Myocardial Necrosis

- Necrotic tissue **permanently ceases function**
- Infarction surrounded by ischemic zone of *potentially viable tissue*
- Infarct size depends on *collateral circulation*
- Necrotic size depends on *metabolic demands and fate of ischemic zone*

Effects of MI

- Decreased LV function:
  - Decreased contractility
  - Abnormal wall motion
  - Altered ventricular wall compliance
  - Decreased stroke volume
  - Decreased EF
  - Increased ventricular end systolic ***and*** end diastolic volume
  - Increased end diastolic pressure

MI Complications: Depend on Location and Extent

- Dysrhythmias
- Ventricles
  - septal defects
  - aneurysms
- Cardiomyopathy
- Cardiac rupture
- Papillary muscle dysfunction
- Thromboemboli
- Pericaditis
- Cardiogenic shock

MI Clinical Manifestations

- Chest pain
  - Non-radiating ***or***
  - Radiating to L arm, jaw, teeth
- Diaphoresis
- N,V
- Sense of impending doom (SNS response)
- 20-60% silent MI-especially DM w/neuropathies

MI Diagnostic Tests

- Cell necrosis leads to enzyme release-MB-CK, CPK, LDH, SGOT
- EKG-pronounced Q, ST elevations, inverted T waves

Convalescent Stage

- After symptoms resolve
- All manifestations of inflammation disappear
- Takes 8 weeks

Complications

- Arteritis and aneurysms-medium and large vessel
- Valvulitis-10-40% develop coronary vasculitis within 2 weeks
- Myocarditis
- Coronary artery aneurysms
  - thrombose
  - or evolve into segmental stenoses in chronic phase
  - (AHA, 2000)

Cardiac Complications

- Ischemia
- MI
- Ruptured aneurysm
- Pericarditis
- Endocarditis
- Dysrhythmias
- CHF

Diagnosis

- Total Clinical Picture!
- 5 manifestations
  - Bilateral conjunctivitis
  - Oropharyngeal manifestations
  - Extremity changes
  - Rashes
  - Cervical lymphadenopathy