Acute Coronary Syndromes

AACN PCCN Review

Acute Coronary Syndromes

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Acute Coronary Syndromes

I. INTRODUCTION

The rupture or disruption of the plaque is caused from internal and/or external factors or triggers.

Definitions of chest pain syndromes

Angina
Myocardial Anoxia

Exertional Angina
Pain that is brought on during times of increased myocardial oxygen demand like exertion, eating, extreme emotions and exposure to cold temperatures, the four Es. These symptoms are typically caused by or a sign of atherosclerosis.

Prinzmetal’s Angina or Variant Angina
Pain that occurs at rest, during sleep or without evidence of provocation. Symptoms are thought to be caused by coronary vasospasm.

Stable Angina
Exertional angina with consistent symptoms which is typically relieved with rest or cessation of cause and possibly nitroglycerine administration.

Unstable Angina
Aka crescendo or pre-infarction angina. Angina that:
   a. Has a recent onset (within 2 months) and severely limits activity
   b. Newly occurs at rest
   c. Differs in characters or symptoms from the person’s ‘typical exertional angina’ (it occurs with less exertion, has a greater intensity or longer duration, requires more interventions before obtaining relief)

Non-CAD Causes
Non-ischemic causes of chest pain must be ruled out, such as:

Cardiac Causes
   a. Acute Pericarditis
   b. Cardiac Tamponade
   c. Acute Myocarditis
   d. Aortic Stenosis
   e. Myocardial Contusion
   f. Mitral Valve Prolapse
g. Cardiomyopathies

**Non Cardiac Causes**

a. Panic Attack/Anxiety  
b. Illicit Drug Use  
c. Gastrointestinal Disorders  
d. Spontaneous Pneumothorax  
e. Pulmonary Embolism  
f. Pulmonary Hypertension  
g. Esophageal Rupture  
h. Costochondritis  
i. Hypovolemia

**II. UNSTABLE ANGINA**

**Pathophysiology**  
Partially occluding thrombus

**Assessment**

**History**

a. Assessment of Angina: PQRST Assessment  
   P: Pain, Placement, Provocation  
   Q: Quality, Quantity  
   R: Radiation, Relief  
   S: Severity, Systems (nausea, sweaty, dizziness)  
   T: Timing (when it started, how long did it last)  
b. Medical History  
c. Medications: Prescription, Over the Counter, Dietary Supplements  
d. Social History  
e. Family History  
f. Major Risk Factors of Atherosclerosis

**12-Lead Electrocardiogram (ECG)**

*Note: Patient can have a ST Segment Elevation Myocardial Infarction (STEMI) or a non-ST Segment Elevation Myocardial Infarction (non-STEMI). All situations are unique and the treatment team must look at entire presentation.*

a. Ischemia: ST Segment Depression  
b. Injury: ST Segment Elevation  
c. Infarction: Q waves  
d. **Brugada Syndrome**: Rare genetic cardiac rhythm disease. Intermittent ST segment elevation in V1-V3 (Brugada’s sign). May lead to syncope, and even sudden cardiac death. More common to present during sleep. More common in males. Tx is AICD
Acute MI 12 lead

<table>
<thead>
<tr>
<th>Location</th>
<th>Indicative Leads</th>
<th>Reciprocal Leads</th>
<th>Coronary Arteries</th>
<th>Major Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td>Leads V1, V2, V3, V4</td>
<td>Leads II, III, aVF</td>
<td>LAD</td>
<td>Cardiogenic Shock, Bundle Branch Blocks, Vent Dysrhythmias</td>
</tr>
<tr>
<td>Inferior</td>
<td>Leads II, III, aVF</td>
<td>Leads I, aVL</td>
<td>RCA</td>
<td>Bradycardia, Heart Blocks</td>
</tr>
<tr>
<td>Lateral</td>
<td>Leads I, aVL, V5, V6</td>
<td>Leads II, III, aVF</td>
<td>Circumflex</td>
<td>Heart Blocks in some</td>
</tr>
<tr>
<td>Septal</td>
<td>Leads V1, V2</td>
<td>Leads V5, V6</td>
<td>RCA</td>
<td>Bundle Branch Blocks</td>
</tr>
<tr>
<td>Posterior</td>
<td>Leads V7, V8, V9</td>
<td>Leads V1, V2</td>
<td>RCA or Circumflex</td>
<td>Heart Blocks</td>
</tr>
</tbody>
</table>

**Biochemical Cardiac Markers**

**Creatine Kinase (CK)**

CK total:
- Male 60 – 170 U/L
- Female 40 – 140 U/L

This enzyme is important in the breakdown of creatine to creatinine. It increases in the serum when muscle damage has occurred. Three CK isoenzymes have been identified; CK-I BB from the brain tissue and smooth muscle, CK-II MB from heart tissue and CK-III MM from muscle tissue. CK serum levels will begin to rise 3-6hr after chest pain, peak in 12-24 hrs and return to normal in 2-3 days.

Isoenzymes:
- CK I BB 0 – 1%  
- CK II MB < 3 - 6%  
- CK III MM 95 – 100%

**Troponin**

cTn T: < 0.1 mcg/L  
cTn I: < 3.1 mcg/L

The troponin complex is found on cardiac and skeletal muscle. Troponin C, T, & I are proteins that work in synchrony to regulate the force and speed of muscle contraction. These proteins modulate the interaction of actin and myosin. The amino acid structures of cardiac troponin T & I (cTn T & cTn I) are uniquely different than skeletal muscle. During periods of cardiac ischemia, intracellular troponin will leak out of the cell. Troponin levels can be detected within 3-5hrs after chest pain, cTnT will peak in 4-6hrs and cTnI 14-18hrs, and cTnT will return to normal 21 days and cTnI in 5-7 days.

**Myoglobin**

50 – 120 mcg/mL

Carbonic Anhydrase III (CA-III): 13 – 29 mcg/L
Myoglobin is a heme protein located on cardiac and skeletal striated muscle. Due to its low molecular wt. it is released very rapidly from the muscle after an ischemic event (faster than troponin or CK-MB). Serum levels will rise within 2 hrs of chest pain, peak in 3-15hrs and return to normal levels in 2 days. Because there is not a cardiac specific myoglobin, many non-cardiac events may cause an elevation. Carbonic Anydydrase III (CA-III) is another cytoplasmic protein found primarily in skeletal muscle. In skeletal muscle damage both CA III and myoglobin rise. In cardiac muscle damage there is only a rise in myoglobin. Therefore a rise in the myoglobin/CA III ratio is more indicative of an AMI than just an elevated myoglobin. A ratio of \( \geq 3.21 \) is considered abnormal and indicates for cardiac damage.

**Early Risk Stratification**

Early identification of the cause and severity of the pain is essential in determining triage and appropriate therapy. The five factors from the patient’s history that increase the likelihood that the ischemia is from CAD are:

a. Nature of Symptoms  
b. Prior History of CAD  
c. Gender & Age  
d. Number of CAD Risk Factors

**Treatment**

Treatment should be initiated as quickly as possible, while assessment is being completed. Immediate general-treatment includes:

a. Oxygen at 4L/min  
b. Aspirin 160-325mg (chewed)  
c. Nitroglycerin SL or spray  
d. Morphine IV (if pain not relieved by NTG)  
e. “MONA” meets the patient (Morphine, Oxygen, Nitroglycerin, Aspirin)  
f. Clopidogrel (Plavix) 600 mg, or prasugel (Effient) 60mg, or Ticagrelor (Brilinta) 180mg now part of AHA ACS guidelines (2013)

Once assessment is complete, patient is identified as having characteristics for one of four categories:

a. Non-Cardiac Diagnosis  
b. Chronic Stable Angina  
c. Possible Acute Coronary Syndrome (ACS)  
d. Definite ACS

**Possible ACS**

a. Give Aspirin – may have already done so  
b. Consider Primary Coronary Intervention in Cath Lab  
c. Consider Antithrombin Tx  
   - ASA  
   - Glycoprotein IIb/IIIa Inhibitor: Abciximab (ReoPro), Eptifibatide (Integrelin), Tirofiban (Aggrastat)
III. ACUTE MYOCARDIAL INFARCTION

Pathophysiology
Completely occlusive thrombus

Assessment
a. History
b. Physical Examination
c. 12-lead Electrocardiogram: ST elevation
d. Biochemical Cardiac Markers

Treatment
a. Give triple anti-thrombin tx
b. NTG if pain present
c. If ST-segment elevation - evaluate for reperfusion
   • Thrombolytics
   • Percutaneous Coronary Interventions
   • Coronary Artery Bypass Grafting

Thrombolytic Agents
Thrombolytic agents have been proven to decrease mortality and complications of acute MI.

Therapeutic Uses
Being given IV, directly into peripheral clot, intracoronary & intracerebral
a. Acute Coronary Thrombosis
b. DVT
c. Massive Pulmonary Emboli
d. Adjunct to PCI
e. Thrombotic Stroke
f. Combination tx of thrombolytic agents and GP IIb/IIIa, UFH, & LMWH have been shown to increase long term perfusion, mortality & morbidity

Absolute Contraindications
a. Active Bleeding
b. Aortic Dissection
c. Cerebral Neoplasm
d. History of Intracranial Hemorrhage
e. Recent (within 2 mo) intracranial or intraspinal surgery or trauma
f. Cerebral Vascular Disease (aneurysm, arteriovenous malformation)
g. Bleeding Diathesis  
h. Severe Uncontrolled Hypertension (> 180/110)

**Relative Contraindications**  
a. Recent (within 10 mo) Major Surgery  
b. Recent (within 10 days) GI or GU bleeding  
c. High likelihood of Left Heart Thrombus (mitral stenosis or A-fib)  
d. Acute Pericarditis or Sub acute Bacterial endocarditis  
e. Significant Liver Dysfunction  
f. Pregnancy  
g. Diabetic Hemorrhagic Retinopathy

**Adverse Effects**  
a. Major Risk is for Bleeding  
b. Should major bleeding occur  
   • Stop infusion & other anticoagulants  
   • Anticipate immediate head CT if ICH suspected  
   • Administer cryoprecipitate, FFP, platelets  
   • Aminocaproic acid (Amicar)

**Interventional Cardiology**  
Percutaneous coronary interventions have increased in both number of procedures and success rates since the first balloon angioplasty was performed in 1977.

**Percutaneous Coronary Interventions (PCI)**  
a. Diagnostic Coronary Angiography  
b. Percutaneous Transluminal Coronary Angioplasty (PTCA)  
c. Coronary Stents

**Nursing Care Concerns**  
a. Pre Procedure  
   • BUN/Creat Levels  
   • Dye Allergy  
   • Hydration Status  
   • Anticoagulation and Antiplatelet Medications  
   • Rate & Rhythm  
   • Electrolyte Balance: Especially Potassium  
   • Limb Circulation  
b. Post Procedure/Potential Complications  
   • Myocardial Ischemia  
   • Stroke  
   • Groin Site Bleeding  
     o Arterial/venous sheaths  
     o Arterial Closure Devices  
   • Distal Circulation  
   • Dysrhythmias
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- Coronary Artery Spasm
- Abrupt Closure/Restenosis
- Coronary Artery Dissection
- Peripheral Vascular Complication
- Discharge Education

**Coronary Artery Bypass Grafting**

**Purpose**  
Revascularize the Heart.

**Procedure**  
a. With the use of cardiopulmonary bypass, hypothermia and cardioplegia the heart is made motionless and bloodless.
b. Grafts are used to supply blood distal to occlusion.
c. Cannulation sites for bypass are typically the aorta & RA.
d. Minimally-invasive approach.
e. Smaller sternal incisions and non-sternal approaches. On and off bypass.

**Graft Option**  
a. Saphenous Vein  
b. Internal Mammary  
c. Radial Artery  
d. Gastric Artery

**Post Op Care: (for PCCN >48 hr post op care)**  
a. Pain  
b. Volume Overload  
c. MI  
d. Stroke  
e. Dysrhythmias  
f. Infection  
g. Decrease CO  
h. Impaired Gas Exchange  
i. Impaired Work of Breathing  
j. Hypoperfusion Complications

**Additional Nursing Concerns**  
a. Pain  
b. Immobility  
c. Risk for Infection  
d. Life Style Modification  
e. Discharge Education  
f. Nutrition
Treatment Continued
a. Give β-blocker
b. Once reperfused evaluate myocardial damage and provide post MI care

Complications of AMI
a. Cardiogenic Shock: infarction of ≥ 40% of the left ventricle
   • Hypotension: SPB < 100mmHg
   • Pulmonary Edema
   • Low Cardiac Output
   • Cardiogenic Pulmonary Edema
   • S&S of Poor Peripheral Perfusion
b. Arrhythmias Associated with Ischemia, Infarction & Reperfusion

Treatment Goals for Cardiogenic Shock
a. Assist Contractility
b. Alleviate Cause of Failure
c. Fluid
d. Pharmacological Agents
e. Coronary Reperfusion
f. Mechanical Assist