Internal Medicine Emergency Lecture Series
Cardiovascular Emergencies
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Introduction
- Review of cardiovascular emergencies
  - Acute Coronary Syndromes
    - STEMI
    - UA/NSTEMI
  - Congestive Heart Failure
  - Aortic dissection
  - Digoxin toxicity
  - Tamponade
  - Summary and Questions

Acute Coronary Syndromes
- ACS - Spectrum of the same disease
- ST Elevation Myocardial Infarction (STEMI)
- Non-ST Elevation Myocardial Infarction (NSTEMI)
- Unstable Angina (UA)

What do you do when your patient has chest pain?
- Ask the nurse what she/he would do in this situation
- Give the nurse your supervisor’s pager #
- Order EKG, ASA, NTG, beta-blockers, heparin, Integritin, morphine, and thrombolytics (just to be safe)
- Go home sick because you now have chest pain
- Take a deep breath and think
Pathophysiology

- Plaque fissure and rupture
- Vessel occlusion – fibrin, platelet aggregates, red blood cells
- Other causes
  - Dynamic obstruction
  - Progressive mechanical obstruction
  - Inflammation/infection
  - Secondary unstable angina (blood loss, SVT, hypotension, thyrotoxicosis, etc.)

Atherosclerotic Lesions
Arterial Disease Progression

- Fatty Streak
- Intermediate Lesion
- Fibrous Plaque
- Plaque Rupture - Advanced Lesion

STEMI

- About 1.5 million patients have AMI in U.S. each year
- In U.S. CAD costs more than $60 billion/yr
- Large indirect cost due to lost productivity
- Death rate has dropped over last 15 years, but still about 30 percent mortality.
- 50% of deaths due to AMI occur within 1 hour of event (mostly due to arrhythmias)
- About 6% mortality for those arriving in hospital treated with thrombolytics

Aspects of MI by Different Techniques

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Differential Diagnosis of ST-Segment Elevation

- Acute myocardial infarction
- Post MI aneurysm
- Acute pericarditis
- Normal variant
- LVH/LBBB (usually in V1-2 or V3)
- Myocarditis
- Tumor invading LV
Differential Diagnosis of ST-Segment Elevation

- Trauma to LV
- Hypothermia/Osborne waves
- After DCCV
- Intracranial hemorrhage
- Hyperkalemia or hypercalcemia
- Brugada pattern
- Type IC antiarrhythmics
- J-pt. elevation
Options for therapy
- Thrombolytics
- Primary Percutaneous Intervention
GISSI, TIMI, TAMI, ASSENT, MITI, GUSTO, LATE, ISIS, SPEED, DANAMI, InTIME, INJECT, RAPID, COMPASS, SHOCK, etc...
Currently first line treatment at UNMC and the VA is thrombolysis unless contraindications

Indications for thrombolytics
- Chest pain consistent with AMI
- EKG – ST elevation > 0.1mV in 2 contiguous leads or presumed new LBBB
- Cardiac enzyme elevation
- < 6 hrs – most benefit
- 6-12 hrs – still beneficial
- > 12 hrs diminishing benefit, may be useful for selected patients

Absolute contraindications to thrombolytics
- Active internal bleeding
- Suspected Aortic dissection
- Recent head trauma or known intracranial tumor
- History of hemorrhagic CVA or cerebrovascular event within 1 year
- Major surgery or trauma < 2 wks prior

Relative contraindications to thrombolytics
- BP > 180/100 on 2 readings
- H/O chronic severe HTN
- Active PUD
- H/O CVA or intracerebral pathology not covered in contraindications
- Known bleeding diathesis or on therapeutic doses of coumadin (INR 2 – 3)
- Prolonged traumatic CPR or recent trauma (2 to 4 weeks)
- Recent (2 to 4 weeks) internal bleeding
- Pregnancy
- Prior streptokinase/anstreplase within 2 years
- Noncompressible vascular punctures

Age > 75 – Risk is high with or without therapy. Advantage is less in this age group, but still have 10 lives saved per 1000 treated

Time is muscle
Infarct size is important in prognosis
- Larger area of infarct leads to decreased EF
- Mortality is higher with larger area of infarction
Desired door-to-drug time is < 30 minutes
General concepts
- Open artery early
- Reduce myocardial energy requirements (i.e. beta blockers, lower blood pressure, ensure adequate oxygenation)
### Time and Thrombolytics

**Graph:**
- Lives saved per 1000 treated
- Time to treatment from symptom onset

#### Comparison of Therapy
- **STEMI**
  - Other therapies:
    - ASA
    - Heparin - UFH or LMWH
    - Beta blockers as tolerated (i.e. metoprolol 5mg IV q 15 min X 3)
    - NTG
    - Morphine
    - Oxygen
    - Monitor rhythm, vitals closely
    - ACE-I for reduced LVEF
    - HMG Co-A reductase inhibitors during hospitalization

- **NSTEMI/UA**
  - UA
    - Rest angina
    - New-onset severe angina
    - Accelerating symptoms
  - NSTEMI UA + biochemical markers (troponins)
  - Risk Assessment
    - History
    - Physical Exam
    - X-ray
    - Laboratory

#### Complications of STEMI
- **Cardiogenic shock**
  - Occurs in about 10% of all AMI
  - 80% fatal with conservative management
  - PCI (percutaneous intervention) is primary treatment
  - May need IABP (intra-aortic balloon pump), inotropes, fluids

- **Ventricular rupture**
  - Ventricular free wall
  - Ventricular septum
  - Treatment is emergency cardiac surgery

- **Acute Mitral Regurgitation**
  - Papillary rupture or ischemia
  - May present up to 1 week after AMI
  - Murmur variable
  - MV annular dilatation from LV failure
  - Echocardiography is test of choice
  - Therapy is urgent cardiac surgery

- **Heart Block**
  - Anterior MI
    - Causes damage to infra-nodal conduction system
    - With 2nd degree AVB need to pace at early stage because may rapidly change to 3rd degree block
  - Inferior MI
    - May cause heart block because of activation of cardiovascular reflexes or due to injury to AVN.
    - Usually pace heart block for symptoms of hypoperfusion

- **Complications of STEMI**
  - Due to myocardial infarction
  - Treatment is necessary to prevent further complications
  - Need to understand the patient's condition to provide appropriate care
NSTEMI/UA

- TIMI Risk Score
  - Age > 65
  - More than 3 coronary risk factors
  - Prior angiographic coronary obstruction
  - ST-segment deviation
  - > 2 anginal events in past 24 hours
  - Use of ASA within last 7 days
  - Positive cardiac serum markers

- Risk of adverse outcome (death, reinfarction, or recurrent ischemia requiring revascularization)
  - 5% with TIMI Risk Score of 0 or 1
  - 41% with TIMI Risk Score of 6 or 7

Medical Management

- Aspirin
- Nitroglycerin
- Heparin
- GP IIb/IIIa inhibitors
- Beta blockers
- HMG Co-A reductase inhibitors
- Clopidogrel
- RISC, TIMI, ATACS, FRISC, CATURE, OASIS, PRISM, VANQWISH, ESSENCE, PURSUIT, PARAGON, ESSENCE, etc...

Guidelines for Classification

- Class I
  - Conditions for which there is evidence and/or general agreement that a given procedure/therapy is useful and effective
- Class II
  - Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of performing the procedure/therapy
- Class IIa
  - Weight of evidence/opinion is in favor of procedure/therapy
- Class IIb
  - Usefulness/efficacy is less well established by evidence/opinion
- Class III
  - Conditions for which there is evidence and/or general agreement that a procedure/therapy is not useful and in some cases harmful

Aspirin

- Irreversibly inhibits cyclooxygenase preventing synthesis of thromboxane A$_2$
- Class I indications
  - ASAP upon admission unless intolerance
  - 30% to 40% nonresponders in pts with ACS

Clopidogrel

- Class I indications:
  - Hospitalized patients unable to take ASA due to hypersensitivity or major GI bleed
  - Hospitalized patients to undergo early noninterventional approach
  - In patients for whom PCI is planned and are not at high risk for bleeding
Clopidogrel

- CURE trial
  - 12,562 ACS (UA/NSTEMI) patients. ASA vs. ASA + clopidogrel
  - 3 to 12 month follow up
  - Composite endpoint of CV death, MI, or stroke: 11.47% vs. 9.28%, p = 0.00005
  - No significant difference in CV death, refractory ischemia
  - Significant differences in MI, stroke, in-hospital refractory ischemia
  - Very low percentage of patients received IIb/IIIa inhibitors

- 10 to 15% of patients with ACS will go to CABG

Glycoprotein IIb/IIIa Inhibitors

- **Class I Indications**
  - In addition to ASA and heparin in patients planned for catheterization and PCI

- **Class IIa**
  - In addition to ASA and LMWH or UFH in patients with ongoing ischemia, elevated troponin, or with other high-risk features in whom invasive management not planned

- **Class IIb**
  - Patients without ongoing ischemia who have no other high-risk features and in whom PCI not planned

- **Class III**
  - Abciximab in patients whom PCI not planned

Heparin

- **Class I indication**
  - Well established efficacy in ACS
  - Highly variable dose-response relationship
  - May be reversed if necessary
  - Short half-life
  - May stimulate platelet activation
  - Thrombocytopenia is a potential complication

- **Low Molecular Weight Heparin**
  - Class I indication
  - In addition to anti-platelet therapy

  - **Class IIa**
    - Preferable to UFH in the absence of renal failure and unless CABG is planned (Recent JAMA article refutes)

  - **Class IIb**
    - Lower incidence of thrombocytopenia
    - Need to be aware of dose adjustment for renal failure
  
  - **Class III**
    - Cannot be reversed

Beta Blockers in ACS

- **Class 1 indication**
  - Limited randomized trial data
  - Practice extrapolated from other experience in ischemic syndrome

Invasive Therapy

- **Class 1 indication**
  - Recurrent angina/ischemia at rest or with low-level activities despite intensive anti-ischemic therapy
  - Elevated troponin
  - New or presumably new ST-segment depression
  - Recurrent angina with CHF symptoms, S3, pulmonary edema, worsening rales, worsening MR
  - High-risk findings on noninvasive test
  - Depressed LV systolic function
  - Hemodynamic instability
  - Sustained VT
  - PCI within 6 months
  - Prior CABG

Braunwald et al. 2002, ACC/AHA Practice Guidelines
Invasive Therapy

- **Class IIa indication**
  - Patients with repeated presentations for ACS despite therapy and without evidence for ongoing ischemia or high risk
- **Class III**
  - Patients with extensive comorbidities in whom risks of revascularization are not likely to outweigh the benefits
  - Acute chest pain and low likelihood of ACS
  - Patients not willing to consent to revascularization regardless of findings

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Troponin level and mortality

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Acute Coronary Syndrome

**ST Elevation MI**
- Primary PCI/ Thrombolytics
- Evaluate Risk Factors
- Medical Therapy Based on Risk
- Functional Study vs. Cath

**UA/ Non-ST Elevation MI**
- Evaluate Risk Factors
- Medical Therapy Based on Risk
- Functional Study vs. Cath

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Patient Presentation

65 y/o male with a history of anterolateral MI 3 years ago. DM, HTN presents to ER with rapid onset of shortness of breath at 9 PM while taking garbage out to end of driveway. Squad called and taken to nearest ER. Currently on 6 l/min via nasal cannula, SpO2 = 87%.

- Home meds include enalapril, metoprolol, ASA, glyburide, digoxin, furosemide
- Physical Exam: BP 210/112 HR 110, RR 24, T 37, SpO2 89%
  - Gen: Moderate respiratory distress
  - Neck: JVD 14 cm
  - Heart: Tach regular, + S4
  - Lungs: Crackles 1/3 of lower lungs b/l
  - Ext: No edema
  - CXR: pulmonary edema with cardiomegaly
  - EKG: Sinus tachycardia with no ST changes

Code called in the Unit and your supervisor offers to go to code while you dictate and write orders on this patient.

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Acute CHF

**Common patient presentation:**
- Shortness of breath, dyspnea on exertion, paroxysmal nocturnal dyspnea, orthopnea, swelling, weight gain
- JVD, PND, S3, S4, parasternal lift, crackles, edema, ascites, or hepatomegaly
- Pulmonary edema with cardiomegaly on CXR
- Elevated BNP (CHF peptide) level, EKG changes depending on etiology

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Acute CHF

**Causes:**
- Ischemia
- Complications of MI (i.e. ventricular rupture, arrhythmia, acute MR)
- Arrhythmia – high degree AVB, tachyarrhythmia
- Tamponade
- Pulmonary embolus
- Myocarditis
- Acute or worsening of valvular lesions
- Acute increase in BP
- Acute renal failure
- Peripartum
- Infiltrative diseases – sarcoid, amyloid
**Acute CHF**

**Framingham Criteria for Diagnosis of CHF**
- **Major**
  - PND or Orthopnea
  - JVD
  - Rales
  - Cardiomegaly
  - Acute pulmonary edema
  - S3
  - HJR
  - Circulation time > 25 seconds

**Minor Criteria**
- Edema
- Night cough
- DOE
- Hepatomegaly
- Pleural effusion
- HR > 120
- Vital capacity < 1/3 of maximum

2 major or 1 major and 2 minor criteria for definite CHF

**Precipitating Factors**
- Noncompliance with diet or medications
- Arrhythmia
- Infection
- Pulmonary embolism
- Anemia or other high output state
- Co-morbidity such as renal failure, hypothyroidism, lung disease
- Ischemia
- Hypertension
- Alcohol or street drugs
- Other cardiotoxins such as chemotherapy agents

**Treatment for CHF (Depressed LVEF)**
- Sitting position
- O2 via any method (NC, mask, BiPap, Ventilator)
- IV loop diuretics (Lasix, Bumex, etc.)
- NTG drip
- MSO4 IV
- IV nesiritide, dobutamine, milrinone
- Foley catheter
- Invasive monitoring - Swan-Ganz catheter, arterial blood pressure line

**Aortic Dissection**

**Typical features:**
- Acute onset of severe pain in chest, back, abdomen
- HTN
- Aortic diastolic murmur
- Pulse deficits
- Acute MI from coronary involvement
- Syncope from tamponade
- CHF from severe AR

**CXR** – useful as first screen and may show widening of mediastinum, deviation of mediastinum to the right

**Definitive diagnosis**
- TEE
- CT angiogram
- MRI
- Aortography
Aortic Dissection

- Surgery for Type I or II (involving the ascending aorta)

Initial Treatment
- IV beta-blockers (propranolol, esmolol, metoprolol, labetalol)
- Sodium nitroprusside
- Goal SBP of 100 to 120 mmHg or lowest possible to perfuse organs
- Reduce HR to about 60 bpm
- Watch for acute AI, acute inferior MI, or neurologic changes, acute renal failure, limb ischemia (indicating extension of dissection into other vessels)

Digoxin Toxicity

- Most common symptoms include:
  - Nausea, vomiting
  - Drowsiness

- Possible arrhythmias
  - VT
  - Sinus bradycardia
  - Heart block
  - Paroxysmal atrial tachycardia with block

- If acute, can induce vomiting, perform gastric lavage, and give charcoal
- Digoxin-immune Fab for VT or VF, high grade AV block not responding to atropine, ingestion of high doses, serum level greater than 10ng/ml, or hyperkalemia
- May cause hyperkalemia – treat same way as isolated hyperkalemia
- High grade AV block can be treated with atropine and temporary pacing
- Treat ventricular arrhythmias with lidocaine, phenytoin, esmolol, magnesium, and synchronized DCCV if unstable

Tamponade

Clinical findings
- Fall in systemic arterial pressure
- Rise in systemic venous pressure
- Heart sounds distant, possible friction rub
- Tachycardia, tachypnea
- Pulsus paradoxus (do not send patient down for Echo before you check)
- Elevated JVD

- Malignancy
- Idiopathic pericarditis
- Uremia
- AMI
- Iatrogenic (procedures)
- Infectious/Tuberculosis
- Radiation
- Medications
- SLE
- Post pericardiotomy

- EKG
  - Electrical alternans indicating swinging motion of heart
  - May also occur in pericarditis (i.e. not 100% specific)
- CXR
  - May be completely normal unless fluid gradually accumulated (i.e. greater than 250 cc)
  - May have water bottle appearance
- Echocardiogram
  - RA diastolic collapse, RV early diastolic collapse, swinging heart, IVC plethora, Inspiratory decrease in MV inflow pattern

Diagnostic studies

- EKG
- CXR
- Echocardiogram
Tamponade

Treatment
- Hemodynamic support with IV fluids
- Pericardiocentesis – echo or fluoroscopy guided
- Pericardiotomy or pericardiectomy

Summary
- Evaluate the patient
- Develop a differential
- Think before you write orders

PROBLEMS
No matter how great and destructive your problem may seem now, remember, you’ve probably only seen the tip of the iceberg.