Acute Coronary Syndrome (ACS)

- The spectrum of clinical conditions ranging from:
  - Unstable angina
  - Non-ST elevation MI
  - ST elevation MI
- Characterized by the common pathophysiology of a disrupted atherosclerotic plaque
Slide 4

Differential Diagnosis

• Non-cardiac
  1. Vascular
  2. Pulmonary
  2. Esophagus
  3. Musculoskeletal
  4. Infectious

Slide 5

Acute Coronary Syndromes

• Unstable Angina
• Non-ST-Segment Elevation MI (NSTEMI)
• ST-Segment Elevation MI (STEMI)

STEMI requires evaluation for acute reperfusion intervention

Slide 6

Pathogenesis of ACS

Vulnerable Plaque
  • Platelet aggregation, adhesion and activation
  • Thrombus formation
  • Vasospasm

Plaque rupture (55-80%)
Exertion
BP, HR
Vasoconstriction

STEMI
Incomplete occlusion
Distal embolization

Unstable Angina
NSTEMI
Complete occlusion
STEMI
Slide 7

Evolution of Infarction/Necrosis

Coronary Artery Occlusion: The Evolution of Infarction
Progression of myocardial necrosis with time since occlusion

- 0 h
- 4 h
- 6-12 h

Normal myocardium

"At risk" myocardium, ischemic but viable

Necrosis starting subendocardially

Normal myocardium

"At risk" myocardium, ischemic but viable

Necrosis extending towards subepicardium

Slide 8

"Time is muscle"

Myocardial Infarction is a true emergency in cardiac care.

Slide 9

COMPLICATIONS OF INFARCTION

- Ventricular Septal Rupture
- Papillary Muscle Rupture
- Ventricular Free Wall Rupture
- Left Ventricular Thrombus
Slide 10

Acute Coronary Syndromes

- Treatment Goals
  - Decrease myocardial oxygen demand
  - Remove physical/psychological stressors
  - Relieve pain
  - Reduce workload of the heart (BP, HR)

- Inhibit further clot formation

- Rapid identification/diagnosis

- Transport for reperfusion therapy if needed

- Decrease amount of myocardial necrosis

- Preserve LV function

- Prevent major adverse cardiac events

- Treat life threatening complications

Slide 11

Evaluation

- Efficient & direct history/physical
- Initiate stabilization interventions

Plan for moving rapidly to indicated cardiac care

Directed Therapies are Time Sensitive!

Slide 12

Ischemic Coronary Syndromes

- Unstable Angina
  - Prolonged chest pain/ischemic symptoms...an atypical presentation of angina without EKG or laboratory evidence of AMI (Injury)
  - Usually associated with significant or progressing occlusion of a coronary artery or severe vasospasm
  - Considered “Pre-infarction Angina”
Diagnosis of Unstable Angina

- Patients with typical angina
  - Increased in severity or duration
  - Has onset at rest or at a low level of exertion
  - Unrelieved by the amount of nitroglycerin or rest that had previously relieved the pain

- Patients not known to have typical angina
  - First episode with usual activity
  - Prolonged pain at rest

Targeted Physical

- Examination
  - Vitals
  - Cardiovascular system
  - Respiratory system
  - Abdomen
  - Neurological status

- Recognize factors that increase risk
  - Hypotension
  - Tachycardia
  - Pulmonary rales, JVD, pulmonary edema
  - New murmurs/heart sounds
  - Diminished peripheral pulses
  - Signs of stroke

Directed Medical Therapy

MONA + BAH

- Morphine
  - Analgesia
  - Reduce pain/anxiety—decrease sympathetic tone, systemic vascular resistance and oxygen demand
  - Careful with hypotension, hypovolemia, respiratory depression

- Oxygen
  - Up to 70% of ACS patient demonstrate hypoxemia
  - May limit ischemic myocardial damage by increasing oxygen delivery
**Slide 16**

- **Nitroglycerin**
  - Analgesia—titrate infusion to keep patient pain-free
  - Dilates coronary vessels—increased blood flow
  - Reduces systemic vascular resistance and preload
    - Careful with hypotension, bradycardia, tachycardia, RV infarction
    - PO nitrates include Imdur (chronic use) or SL nitro or a patch

- **Aspirin** (180-325mg chewed & swallowed)
  - Irreversible inhibition of platelet aggregation (blocks prostaglandins)
  - Stabilizes plaque and arrests thrombus
  - Reduce mortality in patients with STEMI
  - Careful with active PUD, hypersensitivity, bleeding disorders

**Slide 17**

- **Beta-Blockers**
  - Lower heart rate—decreases its work
  - Studies show reduction in risk of progression to MI in patients with threatening or evolving MI symptoms
  - Be aware of contraindications (CHF, Heart block, Hypotension)
  - Reassess for therapy as contraindications resolve

- **ACE-Inhibitors / ARB**
  - Start in patients with anterior MI, pulmonary congestion, LVEF < 40% in absence of contraindication/hypotension
  - Start in first 24 hours
  - ARB as substitute for patients unable to use ACE-I

**Slide 18**

- **Heparin**
  - LMWH or UFH (max 5000u bolus, 1000u/hr)
  - Indirect inhibitor of thrombin
  - Less supporting evidence of benefit in era of reperfusion
  - Adjunct to surgical revascularisation and thrombolytic / PCI reperfusion
  - 24-48 hours of treatment
  - Coordinate with PCI team (UFH preferred)
  - Used in combo with aspirin and/or other platelet inhibitors
Additional medication therapy

- **Clopidogrel** (Plavix)
  - Irreversible inhibition of platelet aggregation
  - Used in support of PTCA intervention or if unable to take aspirin
  - 3 to 12 month duration depending on scenario

- **Glycoprotein IIb/IIIa inhibitors**
  - 
  - Inhibition of platelet aggregation at final common pathway
  - In support of PTCA intervention as early as possible

---

GP IIb/IIIa Receptor Final Pathway to Platelet Aggregation

- GP IIb/IIIa receptors on activated platelets undergo a conformational change allowing recognition and binding of fibrinogen
- Fibrinogen “acts like glue”, bridging GP IIb/IIIa receptors on adjacent platelets, leading to platelet aggregation

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Additional medication therapy

- **Aldosterone blockers**
  - Post-STEMI patients
    - No significant renal failure (Cr < 2.5 men or 2.0 for women)
    - No hyperkalemia > 5.0
    - LVEF < 40%
    - Symptomatic CHF or DM
Slide 22

EKG assessment

- ST Elevation or new LBBB
  - STEMI

- ST Depression or dynamic T wave inversions
  - NSTEMI

- Non-specific ECG
- Unstable Angina

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Slide 23

Risk Stratification

STEMI Patient?

- YES
  - Assess for reperfusion
  - Select & implement reperfusion therapy
  - Directed medical therapy

- NO
  - UA or NSTEMI
    - Evaluate for Invasive vs. conservative treatment
    - Directed medical therapy

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Slide 24

ST-Segment Elevation MI
**Slide 25**

New LBBB

- QRS > 0.12 sec
- L Axis deviation
- Prominent R wave V1-V3
- Prominent S wave 1, aVL, V5-V6
  - with T-wave inversion

**Slide 26**

When to Order Myocardial Injury Tests?

- When you suspect myocardial injury clinically
- Clearly when you have EKG findings specific for MI
  - St segment elevations
  - New LBBB
  - Less than 50% of patients in the ED with myocardial injury will have a diagnostic EKG

**Slide 27**

Cardiac markers (muscle damage)

- Troponin (T, I)
  - Very specific and more sensitive than CK
  - Rises 4-8 hours after injury
  - May remain elevated for up to two weeks
  - Can provide prognostic information
  - Troponin T may be elevated with renal dz, polymyositis

- CK-MB isoenzyme
  - Rises 4-6 hours after injury and peaks at 24 hour
  - Remains elevated 36-48 hours
  - False positives with exercise, trauma, muscle dz, DM, PE and use of IMA for bypass
STEMI cardiac care

**STEP 1: Assessment**

- Time since onset of symptoms
  - 90 min for PCI / 12 hours for fibrinolysis
- Determine if fibrinolysis candidate
  - Meets criteria with no contraindications
- Determine if PCI candidate
  - Based on availability and time to Balloon Rx

**Fibrinolysis preferred if:**
- ≤3 hours from onset
- PTCA not available/delayed
- Door to balloon > 90 min
- No contraindications

**PTCA preferred if:**
- Available
- Door to balloon < 90 min
- Fibrinolysis contraindications
- Late Presentation > 3 h
Slide 31

Absolute contraindications for fibrinolysis therapy in patients with acute STEMI

- Known structural cerebral vascular lesion (e.g., AVM)
- Known malignant intracranial neoplasm
- Ischemic stroke within 3 months
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed-head or facial trauma within 3 months

Slide 32

Relative contraindications for fibrinolysis therapy in patients with acute STEMI

- History of chronic, severe, poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (SBP greater than 180 mm Hg or DBP greater than 110 mmHg)
- History of prior ischemic stroke greater than 3 months, dementia, or known intracranial pathology not covered in contraindications
- Traumatic or prolonged (greater than 10 minutes) CPR or major surgery (less than 3 weeks)
- Recent (within 2-4 weeks) internal bleeding
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulants: the higher the INR, the higher the risk of bleeding

Slide 33

Comparing outcomes
Comparison of Mechanical Intervention and Thrombolysis

<table>
<thead>
<tr>
<th>Mechanical Intervention</th>
<th>Thrombolysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Advantages:</td>
<td></td>
</tr>
<tr>
<td>Scoring very quickly</td>
<td>More available with initial access</td>
</tr>
<tr>
<td>Reduced mortality</td>
<td>Reduced mortality on embolism and reperfusion</td>
</tr>
<tr>
<td>Lower incidence</td>
<td>Disadvantages:</td>
</tr>
<tr>
<td>Lower mortality</td>
<td>Low incidence of stroke</td>
</tr>
<tr>
<td>Lower incidence</td>
<td></td>
</tr>
</tbody>
</table>

Disadvantages:
- Critical blockade on cardiac activity
- Immediate bleeding
- Limited access
- Longer time to treatment

Acute Coronary Syndromes

- **Unstable Angina**
  - May have Typical or Atypical Signs & Symptoms
    - Atypical Presentation
      - Increased frequency or duration of episodes
      - Slight with less action than normal, pain at rest
      - Increased severity of symptoms
      - Requires greater number of NTG tablets to relieve symptoms, maybe no relief
      - They may look sick

Unstable angina/NSTEMI cardiac care

- Evaluate for conservative vs. invasive therapy based upon:
  - Risk of actual ACS
  - TIMI risk score
  - ACS risk categories per AHA guidelines

Low   Intermediate   High
**Risk Stratification to Determine the Likelihood of Acute Coronary Syndrome**

<table>
<thead>
<tr>
<th>Assessment</th>
<th>Findings indicating HIGH likelihood of ACS</th>
<th>Findings indicating INTERMEDIATE likelihood of ACS in absence of high-likelihood findings</th>
<th>Findings indicating LOW likelihood of ACS in absence of high- or intermediate-likelihood findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td>Evidence in support of ischemic heart disease</td>
<td>Coronary artery disease, including myocardial infarction</td>
<td>No evidence of coronary artery disease, including myocardial infarction</td>
</tr>
<tr>
<td>Physical examination</td>
<td>New or presumably new transient mitral regurgitation, hypotension, diaphoresis, pulmonary edema or rales</td>
<td>Extracardiac vascular disease</td>
<td>Chest discomfort reproduced by palpation</td>
</tr>
<tr>
<td>ECG</td>
<td>New or presumably new transient ST-segment deviation (&gt; 0.05 mV) or T-wave inversion (&gt; 0.2 mV) with symptoms</td>
<td>Fixed Q waves</td>
<td>Abnormal ST segments or T waves not documented to be new</td>
</tr>
<tr>
<td>Ser. markers</td>
<td>Elevated cardiac troponin T or I, or elevated CK-MB</td>
<td>Normal</td>
<td>Normal</td>
</tr>
</tbody>
</table>

**TIMI Risk Score**

Predicts risk of death, new/recurrent MI, need for urgent revascularization within 14 days

**Invasive therapy option**

UA/NSTEMI

- Coronary angiography and revascularization within 12 to 48 hours after presentation to ED
- MONA + BAH (UFH)
- Clopidogrel
  - 20% reduction death/MI/Stroke – CURE trial
  - 1 month minimum duration and possibly up to 9 months
- Glycoprotein IIb/IIIa inhibitors
Conservative Therapy for UA/NSTEMI

- Early revascularization or PCI not planned
- MONA + BAH (LMW or UFH)
- Clopidogrel
- Glycoprotein IIb/IIIa inhibitors
  - In certain circumstances (planning PCI, elevated TnI/T)
- Surveillance in hospital
  - Serial ECGs
  - Serial Markers
Case study

• (STEMI)
  – Treatment
  • O2 @ 4l
  • 4 mg IV MS
  • Baby ASA chewed
  • 2.5 mg IV Lopressor X 2 HR 80 BP 140/80
  • Nitroglycerin IV infusion
  • Bolused 5000u Heparin and drip at 1000u/hr
  • PTCA
  • 38 minutes door to balloon time

Angioplasty

• Success rate:
  – 90% of lesions attempted
  – This 90% includes repeat procedures

Major limitation:
  – Restenosis

Factors associated with higher re-occlusion rate:
  • Diabetes
  • Small lumenal diameter
  • Longer more complex lesions
  • Lesions in the left anterior descending artery
Treatment

Stenting

- Stent is introduced into a blood vessel on a balloon catheter and advanced into the blocked area of the artery.
- Balloon is then inflated and causes the stent to expand until it fits the inner wall of the vessel, conforming to contours as needed.
- Balloon is then deflated and drawn back.
- Stent stays in place permanently, holding the vessel open and improving the flow of blood.

Drug-eluting stents

- A drug-eluting stent is coated with an agent that inhibits restenosis.
  - The Cypher stent is coated with an antibiotic called sirolimus (also called rapamycin), which is slowly released into the artery for about 30 days after implantation.
  - Sirolimus is a "cytostatic" drug, which means it inhibits cell growth and division, and T-cell activation and proliferation. T-cells initiate an inflammatory response that commonly follows implantation, and inflammation can lead to restenosis.
- Results...
Angioplasty/Stent Placement

- Angioplasty with or without stent placement
  - Aspirin for life

- Angioplasty with stent placement
  - Clopidogrel (Plavix) for six to 12 months or longer in some cases.

Secondary Prevention

- Disease
  - HTN, DM, HLP

- Behavioral
  - Smoking, diet, physical activity, weight

- Cognitive
  - Education, cardiac rehab program

- Blood Pressure
  - Goals: <140/90 or <130/80 in DM/CKD
    - Maximize use of beta blockers & ACE inhibitors

- Lipids
  - LDL < 100 (70); TG < 200
    - Maximize use of statins; consider fibrates/niacin first line for TG>500; consider omega-3 fatty acids

- Diabetes
  - A1c < 7%
Slide 52

Secondary prevention

- Smoking cessation
  - Cessation-class, meds, counseling
- Physical Activity
  - Goal 30 - 60 minutes daily
  - Risk assessment prior to initiation
- Diet
  - DASH diet, fiber, omega-3 fatty acids
  - <7% total calories from saturated fats

Slide 53

Medication Checklist after ACS

- Antiplatelet agent
  - Aspirin* and/or Clopidorgrel
- Lipid lowering agent
  - Statin*
  - Fibrate / Niacin / Omega-3
- Antihypertensive agent
  - Beta blocker*
  - ACE-I*/ARB
  - Aldactone (as appropriate)

Slide 54

Summary

- ACS includes UA, NSTEMI, and STEMI
- Management guideline focus
  - Immediate assessment/intervention (MONA+BAH)
  - Risk stratification (UA/NSTEMI vs. STEMI)
  - RAPID reperfusion for STEMI (PCI vs. Thrombolytics)
  - Conservative vs Invasive therapy for UA/NSTEMI
- Aggressive attention to secondary prevention initiatives for ACS patients
  - Beta blocker, ASA, ACE-I, Statin
Prevention news...
From 1994 to 2004 the death rate from coronary heart disease declined 33%... But the actual number of deaths declined only 18%.

Getting better with treatment… But more patients developing disease – need for primary prevention focus.