

OBJECTIVES

- Anatomy Review
- Risk Factors
- Discuss causes of impaired blood supply
- UA/NSTEMI /STEMI
- Identify clinical signs and symptoms of MI
- ECG changes associated with acute MI
- Medications
- Core Measures/ Optimizing Outcomes

STATISTICS

- Acute Myocardial Infarction leading cause of death in the US
 - Tremendous economic burden 71.2 billion spent on inpatient hospital costs (1/4 of total hospital costs)
 - 1.5 million Americans experience an AMI yearly
- of those: 38% mortality

CORONARY ARTERY DISEASE

- ★ Significant narrowing of the coronary arteries that prevents adequate blood supply to the myocardium.
- ★ A variable process that will be different with each person and depending on the vessel involved

COMMON CAUSES

- Atherosclerosis: the underlying condition of CAD progressive with periods of stable and unstable disease
-
- Coronary Artery Thrombosis
- Coronary Artery Spasm

ETIOLOGIES “RISK FACTORS”

- ❖ Heredity
- ❖ Smoking
- ❖ High Cholesterol (LDL)
- ❖ HTN
- ❖ Diabetes
- ❖ Obesity



Cholesterol : lipid

- ★ LDL: Bad Cholesterol
 - ★ >160 high < 100 is ideal
 - ★ DM <70 (based on risk factors)
- ★ HDL: Good Cholesterol
 - ★ <40 is low
- ★ Total Cholesterol
 - ★ < 200 desirable
 - ★ > 240 high
- ★ Triglycerides- important prognostic indicator in the diabetic

Smoking : Nicotine

- **Most important modifiable risk**
 - ◆ Increases blood pressure
 - ◆ Decreased exercise tolerance
 - ◆ Increases the tendency for blood clots
 - ◆ Women who use oral contraceptives and smoke have significant increase risk
 - ◆ Increase LDL levels/decrease HDL therefore an increased risk for fatty buildup



Why Quit?

- among cardiac patients, quitting smoking lowers the risk of :
 - subsequent CABG by 300%
 - subsequent nonfatal MI by 32%

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A Teachable Moment

- ◆ Ask: ask every one, and document
- ◆ Advise: in a clear strong and personalized manner urge every tobacco user to quit
- ◆ Assess: is the patient willing to make a quit attempt at this time?
- ◆ Assist: Oklahoma Tobacco Helpline
- 1-800-QUIT NOW
- ◆ Arrange: schedule follow up preferably within the first week after quit date

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Obesity: #6 in the Nation

- More than 20% over idea body weight
- BMI >30
- abdominal wall circumference:
 - >35 women, >40 men
- Increases blood pressure
- Increases LDL/ decreases HDL
- Decreases exercise
- Greater risk for heart failure
- Glucose intolerance/ Insulin Resistance

Hypertension

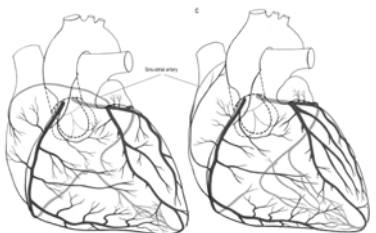
- Elevation of arterial blood pressure >140/90
- Can be either primary (99%) or secondary HTN (1%)
- Increases risk for stroke

Diabetes

- 75% of people with diabetes die of some form of heart or blood vessel disease
- Increase risk for atherosclerosis
- Increase risk for HTN
- Autonomic Neuropathy “Silent Ischemia”

CARDIAC ANATOMY

- supply blood to the electrical conduction system and muscle.



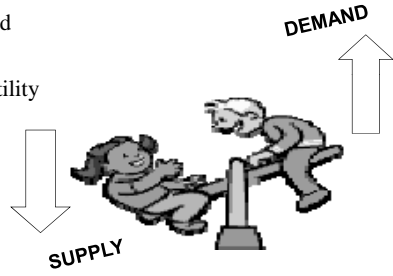
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O2 Supply:

- Coronary Artery Anatomy
- Diastolic Pressure
- Diastolic filling time
- amount of O2 available
 - Hgb
 - SaO2

DEMAND

- Heart Rate
- Afterload
- Preload
- Contractility



SUPPLY AND DEMAND

| Oxygen Supply Affect Supply | Oxygen Demand Affect Demand |
|--------------------------------|-----------------------------------|
| Oxygen ↑ | Nitrates ↓ |
| Nitrates ↑ | Beta-Blockers ↓ |
| Fibrin Clot ↓ | Calcium Channel Blockers ↓ |
| Platelet Aggregation ↓ | Ace-Inhibitors ↓ |
| | Stress & Pain ↑ |

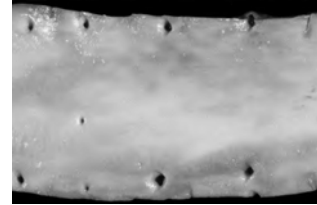
ATHEROSCLEROSIS

- Gradual process
- Cholesterol plaques deposited into the walls
- First seen as a fatty streak on the vessel wall
- Narrowing decreases flow: after it reaches 75%
- Damage to the innermost layer of the artery. Low-density lipoproteins (fatty/cholesterol) in the blood penetrate into the arterial wall, leading to accumulation of lipids in the smooth muscle of the artery.
- Attracts other lipoproteins: Foam cells are the earliest of the arterial streak

ATHEROSCLEROSIS Cont...

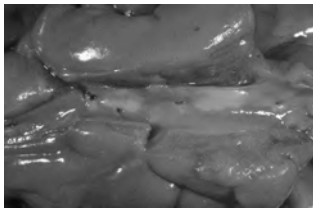
- At the advanced stage, the lesion will be covered in a dense matrix of connective tissue.
- An inflammatory component exist in the endothelial area, which predisposes the plaque to rupture
- Repeated injury and repair of the plaques leads to a fibrous cap protecting the underlying core and components
- Is not stable because it does not have the normal collagen supporting the structure

Fatty Streaks



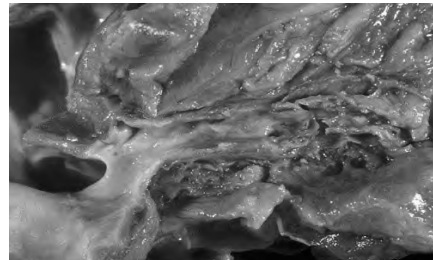
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Atherosclerosis “MILD”



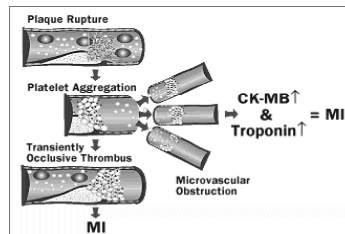
(Adapted from Klatt, 2002)

Atherosclerosis “Severe”



(Adapted from Klatt, 2002)

Unstable Plaque



As the myocardial cell dies, it releases structural molecules, CK-MB and Troponin.

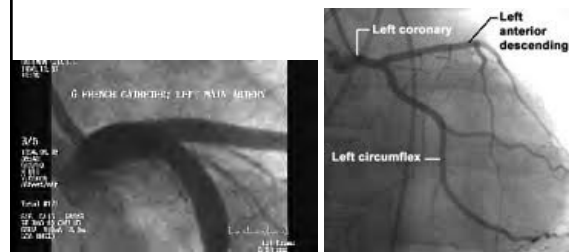
Plaque Rupture

- Exposes the inner lining
- Collagen causes the platelet to change shape
- Signaling agents are released (Adenosine diphosphate, thromboxane A₂, Serotonin, and Epinephrine) Platelet Agonist
- Vasoconstriction further reduces flow
- Platelets release GP IIb-IIIa receptors and form pseudopods
- They bind with fibrinogen and clump together
- Thrombin is produced and converts fibrinogen to fibrin
- A mesh traps RBC, Macrophages, contents of plasma and forms a partial or complete thrombus in the vessel

Platelets

- Platelet activation occurs following injury to the vessel wall
- First a layer of platelets adhere to the vessel wall, then through cross-linking with fibrinogen forms a platelet plug
- drug therapies are aimed at preventing platelets from doing their normal function

Normal Coronary anatomy

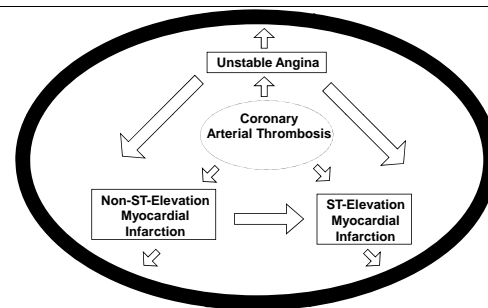


(Adapted from Klatt, 2002)

ACS

- Spectrum of events in which blood flow is suddenly and severely reduced or completely interrupted:
 - A supply and demand mismatch
- ST-segment elevation MI
- Non-ST elevation MI
- Unstable Angina (UA)
- Sudden Death : due to one of the above

Acute Coronary Syndromes (ACS)



Van de Werf F. *Throm Haemost.* 1997; 78(1):210-213.

Angina: SUPPLY

- A flow-demand imbalance between reduced blood flow through narrow coronary arteries and the demand placed on the heart.
- chronic, acute, or unstable.

Unstable Angina:

- Symptoms of angina that are new or increasing: rest or no exertion, usually lasts more than 20 minutes
- Usually due to platelet aggregation in narrowed coronary arteries with chronic atherosclerotic occlusion.
- A frequent cause of morbidity: 1-year mortality of 12% most of this occurring in the first months post discharge

VARIANT ANGINA PRINZMETAL'S ANGINA

- ◆ AKA Coronary Spasm
- ◆ A less common form of angina that is characteristic for chest pain at rest. It is generally not prolonged
- ◆ may cause transient ST segment elevation
- ◆ Troponin may elevate
- ◆ EKG changes may be present

UA/NSTEMI and STEMI

- Therapeutic approaches vary:
 - Treatment the same: RETORE BLOOD FLOW
 - Pathophysiology: similar but not the the same extent
 - each process can advance

Signs and Symptoms of Heart Attack

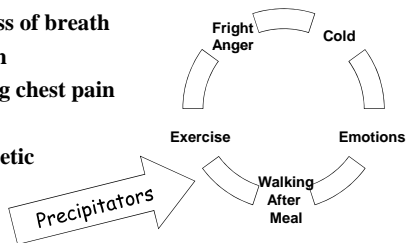
Typical symptoms for both sexes:

- Pain, pressure, a squeezing sensation, or stabbing
- Pain radiating to neck, shoulder, back, arm, or jaw
- Pounding heart or change in heart rhythm
- Difficulty breathing
- Nausea, vomiting, abdominal pain
- Cold sweats or clammy skin
- Dizziness
- Heartburn

MAY HAVE NO PAIN

Impending DOOM

- Burning or feeling of tightness
- Shortness of breath
- Jaw pain
- Crushing chest pain
- Pale
- Diaphoretic
- N/V



Why do people delay?

- Believed it would resolve
- Not the pain they would expect (fullness, heaviness) OR NO PAIN
- Didn't think symptoms were serious
- Decided on "wait and see" approach
- Thought symptoms were related to another condition (muscle strain, heartburn)
- Not aware of the benefit of rapid action
-

Common Physical Presentation

- Alert, anxious, restless, fatigued
- Cool, clammy, diaphoretic
- JVD, extra heart sounds S3 & S4
- Dyspnea, tachypnea, rales
- Nausea & vomiting with chest pain
- Thready or pounding peripheral pulses, irregular pulses

Gender Matters:

- Leading cause of death among women, and one of the most preventable
- Of those who survive an MI, 46% will be disabled by heart failure in six years
- Kills six times as many women every year as breast cancer

Women

- More likely to die within the first year
- Smaller coronary arteries
- more multi vessel disease when they present
- Metabolic syndrome: increased risk of CVD
 - A group of health risks
 - Large waist size
 - Elevated BP
 - Glucose intolerance
 - Low HDL
 - High triglycerides

Symptoms in women

- Shortness of breath (58%)
- Weakness (55%)
- Unusual fatigue (43%)
- Cold sweat (39%)
- Dizziness (39%)
- Nausea (36%)
- Arms weak/heavy (35%)

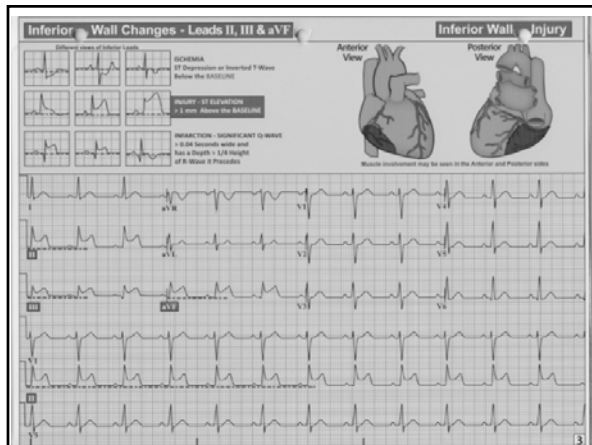
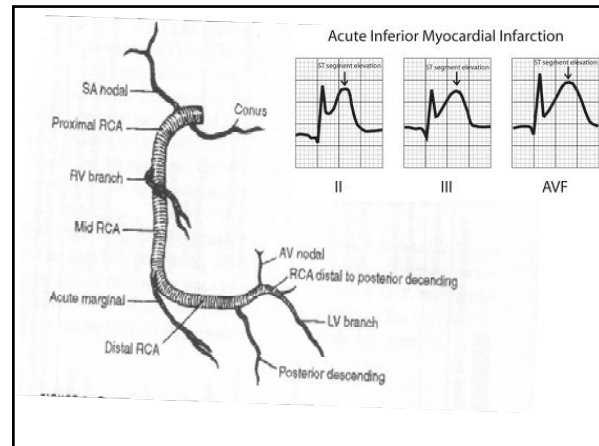
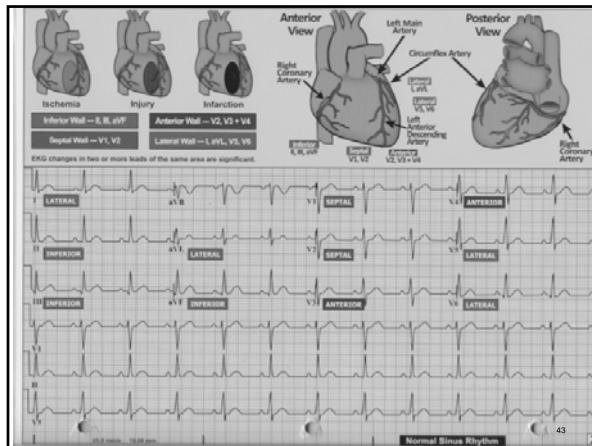
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TROPONIN

- elevates in 3-6 hours remains elevated 14 days
- best cardiac marker for ACS
- It is less useful to "RULE IN" this event because it may lack specificity for ACS, it isn't absolute
 - Sepsis
 - Hypovolemia
 - atrial fib
 - CHF
 - myocarditis
 - Pulmonary embolism
 - Renal failure- excreted by kidneys
 - Myocardial contusion

STEMI

- The most dangerous of the 3 clinical scenarios
- Often associated with sudden death
- Accounts of 1/3 of the ACS cases
- Caused by the sudden rupture of a cholesterol-filled plaque in the coronary artery wall
- Cascade of events that culminates in the formation of a thrombus over the plaque



RIGHT CORONARY ARTERY

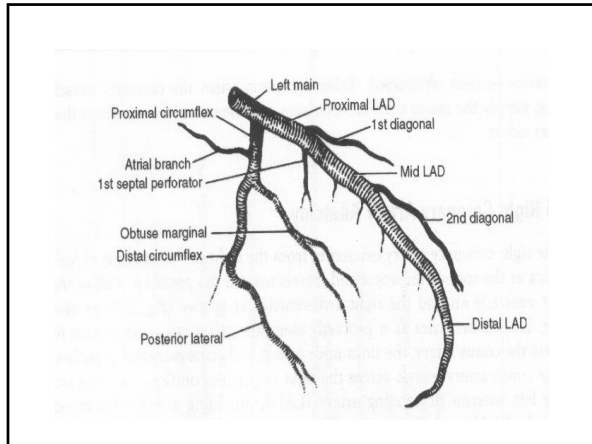
- Inferior AMI
- II, III, aVF
- dependent on RV filling pressure to maintain CO and BP: watch vasodilators and diuretics
- Starting Nitro, decreased BP and increased CP, consider RV infarct, can Dx with V4R

Inferior MI SIGNS AND SYMPTOMS

- Hypotension with clear lung fields
- Sinus Arrest or Brady
- SA and AV nodes often arise from the RCA
- Conduction disturbances are more common
 - more life threatening ventricular arrhythmias
 - AV blocks
 - IVCD
- As a general rule, Inferior MI often less dramatic and better tolerated than an AMI

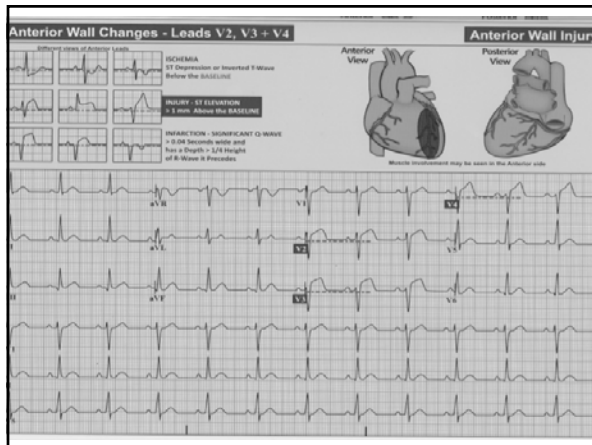
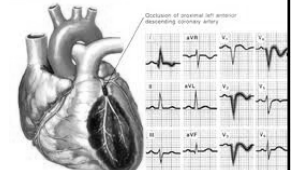
RV involvement

- 20-50% of acute inferiors have RV involvement
- Independent predictor of major complications and in hospital mortality
- Clinical DX: in the setting of inferior STE
 - elevated neck veins
 - clear lung fields
 - hypotension



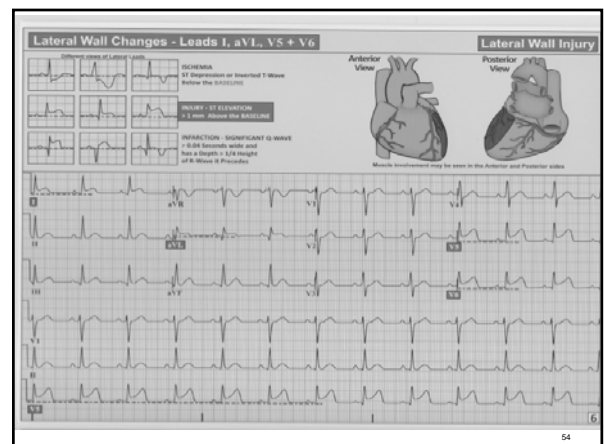
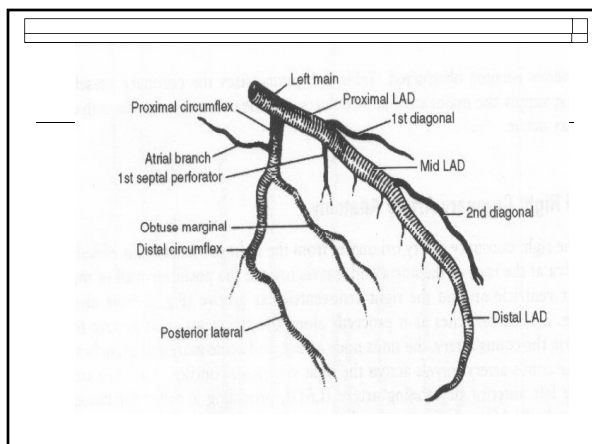
LEFT ANTERIOR DESCENDING ARTERY

- ❖ Supplies the anteroseptal wall
- ❖ Changes in V1-V6 possible, depending on size of infarct
- ❖ conduction involved **Anterior MI**



LAD INVOLVEMENT SIGNS AND SYMPTOMS

- ❑ Referred to as the widow maker
- ❑ presentation is related to low cardiac output
 - contractility decreased
 - bp reduced
 - heart rate usually >100 bpm
 - this area involves the conduction system:
 - ❑ high degree heart block
 - ❑ later BBB
- ❑ Ventricular Aneurysm
- ❑ Cardiogenic Shock
- ❑ Left Ventricular Failure and associated S&S



Left Circumflex Artery

- Lateral wall of left ventricle
- Frequently has no EKG changes
- AV Nodal Blocks

- most at risk for papillary muscle rupture

LCA INVOLVEMENT SIGNS AND SYMPTOMS

- Heart Failure: papillary muscle dysfunction
- Dysrhythmias: typically blocks

Goal: to provide relief of ischemia

- Analgesia: does not stop ischemia
 - Nito (supply and demand)

 - Morphine
 - Pros: reduces anxiety, lower HR lowers SBP
 - Cons: hypotension, nausea vomiting, decrease ventilations

Anti-ischemia Therapy: DEMAND

- Beta Blocker:
- ACE inhibitor
- Ca Channel Blocker
- Nitrates

Goal: to provide relief of ischemia

- Antiplatelet therapy SUPPLY
 - Aspirin
 - Clopidogrel
 - Glycoprotein IIb/IIIa inhibitor

 - Nitrates also affect the supply

Goal: to provide relief of ischemia

- Anticoagulant Therapy: SUPPLY
 - UFH
 - LMWH
 - Direct Thrombin inhibitor (Bivalrudin)
 - Factor Xa inhibitor (fondaparinux)

Beta Blockers “olols”

- Reduces the myocardial oxygen demand and potentially lethal arrhythmias
- Competitively block the effects of catecholamine's (epi and nor epi) on cell membrane beta receptors
- Slows HR and decreases contractility

Beta Blockers:

metoprolol, propranolol, atenolol

- Slows heart rate which helps enhance coronary blood flow
- Should be given ASAP (within 24) of presentation
- Contraindications: worsening heart failure, low-output state
- Significantly lower in-hospital mortality

Calcium channel blockers: supply and demand

- not the current treatment for ACS, “may consider”
-
- ◆ inhibit contraction of myocardial and smooth muscle and cause vasodilatation
- ◆ Decreases myocardial O₂ demand by decreasing SVR and contractility
- ◆ May be added to other medications for symptom relief
- Nifedipine, amlodipine, verapamil, diltiazem

Calcium channel blockers

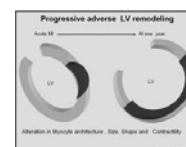
- Should be avoided in patients with significant LV dysfunction *(EF <40%) and pulmonary edema
- Major SE: hypotension, worsening CHF, brady and AV blocks

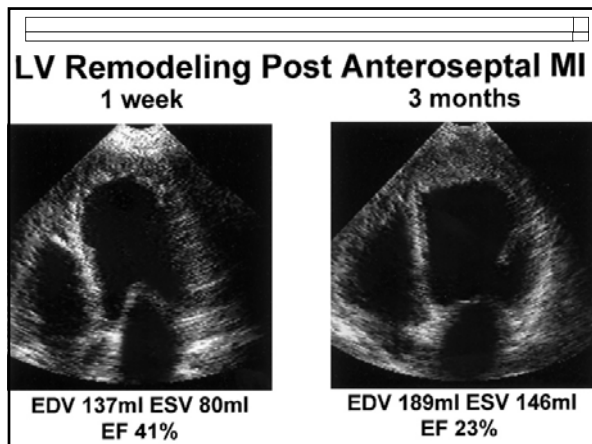
ACE inhibitors / ARB

- Are used to inhibit ventricular remodeling in the damaged myocardium and prevent the development of heart failure

Prognosis of LV FUNCTION

- Ventricular remodeling begins after cell death:
 - Cascade begins
- LV dilatation, and formation of collagen scar
- May continue for weeks until the tensile strength counterbalances the distending forces





ACE Inhibitors : pril's Angiotensin-Converting Enzyme

- Given orally within the first 24 hours for patients who have pulmonary congestion or a EF of less than 40% associated with MI
- Not if the pt is hypotensive
- Significant reductions in adverse outcomes, including mortality and hospitalizations
- Angiotensin-receptor blocker (ARB) can be given to patients who cannot tolerate an ACE

How does an ACE work?

- Angiotensin I – ACE- Angiotensin II
- ACE inhibitors slow the activity of the enzyme which decreases the production of angiotensin II
- Therefore blood vessels dilate: which makes it easier for the heart to pump blood

Side effects of ACE

- Cough
 - Elevated K
 - Hypotension
 - Weakness
- May take up to a month for the coughing to subside

ARB's (sartan's)

- Angiotensin II receptor blocker
- Blocks the action of angiotensin II on the small arteries
- Therefore angiotensin can't cause them to constrict
- Widening the arteries, lowers the pressure
- Can be given to pts who cannot use ACE

Common ARB's

- Valsartan (Diovan)
 - Losartan (Cozaar)
 - Olmesartan (Benicar)
- Common side effects:
- elevated K, hypotension

Antiplatelet Therapy:

Reduces platelet formation and aggregation : these are integral components in the formation of a thrombus after plaque disruption

Aspirin: ASAP after patient arrives in the ER

Clopidogrel: Also used for patient's who cannot tolerate ASA

Continued indefinitely in patients who tolerate it,

ASA: antiplatelet therapy

- Pts with suspected STEMI , enteric coated aspirin 160 mg reduced the 5 week cardiovascular mortality rate by 23% compared with placebo
- Non enteric coated recommended for acute events to allow rapid buccal absorption/chew
- Irreversibly inhibits platelet aggregation by inhibiting an enzyme therefore reduces the production of one of several potent platelet activators

Clopidogrel

- Inhibits platelet activation
- Mechanism of action different than ASA, binds to platelet receptors
- Effects are not manifested for several days when a loading dose is not given
- Optimal loading dose has not been firmly established (300 – 600 mg)
- Timing and duration of treatment after PCI not yet determined
- Still only given to slightly more than 1/2

Plavix Clopidogrel

- Usually withheld for 6-7 days prior to CABG
- Expensive
- 12 % of the population does not metabolize
- P2Y12
- Efficient

Dual antiplatelet therapy

- Aspirin and clopidogrel is beneficial in patients with STEMI
- Initial trials show this combination should be used in patients with STEMI regardless of whether thrombolytic agents are used
- AHA recommendations support a 12 month course of dual therapy: stent

G IIb/IIIa Inhibitors

- Potent inhibitors of platelet aggregation
 - Abciximab (Reopro)
 - Eptifibatid (Integrilin)
 - Tirofiban (Aggrastat)
- Greatest benefit is for those high risk patients who are candidates for early invasive strategy
- Three large studies 10%-30% relative risk reduction in adverse events compared to placebo plus standard therapy

GIIb/IIIa: risks

- Increase the risk of bleeding, particularly at the site of intervention for all pts
- Frequent groin assessment
- Hemoglobin levels and platelet counts should be monitored

Especially with Reopro: highest incident of thrombocytopenia

Anticoagulant Therapy

- ACC/AHA recommends more aggressive antiplatelet/anticoagulant therapy
- No particular agent is recommended
- Heparin: prevents the formation of thrombi, but does not dissolve existing thrombus
- Heparin plus ASA more beneficial than ASA alone: Risk for death or MI reduced by 33%

Heparin

- Pros:
 - Completely reversible
 - Inexpensive
 - Monitoring readily available
- Cons:
 - Possible delay until therapeutic
 - Thrombocytopenia is a possibility

LMWH Enoxaparin (Lovenox)

- Advantages as compared with U Heparin
 - More predictable anticoagulation effect
 - Lower incident of HIT
 - Easier to administer/not used in renal failure
 - Given as fixed-weight base dose
 - 15% reduction in major adverse cardiovascular events compared with UFH
 - 14-19% have received an excess dose of LMWH which is associated with major bleeding and death

Lovenox

- If bleeding occurs, may last awhile, only 60% reversible with protamine
- No testing available
- Can go home with home health
- Thrombocytopenia
- More expensive than heparin

ACC/AHA guidelines

- Enoxaparin (lovenox)
 - Reduce dosage in patients >75
 - Initial intravenous dose should be omitted
 - Reduction of maintenance dosage of 0.75 mg/kg sq every 12 hours should be used
 - Creatinine clearance is <30, DO NOT USE

Bivalirudin: Angiomax:

- A direct thrombin inhibitor: binds to circulating and clot bound thrombin therefore preventing further coagulation
- Approved for use in the ACS setting
- Shorter half-life potential to minimize the risk of bleeding
- Bolus and drip: adjust renal dosing CrCl
- Immediate onset of action

Fondaparinux (Arixtra)

- Inhibits thrombin generation, and thrombus formation by selectively binding to antithrombin III
- Has a long half life:dosing once daily
- STEMI 2.5 mg IV dose followed by 2.5 mg sq once daily
- No labs required, level of anticoagulation is consistent, no antidote available to reverse

Factor Xa Inhibitor

- Fondaparinux (Arixtra)
 - Rate of major bleeding was significantly lower as compared to enoxaparin (Lovenox)
 - Anticoagulant effect is not as easily reversed compared with UFH, therefore UFH is preferred for patients who are likely to have CABG within 24 hours

Quality Indicators for AMI

- Aspirin on arrival and discharge
- Beta Blocker on arrival and discharge
- ACE inhibitors or ARB
- Time to PCI should be within 90 minutes
- Reperfusion Therapy within 30 minutes
- Smoking Cessation
- LDL-Cholesterol Assessment
- Lipid-Lowering Therapy at discharge

Improving Quality: Pre hospital

- high risk period in the first hour
- reduction of door to needle time
- reduction of door to balloon time
- public education, AED, rapid response EMS
- Aspirin chewed in the field (162-325mg po)
- CAPTIM: 30 day mortality with prehospital fibrinolysis vs primary PCI within 2 hours of symptoms (2.2% vs 5.7 %)

Guidelines

- Studies show that adherence to guidelines means better patient outcomes
- More aggressive anticoagulation/antiplatelet therapy
- Selection of a strategy (initial invasive or initial conservative)
- Discharge planning and secondary prevention

MIND THE GAP

- Evidence-based care and actual care delivered
 - Lack of knowledge
 - Lack of acceptance of guidelines
 - Lack of systems for implementation
 - Need for frequent changes in practice due to rapid pace of research

CORE MEASURES: AMI

- Beta blocker 24 hours of arrival and discharge
- ASA 24 hours of arrival and discharge
- Time to thrombolysis less than 30 minutes
- Door to cath time less than 90 minutes
- Smoking Cessation Education
- ACE or ARB at discharge
- Statin /Anti-hyperlipidemic medication at discharge

Thank you



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Reference

- American Heart Association (AHA). (2003). Heart disease and stroke statistics 2003 update. Dallas, TX: National Center.
- Cunningham, S., Del Bene, S., & Vaughan, A. F. (2000). Myocardial ischemia pathogenesis of atherosclerosis. In Woods, S., Froelicher, E., & Motzer, S. (Eds.), *Cardiac nursing* (4th ed., pp.481-531). Philadelphia, PA: Lippincot.
- WELLENS, HEIN, & CONOVER, MARY (1992) THE ECG IN EMERGENCY DECISION MAKING, W.B. SAUNDERS, PHILADELPHIA, PA.
- Klatt, E. C. (2002). Myocardial infarction. Retrieved September 10, 2003, from Florida State University College of Medicine Web site: <http://www.medlib.med.utah.edu/WebPath/webpath.htm>
- Kosnik, L. (1999). Managing acute myocardial infarction-how can the process be improved: Treatment protocols and pathways: improving **the process of care**. *Critical Care Nurse*, 19(5), 1-19.

Reference

- Ross, J. (1999). Blood vessels. Retrieved September 20, 2003, from <http://www.partner2profit.com/redirect.cfm?cocode=ED3B5078&pcode=C31B7657>
- Stringer, K. & Lopez, L. (2002). Myocardial infarction. In Dipiro, J., Talbert, R., Yee, G., Matzke, G. Wells, B. & Posey (Eds.), *L Pharmacotherapy: A pathophysiologic approach* (5th ed., 251-272). New York, NY: McGraw-Hill.
- Talbert, R. (2002). Ischemic heart disease. In Dipiro, J., Talbert, R., Yee, G., Matzke, G. Wells, B. & Posey (Eds.), *L Pharmacotherapy: A pathophysiologic approach* (5th ed., 219-250). New York, NY: McGraw-Hill