Coronary Artery Disease

Dr. Amitesh Aggarwal
“If thou examinist a man for illness in his cardia, and he has pains in his arms, in his breasts and on one side of his cardia...it is death threatening him.”

Ebbell B, The Papyrus Ebers: The greatest Egyptian medical document. 1937, Copenhagen, Levin and Munkfgaard
Coronary anatomy
Spectrum of CAD

- CAD / IHD Stable Angina
- ACS
- UA
- NSTEMI
- STEMI

- Asymptomatic (subclinical)
- ICMP
- SCD
CAD Coronary Artery Disease
IHD Ischemic Heart Disease

- Condition in which there is an inadequate supply of blood and oxygen to a portion of the myocardium;
- Most common cause is atherosclerotic disease of an epicardial coronary artery sufficient to cause a regional reduction in myocardial blood flow and inadequate perfusion of the myocardium.
Angina

Discomfort in the chest or adjacent areas caused by myocardial ischemia but without myocardial necrosis.

(a) retrosternal pressure, pain, discomfort, or heaviness that

(b) radiates to the neck, jaw, left arm, or shoulder,

(c) precipitated by exertion and relieved by rest or nitroglycerin, lasting <10 minutes.
ACS

Spectrum of clinical presentations ranging from those for STEMI to presentations found in NSTEMI or in UA.

In terms of pathology, ACS is almost always associated with rupture of an atherosclerotic plaque and partial or complete thrombosis of the infarct-related artery.
Unstable Angina

Angina pectoris or equivalent ischemic discomfort with at least one of three features:

(1) it occurs at rest (or with minimal exertion), usually lasting >10 min;

(2) it is severe and of new onset (i.e., within the prior 4–6 weeks); and/or

(3) it occurs with a crescendo pattern (i.e., distinctly more severe, prolonged, or frequent than previously)
NSTEMI

Clinical features of UA

+ Evidence of myocardial necrosis
  (elevated cardiac biomarkers)

+ absence of persistent ST elevation
STEMI

WHO DEFINITION (1994)

Two out of three criteria

- Symptoms (chest pain > 20 minutes)
- ECG
- Bio-markers (CK MB, Trop T/I)
Universal Definition of Myocardial Infarction

Type 1; Type 2; Type 3; Type 4a; Type 4b; Type 5
Clinical Classification of MI
Type 1

- Spontaneous MI related to ischaemia due to primary coronary event such as plaque erosion & / or rupture
- It identifies MI due to atherosclerotic coronary arterial occlusion only
- Rise and/or fall of cardiac biomarkers (preferably troponin) above URL with evidence of at least 1 of the following:
  * Symptoms of ischemia
  * ECG changes indicative of new ischemia
  * Development of pathological Q waves
  * Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality
ECG changes in AMI
(In absence of LVH & LBBB)

- **ST elevation**
  - New ST elevation at the J-point in two contiguous leads with the cut-off points: $\geq 0.2$ mV in men or $\geq 0.15$ mV in women in leads $V_2$-$V_3$ and/or $\geq 0.1$ mV in other leads

- **ST depression &/or T-wave changes**
  - New horizontal or down-sloping ST depression $\geq 0.05$ mV in two contiguous leads; and/or T inversion $\geq 0.1$ mV in two contiguous leads with prominent R-wave or R/S ratio $>1$

- **New LBBB**

- **Development of Q-waves**
ECG changes in AMI
(In absence of LVH & LBBB)

- Hyper-acute T wave amplitude with prominent symmetrical T-wave in at least two contiguous leads is earliest manifestation of AMI

- ST segment equivalent – ST depression in V1-V3 with terminally positive T-wave with reciprocal ST elevation in V7-9

- Pseudonormalisation of previously inverted T-waves
Clinical Classification of MI
Type 2

- MI secondary to ischaemia due to either \( \uparrow \) 
  \( O_2 \) demand or decreased supply, eg, coronary artery spasm, coronary embolism, anemia, arrhythmias, HTN or Hypotension

- There is no coronary artery occlusion

- No scope of reperfusion therapy & antithrombotics
Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischaemia, accompanied by presumably new ST-elevation, or new LBBB, or evidence of fresh thrombus in a coronary artery by angiography and/or at autopsy, but death occurring before blood samples could be obtained, or at a time before the appearance of cardiac biomarkers in the blood.
Clinical Classification of MI

Type 4a

- Myocardial infarction associated with PCI
  - Embolisation of clot or debris
  - Slow flow or no reflow
  - Dissection
  - Side branch stenosis
  - Disruption of collaterals
    (increases > 3X URL of biomarker)

Type 4b

- MI associated with stent thrombosis as documented by angiography or at autopsy
Clinical Classification of MI
Type 5

- Myocardial infarction associated with CABG
- Biomarker > 5 X of URL during first 72 h following CABG, when associated with the appearance of new pathological Q-waves or new LBBB, or angiographically documented new graft or native coronary artery occlusion, or imaging evidence of new loss of viable myocardium
Epidemiology

Modified, WHO 2002
Mortality rates for CAD in Asian Indians in different countries
Indian Scenario

- Median age of first heart attack is 53 years
- Incidence of CAD in young Indians is 14%–16%
- Mortality attributable expected to rise by 113% in men and 94% in women from 1985 to 2015
- Indian risk of CAD is $3-4 \times$ White Americans $6 \times$ Chinese $20 \times$ Japanese.
- Manifests a decade earlier in ethnic Indians.
- Prevalence Urban (10.5 %) vs. Rural (4.5 %)
- Prevalence Men (7.4 %) vs. Women (4.5 %)
Coronary Artery Disease Other than Atherosclerosis

- Arteritis
- Takayasu disease
- Trauma
- Laceration, Radiation
- Coronary mural thickening
- Homocysteinuria
- Luminal narrowing
- Spasm (Prinzmetal angina)
- Emboli
- Infective endocarditis
- Congenital Anomalies
- ALCAPA
- O2 Demand-Supply Disproportion
- AS
- Miscellaneous
- Cocaine abuse
RISK FACTORS OF ATEROSCLEROSIS

Non modifiable
- Age
- Sex
- Menopause
- Family history of DM, HTN, IHD etc.
- Genetic profile
- Vascular anomaly (ostial obstruction)

Modifiable
- Smoking
- Hypertension
- Dyslipidemia
- Diabetes
- Insulin resistance
- Exercise/Obesity
- Sedentary life style
- Mental Stress
- Estrogen status
- Cocaine use

Novel Atherosclerosis Risk factors
- Homocysteine
- Fibrinogen
- Lipoprotein ‘a’
- Markers of Fibrinolytic function
- Markers of Inflammation (hs – CRP, IL – 6, PAI)
Abnormal lipids, smoking, hypertension, diabetes, abdominal obesity, psychosocial factors, consumption of fruits & vegetables, alcohol and regular physical activity account for most of the risk of MI worldwide in both sexes and at all ages in all regions.
• Each of these factors significantly increases the risk of CAD
• When these factors combine in single individual, their effects become multiplicative
Vascular Atherosclerosis is an ongoing process from day of creation to day of destruction.

- Fatty streak
- Transitional plaque
- Mature plaque
- Ruptured plaque with thrombus formation

- Thrombus
- Extra cellular lipid pool
- Foam cells
- Smooth muscle cells
- Fibrous cap
Evolution of Atherosclerotic Plaque
Pathophysiology of Stable and Unstable Plaques

Unstable plaque:
- Thin fibrous cap
- Thrombus
- Thick fibrous cap
- Smooth muscle cells
- Lipid rich core and macrophages
- Media

Stable plaque:

29
Non-Stenotic Vulnerable Plaques overall are More Dangerous Since they are far More Frequent than Stenotic Ones

Plaque remodeling

Angina

Discomfort in the chest or adjacent areas

(a) retrosternal pressure, pain, discomfort, or heaviness that

(b) radiates to the neck, jaw, left arm, or shoulder,

(c) precipitated by exertion, emotional stress, medical or surgical illness, morning and relieved by rest or nitroglycerin, lasting <10 minutes.
Anginal equivalents

- Dyspnea without pain
- Atypical location of the pain – epigastric
- Apprehension and nervousness
- Sudden mania or psychosis
- Syncope
- Profound weakness
- Acute indigestion
Unstable Angina/ NSTEMI

Angina pectoris or equivalent ischemic discomfort with at least one of three features:

(1) it occurs at rest (or with minimal exertion), usually lasting >10 min;

(2) it is severe and of new onset (i.e., within the prior 4–6 weeks); and/or

(3) it occurs with a crescendo pattern (i.e., distinctly more severe, prolonged, or frequent than previously)
STEMI

• Angina pectoris or equivalent ischemic discomfort
• May be accompanied by weakness, vomiting, sweating, dizziness, palpitations, cold perspiration, and sense of impending doom
• Pronounced circadian periodicity for the time of onset of STEMI, with peak incidence of events between 6 AM and 12 noon
Atypical presentations of STEMI

- heart failure - dyspnea without pain
- classic angina pectoris without severe or prolonged episode
- atypical location of the pain
- central nervous system manifestations
- apprehension and nervousness
- sudden mania or psychosis
- Syncope
- overwhelming weakness
- acute indigestion
- peripheral embolization
MI patients without chest discomfort

- More likely to be:
  - Older
  - Women
  - Diabetic and/or have prior heart failure
  - Delayed longer before they went to the hospital

- Less likely to be diagnosed as having an MI when admitted
- Less likely to receive fibrinolysis or primary PCI, aspirin, beta-blockers or heparin
- 2.2 times more likely to die during the hospitalization
ANGINA / MI WITH NORMAL CORONARY ARTERIES

- Seen in 6% of pt.
- More common in women (mechanism difficult to establish)
- Tend to be young and have relatively few coronary risk factors, except smoking.
- Usually no history of angina pectoris prior to the infarction.
- The infarction in these patients is usually not preceded by any prodrome,
- Clinical, laboratory, and ECG features of STEMI are otherwise indistinguishable from classical STEMI
- In patients who recover, areas of localized dyskinesis and hypokinesis can often be demonstrated by left ventricular angiography.
PHYSICAL EXAMINATION

- Frequently negative
- But careful search for
  - Valve disease (aortic valve)
  - Left ventricular dysfunction (cardiomegaly, gallop rhythm)
  - Manifestation of arterial diseases (carotid bruits peripheral vascular diseases)
  - Unrelated condition that exacerbate angina (anemia, thyrotoxicosis)
Findings & Implications in MI

CVS

- **General**: Restless, agitated, Levine’s sign
- **Skin**: Cool, clammy, pale, ashen
- **Low-grade fever**: response to myocardial necrosis
- **Hypertension, tachycardia**: ↑ sympathetic tone (AWMI)
- **Hypotension, bradycardia**: ↑ vagal tone (I/P MI)
- **Small-volume pulses**: Low cardiac output
- **Irregular pulse**: Atrial / vent arrhythmias, CHB
- **Paradoxical “ectopic” systolic impulse**: LV dyskinesis, ventricular aneurysm (AWMI)
Findings & Implications in MI

- **Soft S1:** ↓ LV contractility; 1° AV block (IW MI)
- **S4 gallop:** ↓ LV compliance
- **Paradoxically split S2:** Severe LV dys, LBBB
- **S3 gallop, pulmonary rales, pulsus alternans:** LV systolic dys (CHF >25% of myocardium)
- **↑ JVP, Kussmaul’s sign, hypotension, RV S4 and S3 gallops, clear lungs:** RVMI
- **Pericardial friction rub:** Pericarditis
- **Absent pulses and murmur of AR:** Aortic dissection

**CHEST**

May be normal or **few basal crackles:** Pulmonary congestion
EVALUATION

Asymptomatic
- LAB
- IMAGING

Symptomatic
- LAB
- IMAGING ED
RISK FACTORS

- Blood Sugar
- Serum Lipids
- hs-CRP
- Homocysteine
- Fibrinogen
- Lipoprotein (a)
Biomarkers in CAD

Inflammation
CRP, ALB, FBN, LC, SAA

Pro inflammatory markers.
IL-1B, IL-6, TNF-alpha

Endothelium
sICAM, P Selectin

Antiinflammatory cytokines
IL-4, IL-6

Matrix degradation enzymes
mMP-3,9

Cellular adhesion molecules
sIAM-1, SVCAM, SEs, Ps

Soluble cytokines receptors/antagonists
SIL-6, STNF, SIL
Prognosis with Biomarkers

Relative risks of future myocardial infarction among apparently healthy men
Imaging

combination of tests to diagnose the extent and spread of atherosclerosis

- ABI
- Stress testing
- CIMT
- CTA
- Doppler study
- IVUS
- MRA
- Angiography
- CCS
Ankle Brachial Index

- This index is ratio of SBP measured at ankle to SBP measured at brachial artery
- Normal ABI should be $\geq 1.0$
Flow Mediated Dilatation

- Early assessment of atherosclerosis
- Endothelial dysfunction is considered to be the first stage of atherosclerosis.
- Determining efficacy of treatment
- Cuff is inflated to 50 mm Hg above subject’s resting systolic pressure and remains inflated for 4 min. The cuff is then deflated and the 2-min image data are acquired
CIMT

- Carotid IMT measurement is a viable predictor of the presence of coronary atherosclerosis and its clinical sequelae.

**Carotid Artery Segments**

- Internal carotid
- Carotid bulb
- Common carotid
- Near wall
- Far wall
- External carotid
Exercise ECG / TMT

- to assess patients with suspected or proven cardiovascular disease
- to estimate prognosis and to determine functional capacity, the likelihood and extent of CAD and the effects of therapy.

In lead V4, the TMT is abnormal early in the test, reaching 0.3 mV (3 mm) of horizontal ST segment depression at the end of exercise. The ischemic changes persist for at least 1 minute and 30 seconds into the recovery phase. This type is consistent with a severe ischemic response.
MYOCARDIAL PERFUSION SCANNING

- Helpful in pt with uninterpretable exercise test
- Accuracy higher than exercise ECG
- Scintiscan of myocardium at rest and during stress after iv radioactive isotopes thallium.
- If Perfusion defect present during stress but not rest - reversible myocardial ischemia
- Persistence perfusion defect during both phase - previous MI
Patient with reversible perfusion defect in the inferior and anterior wall and an irreversible perfusion defect in the septum
Coronary calcium Scoring

- quantitatively assess coronary calcium using Agatston CCS
- surrogate for plaque burden
- shown to provide powerful prognostic information
- absence of coronary calcium (CCS 0), while not excluding the presence of noncalcified plaque, virtually excludes significant coronary atherosclerosis
Significant Coronary Artery Calcium
(Score >400)
IVUS

**Advantage**
- Vessel wall + lumen visualization
- Plaque characterization

**Disadvantage**
- Need to instrument vessels
- Limited to proximal segments
- Not as well validated for clinical decision making
Angiography Fails to Depict Coronary Arterial Remodeling
Symptomatic

- Serum biomarkers for cardiac damage
- Serum lipids
- Blood sugar
- ECG
- Echo
- Myocardial perfusion scans
- CTA/ MRA
- CAG
post-acute MI and Biomarkers

Normal - UA     Increased - NSTEMI / STEMI
Should be measured at  0hrs, 6-9hrs, 12-24 hrs after admission
## Biomarkers of Cardiac Damage

<table>
<thead>
<tr>
<th>Biomarker</th>
<th>Range of Times to Initial Elevation, h</th>
<th>Mean Time to Peak Elevations (Nonreperfused)</th>
<th>Time to Return to Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>CK-MB</td>
<td>3-12 h</td>
<td>24 h</td>
<td>48-72 h</td>
</tr>
<tr>
<td>cTnI</td>
<td>3-12 h</td>
<td>24 h</td>
<td>7-10 d</td>
</tr>
<tr>
<td>cTnT</td>
<td>3-12 h</td>
<td>12 h–2 d</td>
<td>7-14 d</td>
</tr>
<tr>
<td>Myoglobin</td>
<td>1-4 h</td>
<td>6-7 h</td>
<td>24 h</td>
</tr>
<tr>
<td><strong>CK-MB</strong></td>
<td><strong>Myoglobin</strong></td>
<td><strong>Troponins</strong></td>
<td></td>
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<tr>
<td>------------</td>
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<td>---------------</td>
<td></td>
</tr>
<tr>
<td>- Rapid, cost-efficient, accurate assays</td>
<td>- High sensitivity</td>
<td>- Greater sensitivity and specificity than CK-MB</td>
<td></td>
</tr>
<tr>
<td>- Ability to detect early reinfarction</td>
<td>- Useful in early detection of MI</td>
<td>- Detection of recent MI up to 2 weeks after onset</td>
<td></td>
</tr>
<tr>
<td>- Lack of specificity with skeletal muscle disease/injury</td>
<td>- Detection of reperfusion</td>
<td>- Detection of reperfusion</td>
<td></td>
</tr>
<tr>
<td>- Low sensitivity during early MI (&lt;6 h) or late (&gt;36 h) after symptom onset and for minor myocardial damage</td>
<td>- Most useful in ruling out MI</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Very low specificity with skeletal muscle injury or disease</td>
<td>- Rapid return to normal</td>
<td></td>
</tr>
</tbody>
</table>
ECG

Classic changes of necrosis (Q waves), injury (ST elevation), and ischemia (T wave inversion)
In recovery, the ST segment is earliest change to normalize. Then T wave; the Q wave usually persists for years after infarction.
- Hyperacute T Waves (over 50% of preceding R)
- ST-T elevation (>1mm in limb or precordial leads)
- ST depression in Lead V1, Lead V2 (Posterior MI)
- T Wave inversion
- Q Waves (.04 sec and 1/3 height of R Wave)
- New left ventricular strain pattern
- New Left Bundle Branch Block
Sequence of ECG Changes

1 minute after onset
1 hour or so after onset
A few hours after onset

A day or so after onset
Later changes
A few months after AMI
ECHO
Stress ECHO
Alternative to Myocardial perfusion scanning
Superior to exercise ECG
CT Angiography

- Assessment of symptomatic patients for the assessment of obstructive disease
- Higher radiation dosages contraindicate its use as a screening tool for asymptomatic patients
- Demonstrate the morphological consequences of ischemic heart disease
- Can assess ventricular function and perfusion
- Visualizing coronary arteries
CT Angiography
MRA

- Assessment of Ventricular volumes, mass, function
- Assessment of myocardial infarction and viability
- Stress ventriculography
- Coronary angiography and flow
- Identification of plaque components
- Non-invasive and no radiation
- Not useful for screening purpose
Coronary angiography

• Provide detailed information about the extent & nature of CAD
• Presence of dynamic coronary vascular lesions, such as spasm or thrombosis
• Consequences of CAD, - IMR or LV dysfunction
• Quantification of severity of both diastolic and systolic dysfunction
Treatment (Stable Angina)

- Identification and Treatment of Aggravating Conditions - AV disease, HCM, anemia
- Treatment of Risk Factors – HTN, smoking
- Drug Therapy
- PCI/ CABG
- Enhanced ECP
- Transmyocardial laser revascularization
Treatment (Stable Angina)

**Nitrates**
- systemic venodilation, reduction in LV EDV/P, reducing oxygen requirements; dilation of epicardial coronary vessels; increased blood flow in collateral vessels, improve exercise tolerance
- relieve ischemia in UA, Prinzmetal's variant angina

**Beta-blockers**
- reduce myocardial oxygen demand, inhibiting increases in heart rate, BP, contractility, esp during exercise, relief of angina, ischemia
- reduce mortality and reinfarction in patients after MI
Treatment (Stable Angina)

Calcium antagonists

- Coronary vasodilators, variable reductions in myocardial oxygen demand, contractility, BP
- Indicated when BB contraindicated, poorly tolerated, ineffective. sick-sinus syndrome, AV conduction disturbances, symptomatic PAD, Prinzmetal's angina
- Amlodipine and beta blockers have complementary actions on coronary blood supply and myocardial oxygen demands.
- Beta blockers have shown to improve life expectancy following acute MI while CCB have not
Treatment (Stable Angina)

- **Aspirin/ Clopidogrel**
- **(ACE) inhibitors** - post MI; HTN, chronic IHD, DM, diabetes, LV dysfunction
- **Potassium channel activators** - *nicorandil* - open ATP-sensitive potassium channels in myocytes
- **Metabolic modulators**
  - **Trimetazidine** - exert anti-ischaemic properties without affecting myocardial oxygen consumption and blood supply, affects myocardial substrate utilization by shifting energy production from FFA to glucose oxidation.
  - **Ranolazine** - symptomatic chronic angina max Rx, inhibits late inward sodium current ($I_{Na}$)
  - **Perhexiline, etomoxir**
Treatment Goals (ACS)

- Restore blood flow to prevent infarct expansion - STK/ PCI/ CABG
- Prevent death, complications
- Relieve ischemic chest discomfort
- Prevent coronary artery reocclusion
Early Pharmacotherapy for ACS

- Intranasal oxygen
- IV NTG - control ischemia
- Morphine
- β-blocker - control ischemia
Antiplatelet

- Thromboxane A2 inhibitor
  -- aspirin
- ADP receptor blockers
  - Irreversible (ticlopidine, clopidogrel, prasugrel)
  - Reversible (cangrelor)
- Phosphodiesterase inhibitors
  - Dipyridamol
  - Cilostazol
- Glycoprotein IIb/IIIa antagonists indicated for patients undergoing 1° PCI in combination with ASA, clopidogrel, & UFH
  - Abciximab
  - Tirofiban
  - Eptifibatide
Anticoagulant

- **UFH**
- **LMWH** (enoxaparin, dalteparin)
- **Factor Xa Inhibitor** (fondaparinux)
  - alternative to UFH in patients not undergoing reperfusion
  - receiving fibrinolytics
  - not recommended for use alone in 1° PCI
- **DTI** (Lepirudin, Bivalirudin, Argatroban)
  - option in patients undergoing planned 1° PCI
  - Inhibit clot-bound & circulating thrombin
  - Antiplatelet activity
- **VKA** (warfarin)
Fibrinolytic

Fibrin non-specific agents
- Streptokinase
- Anistreplase
- Urokinase

Fibrin-specific agents
- rt-PA (alteplase)
- Variants of t-PA
  - Substitution (monoteplase, tenecteplase)
  - Deletion (reteplase, lanoteplase, pamiteplase)
## Comparison of Fibrinolytic Agents

<table>
<thead>
<tr>
<th>Agent</th>
<th>Fibrin Specificity</th>
<th>Complete Perfusion at 90 Minutes</th>
<th>Bleeding risk</th>
<th>Administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Streptokinase</td>
<td>+</td>
<td>35%</td>
<td>+++/++</td>
<td>Infusion over 60 minutes</td>
</tr>
<tr>
<td>Alteplase</td>
<td>+++</td>
<td>50-60%</td>
<td>++/+++</td>
<td>Bolus followed by infusions over 90 minutes</td>
</tr>
<tr>
<td>Reteplase</td>
<td>++</td>
<td>50-60%</td>
<td>++/+++</td>
<td>Two bolus doses, 30 minutes apart</td>
</tr>
<tr>
<td>Tenecteplase</td>
<td>++++</td>
<td>50-60%</td>
<td>+/-++</td>
<td>Single bolus dose</td>
</tr>
</tbody>
</table>
Contraindications to Fibrinolysis

**Absolute contraindications**

- active internal bleeding (not including menses)
- previous intracranial hemorrhage at any time
- ischemic stroke within 3 months
- intracranial neoplasm
- structural vascular lesion (e.g., AVM)
- suspected aortic dissection
- closed head or facial trauma within 3 months
Relative Contraindications

- uncontrolled HTN (BP > 180/110 mm Hg)
- ischemic stroke > 3 months
- dementia
- intracranial pathology
- current anticoagulant use
- bleeding diathesis
- traumatic or prolonged CPR (> 10 min)
- major surgery (< 3 wks)
- noncompressible vascular puncture
  - recent liver biopsy
  - carotid artery puncture
- recent internal bleeding (within 2 to 4 wks)
- previous streptokinase use (> 5 days) or prior allergic reaction
- pregnancy
- active peptic ulcer
- history of severe, chronic, poorly controlled HTN
Successful thrombolysis

Clinical
Resolution of chest pain

ECG
> 50% decrease in max ST elevation at 90 minutes after start of STK

Enzymes
Early peak (3 hrs) of CK-MB

CAG
TIMI III flow
PCI

Percutaneous Coronary Interventions

- Balloons 10%
- Stents 80%
- Atherectomy 10%
PCI

- As an alternative to thrombolytic therapy in patients with AMI and STEMI or new or presumed new LBBB who can undergo angioplasty of the infarct artery <12 hr from the onset of ischemic symptoms or >12 hr if symptoms persist, if performed in a timely fashion (performance standard: balloon inflation within 90 ± 30 min of hospital admission) by individuals skilled in the procedure.

- In patients who are within 36 hr of an acute STEMI/Q wave or new LBBB MI who develop cardiogenic shock, are younger than 75 yr, and revascularization can be performed within 18 hr of the onset of shock by individuals skilled in the procedure.

- As a reperfusion strategy in candidates who have a C/I to thrombolytic therapy.

- Objective evidence for recurrent infarction or ischemia.
PCI

- Cardiogenic shock or hemodynamic instability
- Recurrent angina without objective evidence of ischemia or infarction
- Angioplasty of the infarct-related artery stenosis within hours to days (48 hr) following successful thrombolytic therapy in asymptomatic patients without clinical and/or inducible evidence of ischemia
- Spontaneous or provocable myocardial ischemia during recovery from infarction
- Patients with LV ejection fraction <0.4, CHF, or serious ventricular arrhythmias
- All patients after a non-Q-wave MI
CABG

Surgery reroutes or bypasses blood around clogged arteries

Grafts  Arterial  - Radial artery, internal mammary artery
          Venous – Long saphenous vein

Indications
- Triple vessel disease
- Left Main stem disease
- Failed PCI
- Diffuse disease not amenable to PCI
- Severe LV dysfunction or DM

• Patients with ACS (NSTEMI and STEMI) need early invasive strategy
• PCI or CABG depends upon anatomy of vessels and availability of facilities and expertise
PCI vs STK

• STEMI patients should receive either fibrinolysis or primary PCI within 3 hrs of symptom onset
  – PCI: preferred treatment in capable centers/ high risk patients/ failed STK

18TH to 20th century
Balia district to max saket
Post MI Management

- ASA
- Nitrates
- $\beta$-blocker
- ACE-inhibitors
- Statins
- Aldosterone antagonists/diuretics
- Anti-coagulation
  - large infarcts (especially anterior), CHF, LV thrombus, DVT, EF< 35 %, AF
Complications of MI

- Extension / Ischemia
- Arrhythmia
- Pericarditis
- Expansion / Aneurysm
- RV Infarct
- Mechanical
- Acute MI
- Heart Failure
- Mural Thrombus
ARRHYTHMIAS

- Most common complication
- PVCs ~ 90%, SVT ~ 10%, CHB ~ 20% RV infarct
- Bradyarrhythmias – common with inferior MI
- **Ventricular** fibrillation (2-4%)
  - resuscitation
  - defibrillation
- **Atrial** fibrillation
  - may not require treatment
  - If hypotension occurs – DC cardioversion
Indications for Permanent Pacing

- persistent complete (third-degree) AV block
- persistent sinus node dysfunction - symptomatic bradycardia
- intermittent second-degree Mobitz II or third-degree AV block
- second-degree Mobitz II or third-degree AV block with new bundle branch block
Extension / Ischemia

Post infarct angina – 50%
Inc of CK-MB > 50% than previous nadir
Management same as unstable angina

In distribution of infarct vessel:
- IRA reperfusion, then reocclusion
- thrombus propagation, distal embolization

At a distance:
- reduced collateral flow from IRA
- new coronary thrombus
- reduced systemic perfusion pressure
- increased myocardial oxygen consumption

Treatment:
- Pharmacologic (beta blockers, nitrates)
- Urgent revascularization
- Repeat lytics (antibodies to SK)
Pericarditis

- Commonly occurs on 2\textsuperscript{nd} - 3\textsuperscript{rd} day of infarction
- Results from infarction extending to epicardial surface of heart, with associated inflammatory response

Post MI Syndrome (Dressler’s)

- Fever
- Pericarditis
- Pleurisy
- Weeks to month after infarction
- High dose aspirin or corticosteroids
Right Ventricular Infarction

- Associated with occlusion of proximal RCA
- Classic triad by hypotension, ↑JVP, clear lungs
- ECG: ST↑ in RV4
- Echo: RV dilation and hypokinesia

**Management**

- Usually transient ischemic dysfunction with long-term recovery common
- Marked sensitivity to preload reduction (nitrates)
- Fluid volume infusion for hypotension and low cardiac output
Infarct Expansion “Aneurysm”

- Circumscribed non-contractile outpouching of LV
- Usually composed of fibrous tissue + necrotic muscle +/- viable myocardium.
- Develops in 8 – 15% of patients post MI
- Common after AWMI with totally occluded poorly collateralized LAD
- Rarely seen with multivessel disease

Potential consequences:

- Mural thrombus +/- embolization
- Adverse LV remodeling and CHF
- Ventricular rupture
- Ventricular arrhythmias
Differences between a pseudoaneurysm and true aneurysm:

**True aneurysm**
1. Wide base
2. Walls composed of myocardium
3. Low risk of free rupture

**Pseudoaneurysm**
1. Narrow base
2. Walls composed of thrombus and pericardium
3. High risk of free rupture
Infarct Expansion “Aneurysm”
Heart (LV) failure & shock

- Cardiogenic shock:
  - 6 % of STEMI, 2 % of NSTE ACS
  - Mortality rate: 60%
  - LV wall > 40% infarcted

- Diastolic or systolic dysfunction may predominate
  - Extensive LV infarctions
  - Impaired relaxation, compliance
  - Extensive RV infarction or ischemia
  - VSD or acute severe MR
  - Tamponade (with or without free wall rupture)
  - Others - sepsis, beta- or Ca\(^{+2}\)-blocker overdose, pulmonary embolism
Heart (LV) failure & shock

- Correction of hypoxemia, acidosis, bradycardia, AV block, new onset AF
- Mechanical circulatory support + inotropic support
- Correction of hypoxemia, acidosis, bradycardia, AV block, new onset AF
- Mechanical circulatory support + inotropic support
- Intraaortic Balloon Counterpulsation
  - Extremely effective in supporting patients undergoing coronary angiography, PTCA, and CABG in cardiogenic shock.
  - Provides bridging support until an LV assistance device can be implanted or cardiac transplantation can be performed
- LV and biventricular assistance devices
- Percutaneous cardiopulmonary bypass support
LV thrombus and emboli

- Incidence of clinically evident systemic embolism after MI < 2%.
- Increases in patients with AWMI
- Incidence of mural thrombus after MI - 20%
- Systemic embolism -10% of LV thrombus
- Risk is highest in the first 10 days but persists at least 3 months
Mechanical complications

Ventricular septal rupture
2-5 days post MI

Free wall rupture
within 2 weeks post MI

Ischemic MR
13 hours post MI
Ventricular Septal Rupture

- Incidence - 1-3% of transmural MIs
- Medical stabilization and IABP
- Early surgical repair for decompensated pts
- Small asymptomatic VSDs may not require repair
- Sudden appearance of loud systolic murmur and thrill medial to the apex along the left sternal border in the 3rd or 4th intercostal space, accompanied by hypotension with or without signs of LV failure, is characteristic
Acute Mitral Regurgitation

- Transient MR common in early MI (20-40%)
- Persistent MR, even mild, associated with increased long-term mortality post-MI
- Due to papillary muscle or chordal rupture or dilation of ventricle and annulus
- Most common with inferior MI
- Early transient late apical systolic murmur thought to represent papillary muscle ischemia
- Sudden hemodynamic deterioration common
- Stabilize medically, IABP, then surgical repair
Free Wall Rupture

- Less frequent (1-3.4%), but earlier with STK
- Uncontained $\rightarrow$ sudden death or asystole
- Pseudoaneurysm $\rightarrow$ transient hypotension, bradycardia, repetitive emesis, restlessness
- Echocardiogram usually diagnostic
- Surgical repair - may require pericardiocentesis for uncontained rupture
Post MI Complications

“ACT RAPID”

- Arrhythmias
- Congestive Heart Failure
- Tamponade / Thromboembolic disorder
- Rupture (Ventricle, septum, papillary muscle)
- Aneurysm (Ventricle)
- Pericarditis
- Infection
- Death / Dressler’s Syndrome
PREVENTION

PRIMORDIAL

• Preventing spread of CHD risk factors and lifestyles that have not yet appeared or become endemic by Mass education

PRIMARY

• Aims at reversing risk factors that have established themselves

• Population strategy, High risk strategy

• It includes :- TLS modifications
2°MI Prevention Goals

- Control modifiable CAD risk factors
- Prevent development of systolic HF
- Prevent recurrent MI, stroke
- Prevent death, including SCD
2°MI Prevention Drugs

- ASA
- \( \beta \)-blocker
- ACE- inhibitors
- Statins
Exercise

- Walking, jogging or bike riding
- Intensity – 70 – 85% of Heart Rate max
- Duration – 30 minutes with warm up 5 minutes and cool down 5 minutes
- Frequency - 5 times a week

Dietary goals

- Fat limited to 20-30% of total daily intake
- Saturated fats upto 10%
- PUFA upto 10%
- Refined carbohydrate
- Protein upto 15%
- Fibre upto 20-30mg%
- Cholesterol <200mg%
17th dynasty princess, “Ahmose-Meryet-Amon"
1550-1580 BCE
Horus Study of Ancient Egyptian Mummies

♂ 33M  40±10.2 y
♀ 17F  37.6±12 y
Thank you

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