Management of Ischemic Heart Disease (IHD)

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442 course

1

Introduction

- Coronary heart disease (CHD) is the most common form of heart disease

Most common cause of death

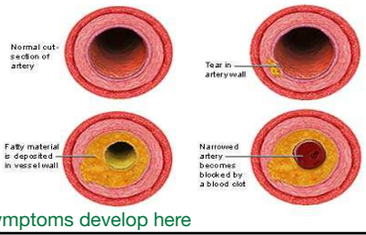
- An estimated 330 000 people have a myocardial infarct each year
- Approximately 1.3 million people have angina each year

2

Here in SA we are 10 years younger in terms of incidence of ischemic heart
In western society it's 65 years old
In SA society it's 55 years old
due to emergence of sedentary life style, smoking, DM.

Introduction

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



Symptoms develop here

3

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

4

Atheroma: low density lipoprotein engulfed with microcytes, lymphocytes
Deposited in the wall and forms whats known as fatty streaks
With progression its causes lumen narrowing

Defining myocardial infarction: ischemia means imbalance between o2 demand and blood supply

Whenever you have reduction pf blood flow to the heart and with the patient exercising, ischemia and chest pain will develop. This happens with every organ, ischemia of the legs > claudication, ischemia of the brain > stroke.

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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

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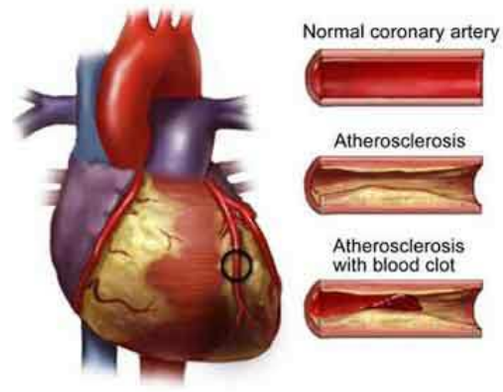

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Two main arteries
Left main coronary artery branches into two major arteries
Left anterior descending artery LAD (most important one, supplies. 70% of myocardium)
Left circumflex artery LCA

Right coronary artery will give further branches and go posteriorly then downward and gives:
Posterior descending artery PDA
Posterior left ventricular artery PLV

Ischaemic Heart Disease

- **Angina**
  - **Stable**
  - **Unstable**
  - **Prinzmetal's**

Comes at night, awakens from sleep and its due to vasospasm

- **Myocardial Infarction**
  - **NSTEMI**
  - **STEMI**

7

Clinic pathological correlation

Clinical Problem	Pathology
Stable angina	Ischaemia due to fixed atheromatous stenosis of one or more coronary arteries
Unstable angina	Ischaemia caused by dynamic obstruction of a coronary artery due to plaque rupture with superimposed thrombosis and spasm
Myocardial infarction	Myocardial necrosis caused by acute occlusion of a coronary artery due to plaque rupture and thrombosis
Heart failure	Myocardial dysfunction due to infarction or ischaemia
Arrhythmia	Altered conduction due to ischaemia or infarction
Sudden death	Ventricular arrhythmia, asystole or massive myocardial infarction

8

Angina: IMPORTANT
1) chest discomfort: heaviness, tightness and rarely described as pain and its diffuse (visceral) difficult to localized
2) comes with exertion and/or emotional distress (following hearing bad news, loss of loved ones, loss of money
3) relived by stress

If all three components present > **stable** angina
If only 2 > **atypical** angina
If only 1 > **myocardial** pain

Stable angina: When the patient gets pain at certain known level of exertion for 1 year e.g. someone lives in the 1st floor, he climbs the 1st floor normally but when he climb the 2nd floor he gets the pain

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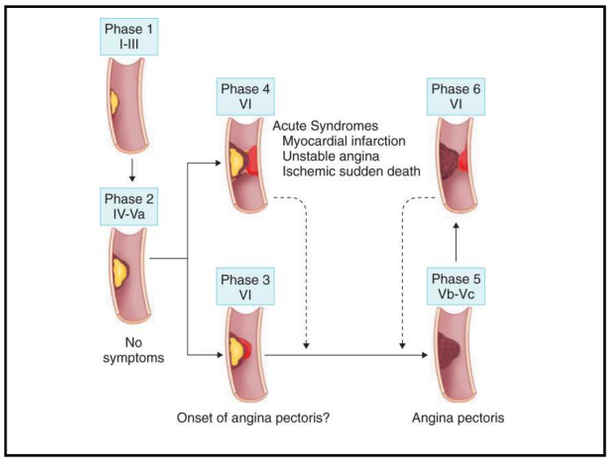
Unstable angina: When the patient has a new onset, symptoms at rest or progression in stable angina e.g. the patient starts feeling the pain with climbing the 1st floor



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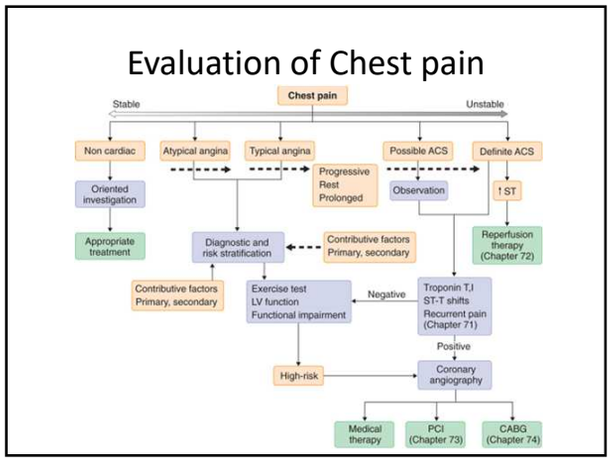


10

Note that 30% of adult visits to the ER is due to chest pain
While pediatrics it's due to cough and diarrhea

Acute Coronary Syndromes

	Stable Angina	Unstable Angina	STEMI	NSTEMI
Character of pain	Exertional pain	Rest pain	Rest pain	Rest pain
Relievers	Responds to GTN	No GTN effect	No GTN effect	No GTN effect
Enzymes	Normal	Normal	Elevated	Elevated
ECG	Often normal	Often ST depression	ST segment elevation	No ST segment elevation

11 This isn't important

Angina

- When ischemia results it is frequently accompanied by chest discomfort: Angina Pectoris
- In the majority of patients with angina, development of myocardial ischemia results from a combination of fixed and vasospastic stenosis

12

Chronic Stable Angina

- May develop sudden increase in frequency and duration of ischemic episodes occurring at lower workloads than previously or even at rest
- Known as unstable angina: up to 70% patients sustain MI over the ensuing 3 months

13

Angina: cont

- Patients with mild obstruction coronary lesions can also experience unstable angina
- >90% of Acute MI result from an acute thrombus obstructing a coronary artery with resultant prolonged ischemia and tissue necrosis

14

Treatment of Angina

- Treatment of Chronic Angina is directed at minimizing myocardial oxygen demand and increasing coronary flow
- Where as in the acute syndromes of unstable angina or MI primary therapy is also directed against platelet aggregation and thrombosis

15

Epidemiology

- Modifiable Factors: hyperlipidemia- ^ LDL (<130 normal) or low HDL (>60 normal), Hypertension, cigarette smoking and diabetes, obesity, BMI of >30
- Non-Modifiable Factors: advanced age, male sex, family medical history: male <55 y/o, female <65 y/o
- Other: sedentary lifestyle and stressful emotional stress

Very important factor we should not neglect

16

Quality

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

17

Signs & Symptoms accompany Angina

- Dyspnea, nausea, diaphoresis resolve quickly after cessation of angina
- Angina is a diffuse sensation rather than discrete

18

Ischemic Heart Disease

- Imbalance between Myocardial oxygen supply and demand = Myocardial hypoxia and accumulation of waste metabolites due to atherosclerotic disease of coronary arteries

19

Stable Angina

- Stable Angina: chronic pattern of transient angina pectoris precipitated by physical activity or emotional upset, relieved by rest with in few minutes
- Temporary depression of ST segment with no permanent myocardial damage

20

Angina Pectoris

- Angina Pectoris: uncomfortable sensation in the chest or neighboring anatomic structures produced by myocardial ischemia

21

Variant Angina

- Typical anginal discomfort usually at rest
- Develops due to coronary artery spasm rather than increase myocardial oxygen demand
- Transient shifts of ST segment – ST elevation

22

Unstable Angina

- Increased frequency and duration of Angina episodes, produced by less exertion or at rest = high frequency of myocardial infarction if not treated

23

Silent Ischemia

- Asymptomatic episodes of myocardial ischemia
- Detected by electrocardiogram and laboratory studies

These are asymptomatic repeated MI frequently with patients with long standing DM due to autonomic neuropathy

24

Myocardial Infarction

- Region of myocardial necrosis due to prolonged cessation of blood supply

And chronic inflammatory process

- Results from acute thrombus at side of coronary atherosclerotic stenosis
- May be first clinical manifestation of ischemic heart disease or history of Angina Pectoris

25

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
- Myocardial Ischemia displays a circadian rhythm threshold for Angina it is lower in morning hours.

26

Physical Examination

Arcus senilis Seen in the eyes

- Arcus senilis, xanthomas, funduscopic exam: AV nicking, exudates Result from HTN or DM
- Signs and symptoms: hyperthyroidism with increased myocardial oxygen demand, hypertension, palpitations
- Auscultate carotid and peripheral arteries and abdomen: aortic aneurysm
- Cardiac: S4 common in CAD, increased heart rate, increased blood pressure

27

Ischemia

- Myocardial ischemia may result in papillary muscle 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

28

50% of patients have carotid artery disease
90% of patients who have peripheral disease have heart disease not yet manifested

If you're asked in an exam about investigations always categorize into
Basic routine: CBC, LFT, renal profile, electrolytes, complete metabolic panel
Specific: cardiac markers (bnp, troponin, ck-mb), ecg,echo
Radiological:
Pathological: biopsy in very limited conditions e.g. heart transplant
Note that *bnp* indicated high loading of the heart > heart failure

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

29

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 spasm which is less often

ST segment elevation: STEMI
ST segment depression: NSTEMI or unstable angina

30

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

31

Other Diagnostic Tests

- Radionuclide studies
- Myocardial perfusion scintigraphy
- Exercise radionuclide ventriculography
- Echocardiography
- Ambulatory ECG monitoring
- Coronary arteriography Diagnostic and therapeutic

32

A patient comes to the clinic and we exercise / stress the patient to induce ischemia, but its important to check patient's fitness and ability to perform exercise. We observe for St segment changes, if it changes we label the test **+ve**
If we want to stress an old patient we give Dobutamine (physiological stress)

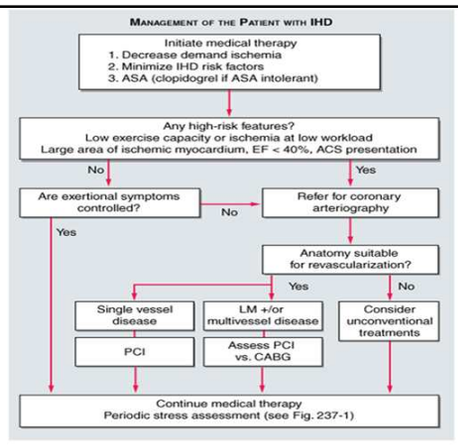
Management goal (very imp question in every case, It doesn't mean treatment only, so:
1) say you will treat as an outpatient or inpatient? In this case admit the patient
2) we start medical therapy. Primary prevention medications
3) thorough clinical investigations like echo, ecg, blood work, then start medications accordingly

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

Now its changed to people at risk <70
Had MI already <55

33



34

Pharmacologic Therapy

- Therapy is aimed in restoring balance between myocardial oxygen supply and demand
- Useful Agents: nitrates, beta-blockers and calcium channel blockers

35

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

By activation of cyclic GMP

36

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 X2 nitro's: unstable angina or MI

37

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

38

Patient will become dependent, everytime you need to increase the dose
It causes headaches

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

39

They act on B1 receptor and therefore decrease contractility and slow down heart rate, basically relax the heart
Induce coronary vasodilation > improve coronary blood flow

Beta -1

- Block myocardial receptors with less effect on bronchial and vascular smooth muscle- patients with asthma, intermittent claudication

40

Beta-Agonist blockers

- With partial B-agonist activity:
- Intrinsic sympathomimetic activity (ISA) have mild direct stimulation of the beta receptor while blocking receptor against circulating catecholamines
- Agents with ISA are less desirable in patients with angina because higher heart rates during their use may exacerbate angina
- not reduce mortality after AMI

41

Beta-blockers

- Duration of beta-blockers depends on lipid solubility
- Accounts for different dosage schedules

42

Contraindications

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

43

Side Effects

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

44

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

45

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

46

2nd or 3rd line not recommended to every patient
Negative ionotropic effect, thus decreases contractility
Vasodilation effect
If patient is not controlled with BB give CCB still not controlled? Nitrates

Drug Selection

- Chronic Stable Angina: beta blocker and long acting nitrate or calcium channel blocker (not verapamil: bradycardia) or both.
- If contraindication to BB a CCB is recommended (bronchospasm, IDDM, or claudication) any of CCB approved for angina are appropriate.

47

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

48

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

49

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

50

Combination Therapy Include:

- Nitrate and beta blocker
- Nitrate and verapamil or cardizem for similar reasons
- Long acting dihydropyridine calcium channel blocker and beta blocker
- A dihydropyridine and nitrate is often not tolerated without concomitant beta blockade because of marked vasodilatation with resultant head ache and increased heart rate

51

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

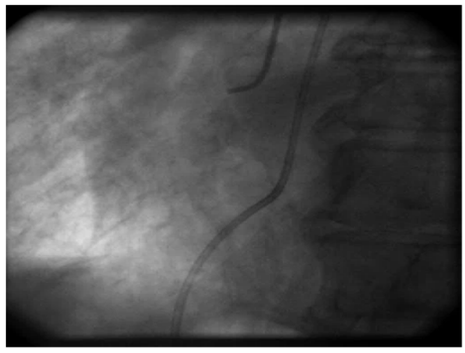
52

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

53

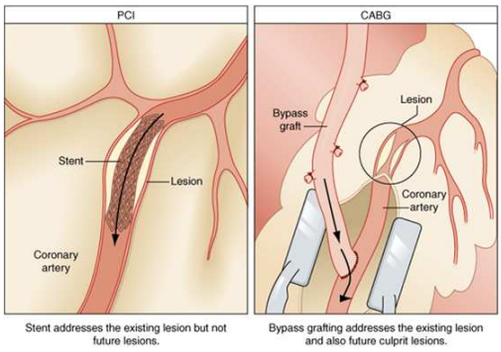
Cardiac Cath



54

PCI: primary coronary intervention

PCI vs CABG



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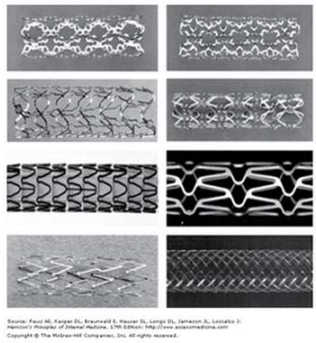
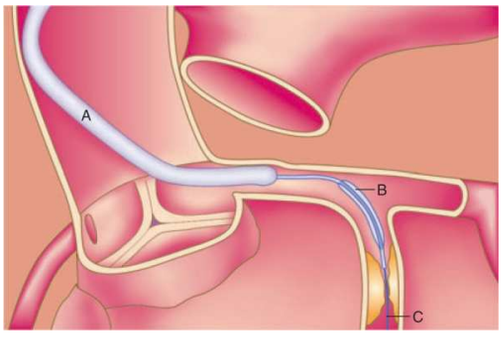
Metal stents



56

PCI



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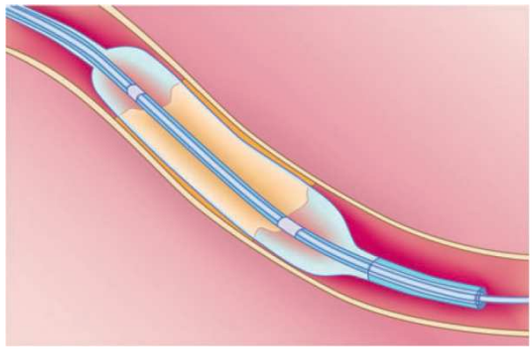
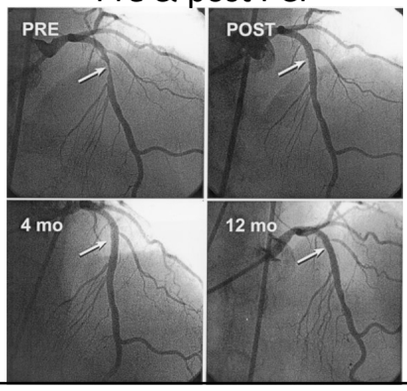
PCI



58

Focal stenosis

Pre & post PCI



59 They put a stent you see lumen widely open and flow very nice

Acute Coronary Syndromes

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

Similar pathophysiology

Similar presentation and early management rules

STEMI requires evaluation for acute reperfusion intervention

60

Diagnosis is simply by cardiac markers and ECG

ST segment elevation > STEMI > immediately cath lab and PCI
(Time is muscle) less than <90 minutes

ST depression > chest pain > unstable angina
+ve cardiac markers > NSTEMI
-ve cardiac markers > unstable angina
Both treated the same but not necessarily within 90 min but within 24 hrs of admition

Diagnosis of Acute MI
STEMI / NSTEMI

- At least 2 of the following
  - Ischemic symptoms
  - Diagnostic ECG changes
  - Serum cardiac marker elevations

61

Diagnosis of Angina

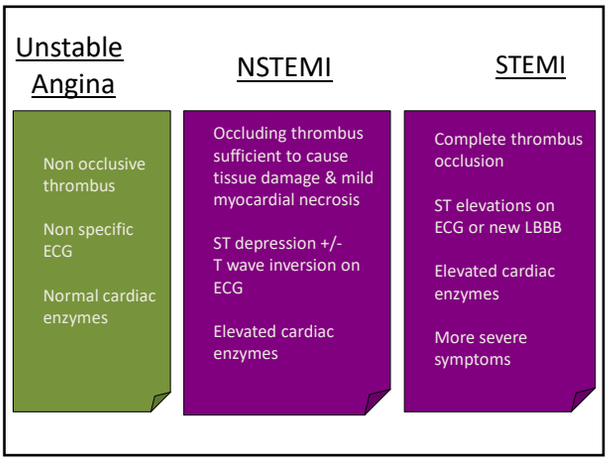
- Typical angina—All three of the following
  - Substernal chest discomfort
  - Onset with exertion or emotional stress
  - Relief with rest or nitroglycerin
- Atypical angina
  - 2 of the above criteria
- Noncardiac chest pain
  - 1 of the above

62

Diagnosis of Unstable Angina

- Patients with typical angina - An episode of 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 or at rest within the previous two weeks
  - Prolonged pain at rest

63



64

Stabilize, check vitals, if o2 < 90% give o2
If BP is elevated lower it
If BP is low give ionotropic support
Start loafing with a 300g dose of aspirin
Anticoagulants IV heparin
Dual antiplatlets
BB and statins as early as possible

And treat accordingly
STEMI > cath lab
NSTEMI > icu, stabilize and next day send to cath lab

Very important any Patient with MI
Aspirin
Statin
BB

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Acute Management

- Initial evaluation & stabilization
- Efficient risk stratification
- Focused cardiac care

65

Evaluation

- Efficient & direct history
- Initiate stabilization interventions

Occurs simultaneously

Plan for moving rapidly to indicated cardiac care

66

Chest pain suggestive of ischemia

Immediate assessment within 10 Minutes

Initial labs and tests	Emergent care	History & Physical
– 12 lead ECG	■ IV access	■ Establish diagnosis
– Obtain initial cardiac enzymes	■ Cardiac monitoring	■ Read ECG
– electrolytes, cbc lipids, bun/cr, glucose, coags	■ Oxygen	■ Identify complications
– CXR	■ Aspirin	■ Assess for reperfusion
	■ Nitrates	

67

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

68

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

69

ECG assessment

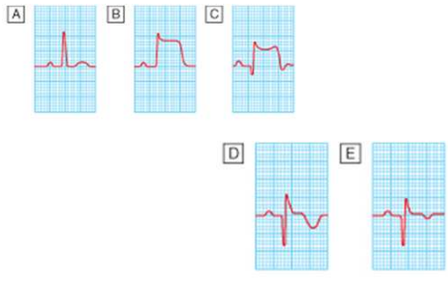
ST Elevation or new LBBB
STEMI

ST Depression or dynamic T wave inversions
NSTEMI

Non-specific ECG
Unstable Angina

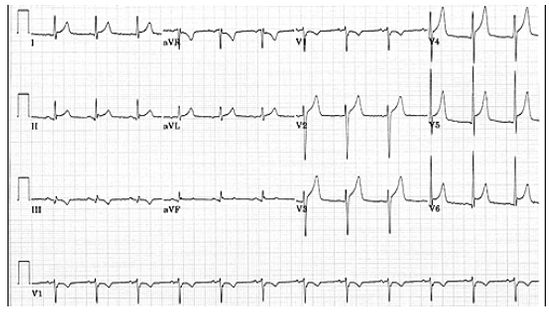
70

ECG changes with MI



71

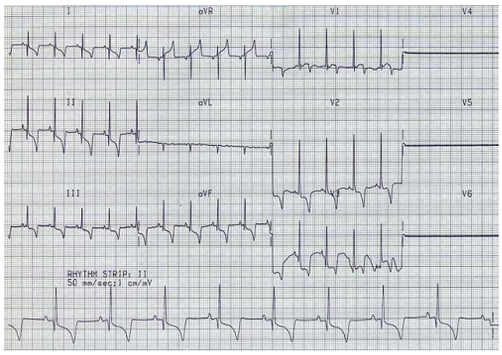
Normal or non-diagnostic ECG



72

Diffuse ST segment elevation due to early repolarization, you see here a smily face
Unlike MI sad face

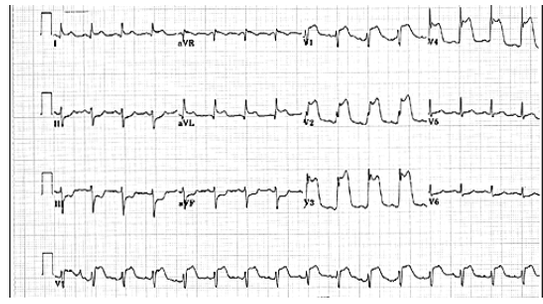
ST Depression or Dynamic T wave Inversions



73

Unstable angina

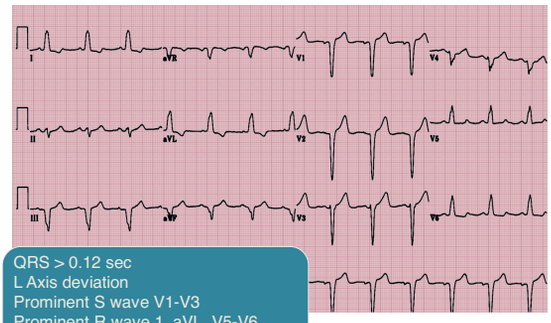
ST-Segment Elevation MI



74

Its localized in ST elevation you can localize but in ST depression you can not
So here its acute anterolateral MI
LAD occlusion

New LBBB



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

75

Cardiac markers

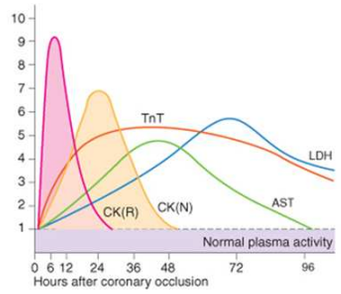
- Troponin (T, I)
  - Very specific and more sensitive than CK
  - Rises 4-8 hours after injury
  - May remain elevated for up to two weeks
  - Can provide prognostic information
  - Troponin T may be elevated with renal dz, poly/dermatomyositis
- CK-MB isoenzyme
  - Rises 4-6 hours after injury and 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

76

If a patient presented with chest pain and the cardiac markers were negative don't discharge and repeat after 6 hours

Peak of troponin 12 hrs and stays for 2 weeks
Peak for CK-mb 34 and stays for 3 to 5 days

Cardiac markers



77

Risk Stratification

Based on initial Evaluation, ECG, and Cardiac markers

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

78

Cardiac Care Goals

- Decrease amount of myocardial necrosis
- Preserve LV function
- Prevent major adverse cardiac events
- Treat life threatening complications

79

STEMI 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

80

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

81

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) Risk of bleeding
- 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

82

If you're in a hospital with no cath lab, transfer your patient. If not possible give fibrinolysis it helps in degradation of platelet plug given in the 1st hour of chest pain
And within less than 12 hours
After that its not recommended

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

83

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

84

- **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

85

- **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

86

For diabetics or EF <40

- **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

87

Indicated for all patient with ACS
Only for acute management until discharge or after 8 weeks, whatever comes first

Combine it with aspirin for the first year then continue on aspirin alone

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

88

Additional medication therapy

- **Aldosterone blockers** (class I, level A)
  - Post-STEMI patients
    - no significant renal failure (cr < 2.5 men or 2.0 for women)
    - No hyperkalemis > 5.0
    - LVEF < 40%
    - Symptomatic CHF or DM

89

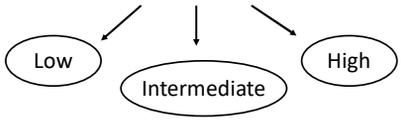
STEMI care CCU

- 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

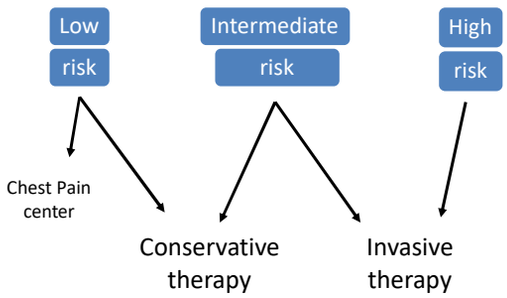
90

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



91



92

Invasive therapy option UA/NSTEMI

- Coronary angiography and revascularization within 12 to 48 hours after presentation to ED
- For high risk ACS (class I, level A)
- 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

93

Conservative Therapy for UA/NSTEMI

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

94

Secondary Prevention

- Disease
  - HTN, DM, HLP
- Behavioral
  - smoking, diet, physical activity, weight
- Cognitive
  - Education, cardiac rehab program

95

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%

96

Secondary prevention
behavioral intervention

- 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

97

Thank You

98