ISCHEMIC HEART DISEASE
Introduction

• Coronary heart disease (CHD) is the most common form of heart disease
• An estimated 330 000 people have a myocardial infarct each year
• Approximately 1.3 million people have angina each year
Introduction

- Disease of the coronary arteries is almost always due to atheroma and its complications — particularly thrombosis.
Myocardial Ischemia

• Results when there is an imbalance between myocardial oxygen supply and demand

• Most occurs because of atherosclerotic plaque with in one or more coronary arteries

• Limits normal rise in coronary blood flow in response to increase in myocardial oxygen demand
**Oxygen Carrying Capacity**

- The oxygen carrying capacity relates to the content of hemoglobin and systemic oxygenation.
- When atherosclerotic disease is present, the artery lumen is narrowed and vasoconstriction is impaired.
- Coronary blood flow cannot increase in the face of increased demands and ischemia may result.
Ischaemic Heart Disease

- Angina
  - Stable
  - Unstable
  - Prinzmetal’s

- Myocardial Infarction
  - NSTEMI
  - STEMI
### Acute Coronary Syndromes

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<td>Often ST depression</td>
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Quality

• Tightness, squeezing, heaviness, pressure, burning, indigestion or aching sensation
• It is rarely “PAIN”
• radiation
• Never: sharp, stabbing, prickly, spasmodic, or pleuritic
• Lasts a few seconds < 10 minutes
• Relieved by NTG s/l
• Levine Sign: clench fist to sternum
Signs & Symptoms accompany Angina

- Dyspnea, nausea, vomiting, diaphoresis resolve quickly after cessation of angina (autonomic activation)
- Angina is a diffuse sensation rather than discrete
- Myocardial ischemia may result in papillary muscle damage and mitral regurgitation
- Ischemic induced left ventricular wall motion abnormalities may be detected as an abnormal precordial bulge on chest palpation
- A transient S3 gallop and pulmonary rales = ischemic induced left ventricular dysfunction
Silent Ischemia

- Atypical chest pain, usually seen in diabetics and females.
- Asymptomatic episodes of myocardial ischemia.
- Detected by electrocardiogram and laboratory studies.
Myocardial Infarction

• Region of myocardial necrosis due to prolonged cessation of blood supply
• Results from acute thrombus at side of coronary atherosclerotic stenosis
• May be first clinical manifestation of ischemic heart disease or history of Angina Pectoris
Precipitants

- Exertion: walking, climbing stairs, vigorous work using arms, sexual activity
- Vasoconstriction: extremities, increased systemic vascular resistance, increased in myocardial wall tension and oxygen requirements
- After a heavy meal, sudden exposure to cold, emotional stress
Diagnostic Tests

• Blood tests include serum lipids, fasting blood sugar, Hematocrit, thyroid (anemias and hyperthyroidism can exacerbate myocardial ischemia)

• Resting Electrocardiogram: CAD patients have normal baseline ECGs
  – pathologic Q waves = previous infarction
  – minor ST and T waves abnormalities not specific for CAD
Electrocardiogram

• Electrocardiogram: is useful in diagnosis during cc: chest pain
• When ischemia results in transient horizontal or downsloping ST segments or T wave inversions which normalize after pain resolution
• ST elevation suggest severe transmural ischemia or coronary artery artery spasm which is less often
Normal or non-diagnostic ECG
ST Depression or Dynamic T wave Inversions
ST-Segment Elevation MI
ECG changes with MI
Exercise Stress Test

- Used to confirm diagnosis of angina
- Terminate if hypotension, high grade ventricular disrrhythmias, 3 mm ST segment depression develop
- (+): reproduction of chest pain, ST depression
- Severe: chest pain, ST changes in 1st 3 minutes, >3 mm ST depression, persistent > 5 minutes after exercise stopped
- Low systolic BP, multifocal ventricular ectopy or V-tach, ST changes, poor duration of exercise (<2 minutes) due to cardiopulmonary limitations
Cardiac markers

• 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, poly/dermatomyositis

• CK-MB isoenzyme
  – Rises 4-6 hours after injury and peaks at 24 hours
  – Remains elevated 36-48 hours
  – Positive if CK/MB > 5% of total CK and 2 times normal
  – Elevation can be predictive of mortality
  – False positives with exercise, trauma, muscle dz, DM, PE
Cardiac markers
Other Diagnostic Tests

• Radionuclide studies
• Myocardial perfusion scintigraphy
• Exercise radionuclide ventriculography
• Echocardiography
• Ambulatory ECG monitoring/holters monitor
• Coronary arteriography
Management Goals to reduce Anginal Symptoms

• Prevent complications – myocardial infarction, and to prolong life
• No smoking, lower weight, control hypertension and diabetes
• Patients with CAD – LDL cholesterol should achieve lower levels (<100)
• HMG-COA reductase inhibitors are effective
MANAGEMENT OF THE PATIENT WITH IHD

Initiate medical therapy
1. Decrease demand ischemia
2. Minimize IHD risk factors
3. ASA (clopidogrel if ASA intolerant)

Any high-risk features?
Low exercise capacity or ischemia at low workload
Large area of ischemic myocardium, EF < 40%, ACS presentation

No

Are exertional symptoms controlled?

Yes

Refer for coronary arteriography

No

Anatomy suitable for revascularization?

Yes

Single vessel disease
PCI

LM +/or multivessel disease
Assess PCI vs. CABG

No

Consider unconventional treatments

Continue medical therapy
Periodic stress assessment (see Fig. 237-1)
Pharmacologic Therapy

• Therapy is aimed in restoring balance between myocardial oxygen supply and demand

• Useful Agents: nitrates, beta-blockers and calcium channel blockers
Nitrates

- Reduce myocardial oxygen demand
- Relax vascular smooth muscle
- Reduces venous return to heart
- Arteriolar dilators decrease resistance against-which left ventricle contracts and reduces wall tension and oxygen demand
Nitrates: cont

- Dilate coronary arteries with augmentation of coronary blood flow
- Side effects: generalized warmth, transient throbbing headache, or lightheadedness, hypotension
- ER if no relief after nitrates: unstable angina or MI
Problems with Nitrates

- Drug tolerance
- Continued administration of drug will decrease effectiveness
- Prevented by allowing 8 – 10 hours nitrate free interval each day.
- Elderly/inactive patients: long acting nitrates for chronic antianginal therapy is recommended
- Physical active patients: additional drugs are required
Beta Blockers

- Prevent effort induced angina
- Decrease mortality after myocardial infarction
- Reduce Myocardial oxygen demand by slowing heart rate, force of ventricular contraction and decrease blood pressure
Contraindications:

- Symptomatic CHF, history of bronchospasm, bradycardia or AV block, peripheral vascular disease with s/s of claudication

Side effects:

- Bronchospasm (RAD), CHF, depression, sexual dysfunction, AV block, exacerbation of claudication, potential masking of hypoglycemia in IDDM patients
Calcium Channel Blockers

• Anti-anginal agents prevent angina
• Helpful: episodes of coronary vasospasm
• Decreases myocardial oxygen requirements and increase myocardial oxygen supply
• Potent arterial vasodilators: decrease systemic vascular resistance, blood pressure, left ventricular wall stress with decrease myocardial oxygen consumption
Calcium Channel Blockers

• Secondary agents in management of stable angina
• Are prescribed only after beta blockers and nitrate therapy has been considered
• Potential to adversely decrease left ventricular contractility
• Used cautiously in patients with left ventricular dysfunction
Amlodipine and Felodipine

• Are newer CCB
• Decrease (-) inotropic effects
• Amlodipine is tolerated in patients with advanced heart failure without causing increase mortality when added with ace inhibitor, diuretic, and digoxin
Drugs

• Verapamil and Cardizem is preferred because of effect on slowing heart rate
• Patients with resting bradycardia or AV block, a dihydropyridine calcium blocker is better choice
• Patients with CHF: nitrates preferred amlodipine should be added if additional therapy is needed
Drugs

• Primary coronary vasospasm: no treatment with beta blockers, it could increase coronary constriction
• Nitrates and CCB are preferred
• Concomitant hypertension: BB or CCB are useful in treatment
• Ischemic Heart Disease & Atrial Fibrillation: treatment with BB, verapamil or Cardizem can slow ventricular rate
Combination Therapy

• If patients do not respond to initial antianginal therapy – a drug dosage increase is recommended unless side effects occur.

• Combination therapy: successful use of lower dosages of each agent while minimizing individual drug side effects
Combinations

- Beta blockers should be combined only very cautiously with verapamil or cardizem because of potential of excessive bradycardia or CHF in patients with left ventricular dysfunction.
Other methods

• Patients with 1 – 2 vessel disease with normal left ventricular function are referred for catheter based procedures
• Patients with 2 and 3 vessel disease with widespread ischemia, left ventricular dysfunction or DM and those with lesions are not amendable to catherization based procedures and are referred for CABG
PCI vs CABG

PCI

Stent addresses the existing lesion but not future lesions.

CABG

Bypass grafting addresses the existing lesion and also future culprit lesions.
Pre & post PCI
Acute Management

• Initial evaluation & stabilization

• Efficient risk stratification

• Focused cardiac care
Evaluation

- Efficient & direct history
- Initiate stabilization interventions

Plan for moving rapidly to indicated cardiac care
Chest pain suggestive of ischemia

Immediate assessment within 10 Minutes

- 12 lead ECG
- Obtain initial cardiac enzymes
- electrolytes, cbc, lipids, bun/cr, glucose, coags
- CXR

- IV access
- Cardiac monitoring
- Oxygen
- Aspirin
- Nitrates

- Establish diagnosis
- Read ECG
- Identify complications
- Assess for reperfusion

Initial labs and tests
Emergent care
History & Physical
Focused History

• Aid in diagnosis and rule out other causes
  – Palliative/Provocative factors
  – Quality of discomfort
  – Radiation
  – Symptoms associated with discomfort
  – Cardiac risk factors
  – Past medical history - especially cardiac

• Reperfusion questions
  – Timing of presentation
  – ECG c/w STEMI
  – Contraindication to fibrinolysis
  – Degree of STEMI risk
Targeted Physical Examination

• 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
ECG assessment

- ST Elevation or new LBBB
  - STEMI

- ST Depression or dynamic T wave inversions
  - NSTEMI

- Non-specific ECG
  - Unstable Angina
Risk Stratification

Based on initial Evaluation, ECG, and Cardiac markers

STEMI Patient?

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

YES

UA or NSTEMI

- Evaluate for Invasive vs. conservative treatment
- Directed medical therapy

NO
Cardiac Care Goals

• Decrease amount of myocardial necrosis
• Preserve LV function
• Prevent major adverse cardiac events
• Treat life threatening complications
STE MI cardiac care

- **STEP 1**: Assessment
  - Time since onset of symptoms
    - 90 min for PCI / 12 hours for fibrinolysis
  - Is this high risk STEMI?
    - KILLIP classification
      - If higher risk may manage with more invasive rx
  - Determine if fibrinolysis candidate
    - Meets criteria with no contraindications
  - Determine if PCI candidate
    - Based on availability and time to balloon rx
Fibrinolysis indications

- ST segment elevation >1mm in two contiguous leads
- New LBBB
- Symptoms consistent with ischemia
- Symptom onset less than 12 hrs prior to presentation
Absolute contraindications for fibrinolysis therapy in patients with acute STEMI

• Any prior ICH
• Known structural cerebral vascular lesion (e.g., AVM)
• Known malignant intracranial neoplasm (primary or metastatic)
• Ischemic stroke within 3 months EXCEPT acute ischemic stroke within 3 hours
• Suspected aortic dissection
• Active bleeding or bleeding diathesis (excluding menses)
• Significant closed-head or facial trauma within 3 months
STEMI cardiac care

• **STEP 2:** Determine preferred reperfusion strategy

**Fibrinolysis** preferred if:
- <3 hours from onset
- PCI not available/delayed
  - door to balloon > 90min
  - door to balloon minus door to needle > 1hr
- Door to needle goal <30min
- No contraindications

**PCI** preferred if:
- PCI available
- Door to balloon < 90min
- Door to balloon minus door to needle < 1hr
- Fibrinolysis contraindications
- Late Presentation > 3 hr
- High risk STEMI
  - Killup 3 or higher
- STEMI dx in doubt
Medical Therapy
MONA + BAH

- **Morphine** (class I, level C)
  - Analgesia
  - Reduce pain/anxiety—decrease sympathetic tone, systemic vascular resistance and oxygen demand
  - Careful with hypotension, hypovolemia, respiratory depression

- **Oxygen** (2-4 liters/minute) (class I, level C)
  - Up to 70% of ACS patient demonstrate hypoxemia
  - May limit ischemic myocardial damage by increasing oxygen delivery/reduce ST elevation
• **Nitroglycerin** (class I, level B)
  - Analgesia—titrate infusion to keep patient pain free
  - Dilates coronary vessels—increase blood flow
  - Reduces systemic vascular resistance and preload
  - Careful with recent ED meds, hypotension, bradycardia, tachycardia, RV infarction

• **Aspirin** (160-325mg chewed & swallowed) (class I, level A)
  - Irreversible inhibition of platelet aggregation
  - Stabilize plaque and arrest thrombus
  - Reduce mortality in patients with STEMI
  - Careful with active PUD, hypersensitivity, bleeding disorders
• **Beta-Blockers** (class I, level A)
  • 14% reduction in mortality risk at 7 days at 23% long term mortality reduction in STEMI
  • Approximate 13% 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** (class I, level A)
  • 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
• **Heparin** (class I, level C to class IIa, level C)
  – LMWH or UFH (max 4000u bolus, 1000u/hr)
    • Indirect inhibitor of thrombin
    • less supporting evidence of benefit in era of reperfusion
    • Adjunct to surgical revascularization 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
    • Changing from one to the other not recommended
Additional medication therapy

• **Clopidodrel** (class I, level B)
  • Irreversible inhibition of platelet aggregation
  • Used in support of cath / PCI intervention or if unable to take aspirin
  • 3 to 12 month duration depending on scenario

• **Glycoprotein IIb/IIIa inhibitors**
  (class IIa, level B)
  • Inhibition of platelet aggregation at final common pathway
  • In support of PCI intervention as early as possible prior to PCI
STEMI care CCU

• Monitor for complications:
  • recurrent ischemia, cardiogenic shock, ICH, arrhythmias

• Review guidelines for specific management of complications & other specific clinical scenarios
  • PCI after fibrinolysis, emergent CABG, etc...

• Decision making for risk stratification at hospital discharge and/or need for CABG
Secondary Prevention

• Disease
  – HTN, DM, HLP

• Behavioral
  – smoking, diet, physical activity, weight

• Cognitive
  – Education, cardiac rehab program
Secondary Prevention
disease management

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

• 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%
Secondary prevention
behavioral intervention

• Smoking cessation
  – Cessation-class, meds, counseling

• Physical Activity
  – Goal 30 - 60 minutes daily
  – Risk assessment prior to initiation

• Diet
  – DASH(dietary approach to stop hypertension) diet, fiber, omega-3 fatty acids
  – <7% total calories from saturated fats
Evaluation of Chest pain
Thank You