Coronary artery disease: Prevention, Recognition, Treatment, and Management

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• Prevalence of coronary artery disease, risk factors and application of risk scores
• Signs of coronary artery disease, tests that are routinely used and available for diagnosis, implications of the results
• Management of coronary artery disease and practical matters when it comes to follow up
• Cardiovascular diseases includes group of diseases that includes both the heart and blood vessels

• Primary goal for today’s talk will be to focus on coronary artery disease which is specifically the disease of blood vessels
Coronary heart disease includes both coronary artery disease and acute coronary syndromes.

Acute coronary syndrome is a subset of coronary heart disease.

Coronary heart disease is a major cause of morbidity and mortality in developed countries.
Case

• 49 year old male with a history of hypertension, who presents to family practice clinic with heartburn and belching that started the night before.
• This feeling was substernal radiating throughout the chest and into the back to the interscapular region
• +++sweating with these symptoms
• Now he has vague achiness throughout the chest
• Tried Rolaid and Tums but did not get relief
• Referred for EKG and labs.
• Coronary heart disease is the leading cause of death in the US
• Incidence of mortality has declined over the years, however it is still the cause of a 1/3 of all deaths in patients over the age of 35.
• 15.5 million persons ≥20 years of age in the USA have coronary heart disease (2016 Heart disease and Stroke updates)
Data from 44 years of follow-up in the original Framingham study cohort and 20 years of surveillance of their offspring has led to the following observations for initial incidence of heart disease:

- For people aged 40 years, lifetime risk of developing CHD was 49% in men and 32% in women
- For people who reached 70 years, lifetime risk was 35% and 24% in women
• Incidence for total coronary events rise steeply with age
• Women lag behind men by 10 years
• For more serious events such as myocardial infarction and death, women lag behind men by 20 years
• Serious manifestations such as myocardial infarction and sudden cardiac death were infrequent in premenopausal women
• Burden of heart disease was significantly higher in post menopausal women.
• Patients are typically asymptomatic for years
• Despite the lack of symptoms, presence and extent of non-obstructive coronary artery disease are associated with a worse prognosis compared with patients with no evidence of CHD.
Primary Prevention

• Lifestyle Modification
  – Exercise – 150 minutes of moderate intensity or 75 minutes of vigorous aerobic/week
  – Diet –
    • DASH (Dietary Approaches to Stop Hypertension) diet (AHA)
    • Reducing Saturated fat intake, 5 portions of fruits/vegetables/week (NICE)
    • Switch from saturated to poly-unsaturated fats, increase in fibre, fruit, vegetable, and fish intake, adherance to Mediterranean type diet (ESC)
  – Smoking Cessation
    • Smoking doubles 10-year CVD mortality rate
Primary Prevention

• Weight
  – BMI > 25 is a risk factor for CVD

• Alcohol
  – Controversial

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5331469/
Primary Prevention

• Lipid lowering therapy
• Anti-hypertensive therapy
• Blood glucose
• Anti-platelet therapy
  – Anti-platelet therapy is a significant contributor to secondary prevention but should be avoided in primary prevention in those without comorbidities due to increased bleeding risk with no evidence of CVD risk reduction.
  – In patients with DM the advice is conflicting: ESC guidelines maintain that the bleeding risk exceeds the benefits of aspirin therapy, whilst the American College of Chest Physicians recommend aspirin therapy in patients with DM and 10-year CVD event risk of $\geq 10\%$
CORONARY HEART DISEASE

• Stable Angina
• Acute coronary Syndromes
  – Unstable Angina
  – Non ST-Elevation Myocardial Infarction
  – ST- Elevation Myocardial Infarction
Angina: CCS classification

<table>
<thead>
<tr>
<th>Class</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I</td>
<td>No angina with ordinary physical activity (e.g., walking, climbing stairs). Angina with strenuous or prolonged exertion.</td>
</tr>
<tr>
<td>Class II</td>
<td>Early-onset limitation of ordinary activity (e.g., walking rapidly or walking &gt;2 blocks; climbing stairs rapidly or climbing &gt;1 flight); angina may be worse after meals, in cold temperatures, or with emotional stress.</td>
</tr>
<tr>
<td>Class III</td>
<td>Marked limitation of ordinary activity.</td>
</tr>
<tr>
<td>Class IV</td>
<td>Inability to carry out any physical activity without chest discomfort. Angina occurs during rest.</td>
</tr>
</tbody>
</table>
• There has been a relative increase in non-ST elevation myocardial infarction in relation to ST elevation myocardial infarction.
• Change in proportion is because of an absolute decrease in the incidence of ST elevation myocardial infarction.
Although many cases of myocardial infarction appear to occur without warning, there are people where heart disease is detectable prior to the occurrence of a sudden event.

Monitor for ominous coronary risk profile and signs of pre-symptomatic CHD.

2-4% of general population has silent coronary ischemia which can be detected with an exercise test or on an EKG.

Prevalence of this condition is considerably higher in men with two or more major coronary risk factors.
EKG

• Signs on EKG that might reflect silent heart disease
  – Left ventricular hypertrophy,
  – Intraventricular conduction disturbances
  – Nonspecific repolarization abnormalities
## The 5 ECG AMI Patterns on the 12 Lead ECG

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Leads/Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td>V Leads, 1, L</td>
</tr>
<tr>
<td>Inferior</td>
<td>2, 3, F</td>
</tr>
<tr>
<td>Lateral</td>
<td>I, L, V₅, V₆</td>
</tr>
<tr>
<td>Right Sided</td>
<td>Deep ST ↓ V₁, V₂ in inferior AMI</td>
</tr>
<tr>
<td>Posterior</td>
<td>V₂: especially in inferior AMI R &gt; S ST ↓ T wave upright</td>
</tr>
</tbody>
</table>

**ACS: ECG**
Risk Factors

- Coronary risk factor profiles of patients with previously unrecognized myocardial infarction is similar to that of patients with clinically recognized myocardial infarction.
- Hypertension & Diabetes $\Rightarrow$ higher likelihood
Risk scores

• Lifetime ASCVD Risk calculator at initial visit
  – Estimates of lifetime risk for ASCVD provided for adults 20 through 59 years of age
  – Primary use is to facilitate discussion regarding risk reduction through lifestyle change
  – Tools.acc.org/ASCVD-Risk-Estimator-Plus
  – App stores: “ASCVD risk estimator plus”
TIMI Risk Score

Predicts risk of death, new/recurrent MI, need for urgent revascularization within 14 days

1) Age ≥ 65 (1 point)
2) ≥ 3 CAD Risk Factors (1 point)
3) Known CAD (stenosis ≥50%) (1 point)
4) ASA use in past 7 days (1 point)
5) Recent (<24H) severe angina (1 point)
6) ST deviation ≥ 0.5 mm (1 point)
7) ↑ Cardiac Markers (1 point)

RISK SCORE = Total Points

Risk Score:
- 0/1: 4.7
- 2: 8.3
- 3: 13.2
- 4: 19.9
- 5: 26.2
- 6/7: 40.9

Antman et al. JAMA 2000; 284:835
GRACE Score

- Age
- Killip Class
- Systolic blood pressure
- Presence of ST segment deviation
- Cardiac arrest during presentation
- Serum Creatinine concentration
- Presence of elevated serum cardiac biomarkers
- Heart rate
## GRACE risk score

<table>
<thead>
<tr>
<th>Risk category (tertile)</th>
<th>GRACE risk score</th>
<th>In-hospital death (percent)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>≤108</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Intermediate</td>
<td>109-140</td>
<td>1-3</td>
</tr>
<tr>
<td>High</td>
<td>&gt;140</td>
<td>&gt;3</td>
</tr>
</tbody>
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<th>Risk category (tertile)</th>
<th>GRACE risk score</th>
<th>Post-discharge to six-month death (percent)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>≤88</td>
<td>&lt;3</td>
</tr>
<tr>
<td>Intermediate</td>
<td>89-118</td>
<td>3-8</td>
</tr>
<tr>
<td>High</td>
<td>&gt;118</td>
<td>&gt;8</td>
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Angina Pectoris

- Sudden severe chest pain or heaviness radiating to the jaw, back, or neck
- Associated with tachypnea, diaphoresis, or nausea
- Typically associated with exertion
Acute Coronary Syndromes (ACS)- Definition

• Spectrum of conditions compatible with acute myocardial ischemia and/or infarction that are usually due to an abrupt reduction in coronary blood flow
• Hallmark of ACS is sudden imbalance between myocardial oxygen consumption and demand – usually the result of coronary artery obstruction
Algorithm for evaluation and management of patients suspected of having ACS

A
Symptoms suggestive of ACS

B1
Noncardiac diagnosis

C1
Treatment as indicated by alternative diagnosis

B2
Chronic stable angina

C2
See ACC/AHA Guidelines for Chronic Stable Angina

B3
Possible ACS

B4
Definite ACS

C3
ST-elevation

D1
Nondiagnostic ECG
Normal initial serum cardiac biomarkers

D2
ST and/or T wave changes
Ongoing pain
Positive cardiac biomarkers
Hemodynamic abnormalities

E1
Observe
12 hours or more from symptom onset

D3
Evaluate for reperfusion therapy
ACS

Presentation

Working Dx

ECG

Cardiac Biomarker

Final Dx

Ischemic Discomfort

ACS

No ST Elevation

ST Elevation

No ST Elevation

ST Elevation

NSTE-ACS

STEMI *

UA

Unstable Angina

NSTEMI *

ST Elevation

Myocardial Infarction

NQMI

QwMI

Noncardiac Etiologies
ACS: Pathophysiology

• Flow reduction may be related to
  – a completely occlusive thrombus or
  – subtotally occlusive thrombus
  – The spectrum of clinical presentations including UA, NSTEMI, and STEMI is referred to as ACS.
  – Patients with noncardiac etiologies make up the largest group presenting to the ED with chest pain
**Unstable Angina**

- Non-occlusive thrombus
- Non-specific ECG
- Normal cardiac enzymes

**NSTE MI**

- Non-occlusive thrombus sufficient to cause tissue damage & mild myocardial necrosis
- ST depression +/- T wave inversion on ECG
- Elevated cardiac enzymes

**STEMI**

- Complete thrombus occlusion
- ST elevations on ECG or new LBBB
- Elevated cardiac enzymes
- More severe symptoms
Pathophysiology of Stable Angina and ACS

### Decreased O₂ Supply
- Flow-limiting stenosis
- Anemia
- Plaque rupture/clot

### Increased O₂ Demand

#### O₂ supply/demand mismatch → Ischemia

#### Myocardial ischemia → necrosis

ACS

Pathophysiology of Stable Angina and ACS
ACS Pathophysiology
Evolution of Coronary Thrombosis

- Healing thrombus, ↑SMC, WBC
- Sudden growth in atherosclerotic plaque: Patient remains asymptomatic or develops stable angina
- Thrombosis < Fibrinolysis
- Partial clot resolution: Unstable angina NSTEMI
- Transient occlusion: angina
- Clot continues to propagate: STEMI

"Vulnerable" plaque
- Thin fibrous cap
- Large lipid pool
- ↑White blood cells (WBCs)
- ↓Smooth muscle cells (SMCs)

Plaque rupture
- Prothrombotic molecules exposed and thrombus forms
1) normal artery
2) extracellular lipid in the subintima
3) fibrofatty stage
4) procoagulant expression and weakening of the fibrous cap.

ACS DEVELOPS WITH…
5) disruption of the fibrous cap, which is the stimulus for thrombogenesis.
6) Thrombus resorption may be followed by collagen accumulation and smooth muscle cell growth.

Thrombus formation and possible coronary vasospasm reduce blood flow in the affected coronary artery and cause ischemic chest pain.
ACS: Initial Evaluation

- History and Physical
- ECG
- Cardiac biomarkers
- Risk assessment models
- Clinical prediction algorithms
ACS: History

- **Ischemic chest pain** — Ischemic pain has a number of features that tend to distinguish it from noncardiac pain (OPQRST mnemonic)
  - **Onset**
  - **Provocation and palliation** — Ischemic pain is generally provoked by an activity, such as exercise, which increases cardiac oxygen demand
  - **Quality** —
  - **Radiation** — Ischemic pain often radiates to other parts of the body including the upper abdomen (epigastrium), shoulders, arms (upper and forearm), wrist, fingers, neck and throat, lower jaw and teeth (but not upper jaw), and not infrequently to the back (specifically the interscapular region).
  - **Site** — Ischemic pain is not felt in one specific spot, but rather it is a diffuse discomfort that may be difficult to localize.
  - **Time course** — Angina is usually brief (two to five minutes) and is relieved by rest
ACS: History

• Features of non-cardiac chest pain:
  – Pleuritic pain, sharp or knife-like pain related to respiratory movements or cough
  – Primary or sole location in the mid or lower abdominal region
  – Any discomfort localized with one finger
  – Any discomfort reproduced by movement or palpation
  – Constant pain lasting for days
  – Fleeting pains lasting for a few seconds or less
  – Pain radiating into the lower extremities or above the mandible
ACS: Physical Exam

- Evaluate for signs of
  - Heart Failure
  - Cardiogenic Shock
  - Neurologic symptoms
  - Aortic Dissection
Tests

• Non-invasive testing
  – Echocardiogram
    • Ejection fraction
    • Wall motion abnormalities
    • Valvular abnormalities (example: severe aortic stenosis)
    • Congenital conditions like hypertrophic cardiomyopathy

  – Stress testing

• Lab testing
Implications of test results

• Stress tests
  – Routine treadmill stress test
  – Stress echocardiogram
  – Treadmill nuclear stress tests
  – Pharmacologic nuclear stress tests
• Cardiac Catheterization
• **Stents**

  – **Drug eluting stents**
    * Dual antiplatelet therapy for at least one year
    * Aspirin 81mg daily (lifelong)
    * AND
      – Plavix 75mg daily for one year OR,
      – Effient 10mg daily for one year OR,
      – Brilinta 90mg twice daily for one year

  – **Bare metal stents**
    * Dual antiplatelet therapy for at least one month
Management of CAD

- Dual antiplatelet therapy
- High-intensity statin therapy
- Beta blocker
- ACE Inhibitor
Alternate causes of oxygen mismatch

- vasospastic [Prinzmetal] angina,
- coronary embolism
- coronary arteritis
- noncoronary causes of myocardial oxygen supply-demand mismatch (e.g., hypotension, severe anemia, hypertension, tachycardia, hypertrophic cardiomyopathy, severe aortic stenosis);
- nonischemic myocardial injury (e.g., myocarditis, cardiac contusion, cardiotoxic drugs); and
- multifactorial causes that are not mutually exclusive (e.g., stress [Takotsubo] cardiomyopathy, pulmonary embolism, severe heart failure, sepsis)
Practical Matters
Back to the Case

• Patient was taken emergently to the cardiac cath lab

• Cath showed 100% occlusion of the proximal RCA