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ACUTE CORONARY SYNDROMES

"ACS"

Raed Abu Sham'a, MD

Internist and cardiologist

Cardiac pacing and Electrophysiologist Senior Medical Education Officer



ACUTE CORONARY SYNDROMES LEARNING OBJECTIVES

- Define acute coronary syndromes (ACS)
- Understand the pathophysiology



- Be capable of risk stratification
- Aware of medications and strategies employed to manage ACS
- Use basic principles of ECG interpretation and infarct localization
- Apply knowledge to case studies



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CASES

- 67 year old male
 - 8/10 chest pain with radiation
 - Nausea, diaphoresis, unwell
- 65 year old female
 - Chest pain off and on for 1 month worse recently
 - associated diaphoresis and nausea
- 37 year old male
 - Chest pain
 - No associated symptoms



DEFINITION

• "constellation of symptoms manifesting as a result of acute myocardial ischemia"

Pollack et.al. 41(3), 2003

Spectrum of disease:

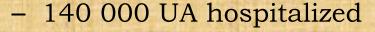
- Unstable Angina (UA)
- Non ST Elevation MI (NSTEMI)
- ST Elevation MI (STEMI)





EPIDEMIOLOGY

- Among leading cause of death and hospitalizations world wide
- Canada:
 - 80 000 AMI/year
 - 20 000 deaths



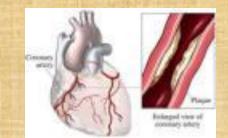


- death or nonfatal AMI within one year for 10 000 discharged
- 500 000 ED visits for evaluation of chest pain and associated symptoms
- >12% confirm myocardial injury



ETIOLOGY

- Atherosclerotic plaque rupture *
 - inflammation
 - thrombosis
- Vasospasm
- Dissection
- Decreased oxygen delivery (e.g. anemia, hypotension)
- Increased oxygen consumption (e.g. sepsis, thurotoxicosis)



ACUTE CORONARY SYNDROMES PATHOPHYSIOLOGY

Atheromatous plaque

· Contained within coronary intima by thin cap

Within the core, lipid laden "foam cells" produce the procoagulant,
 tissue factor (TF)

Rupture occurs at the shoulder



ACUTE CORONARY SYNDROMES PATHOPHYSIOLOGY

- TF + VIIa, generates Xa = thrombin production
- Platelets are activated by exposure to:
 - collagen, von Willebrand factor, thrombin
- Further activation and induction of vasospasm with:
 - adenosine diphosphate, thromboxane A2 and prostacyclins
- Activated platelets cross link fibrinogen



• RESULT: occlusive thrombus



CLINICAL FEATURES

- History:
- symptom onset, duration, exacerbators, palliators
- cocaine use
- Physical Examination:
 - vital signs
 - inspection
 - · distress, work of breathing, pulsations
 - palpation
 - edema, peripheral pulses, thrill/bruits, PMI, JVP
 - auscultation
 - heart sounds, murmurs, bruits
 - · pulmonary adventitia





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CLINICAL FEATURES

ACS associated symptoms:

- Diaphoresis *
- Nausea and vomiting
- Dyspnea
- Lightheadedness/Syncope
- Palpitations





ACUTE CORONARY SYNDROMES Stable Angina

- Does <u>not</u> predict acute events
- Marker of established coronary artery disease (CAD)
 - Fixed lesion / partially occluded vessel
 - Mismatch in oxygen supply and demand
- Precipitants:
 - Exercise
 - Cold
 - Stress
- Duration:
 - </= 15 to 20 minutes
- Relief:
 - Rest
 - Nitroglycerine



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CLINICAL FEATURES

Anginal Equivalents:

angina = visceral sensation that is poorly defined and localized

- Diaphoresis
- Dyspnea
- Discomfort in areas of radiation (jaw, shoulder, arm)
- GI complaints (inferior AMI)
- Dizziness, weakness, presyncope

Atypical Presentations:

- Up to 30%
- Female, Elderly, Diabetic patients





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Unstable Angina

- Clinical Presentation:
- I. New Onset Angina
 - Within past 1-2 months
 - CCS III or IV
- II. Crescendo Angina
 - Previous stable angina which has become <u>more</u> frequent, severe, prolonged, easily induced or <u>less</u> responsive to nitroglycerine
- III. Rest Angina
 - Angina occurring at rest and lasting more than 15-20 minutes





CANADIAN CARDIOVASCULA SOCIETY(CCS)

CLASSIFICATION FOR ANGINA

Can J Cardiol 1996; 12: 1279-92

• Class I:

- Ordinary physical activity

• Class II:

- Slight limitation of ordinary physical activity
- Angina occurs with walking > 2 blocks, climbing stairs, emotional stress

• Class III:

- Severe limitation of ordinary physical activity
- Angina occurs with walking < 1-2 blocks or climbing <1 flight of stairs in normal conditions

• Class IV:

Inability to carry out physical activity without discomfort:
 angina may be present at rest

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ACUTE CORONARY SYNDROMES Unstable Angina/NSTEMI

- UA/NSTEMI
 - Patent culprit artery, ulcerated plaque and associated thrombus
 - Significant risk of of thrombotic reocclusion

• Unstable Angina = ACS without abnormal levels of serum biomarkers for myocardial necrosis (Ti,Tt,CK-MB)

• **NSTEMI** = ACS *with* positive markers



NSTEMI

- Heterogeneous population
 - Atypical presentation
 - Variable age
 - Medical burden
 - renal insufficiency
 - Perceived difficulty with interpreting biomarkers



INTERPRETATION OF TROPONINS

Troponin I

- High sensitivity and specificity
- Appears within 6 hours of injury
- Requires up to 14 days for clearance
 - Not useful with reinfarction
- Spectrum
 - Higher the troponin, the greater the risk

- False positive:
 - CHF, pericarditis, myocarditis, contusion, cardiomyopathy
 - Shock
 - Renal insufficiency
 - Pulmonary emboli



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STEMI

- Complete thrombotic occlusion of a major epicardial artery

• Presentation:

- Characteristic symptoms of cardiac ischemia
 - More prolonged and severe symptoms
 - Little response to nitroglycerine
- Specific EKG changes on serial EKGs
- Elevation of serum markers for cardiac injury



WHO definition of AMI



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THE ELECTROCARDIOGRAM

- 12 lead EKG
 - Cornerstone of initial evaluation
 - Within 10 minutes of presentation
- Previous EKG tracings
 - Compare
- Serial EKGs
 - Essential



THE ELECTROCARDIOGRAM INFARCT LOCATION

- II, III, AVF

: Inferior

- V1 - V4

: Anteroseptal

- I, aVL, V5-V6

: Lateral

- V1-V2 tall R, ST depression: True posterior



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ACUTE CORONARY SYNDROMES ELECTROCARDIOGRAPHY

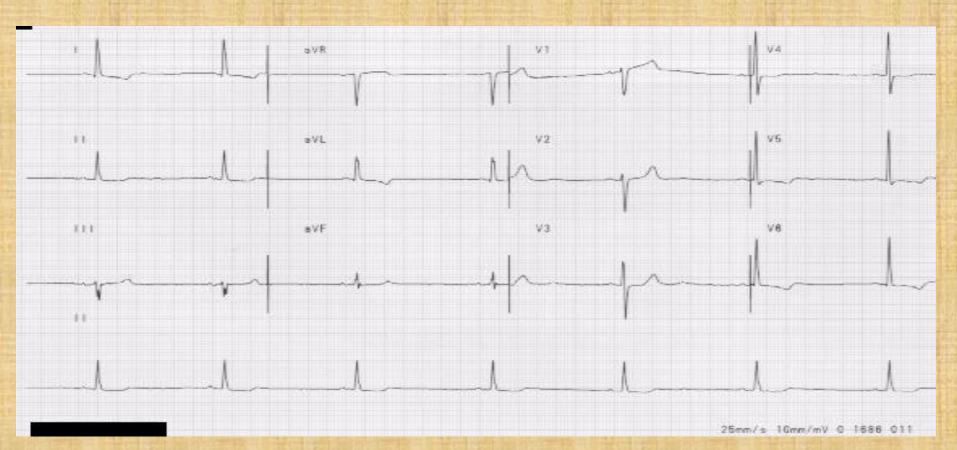
- Ischemia:
 - Mismatch between perfusion and oxygen demand
 - Goal:
 - Reduce oxygen demands and/or Increase perfusion
- EKG Changes:
 - ST and T wave changes
 - ST segment depression
 - T waves
 - flattened, inverted, tall and peaked, symmetrical



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ELECTROCARDIOGRAM ISCHEMIA



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ACUTE CORONARY SYNDROMES ELECTROCARDIOGRAPHY

- Injury:
 - Prolonged ischemia (minutes)
 - Can "salvage" with reperfusion
- EKG changes:
 - ST segment elevation
 - > 1 mm in 2 or more anatomically contiguous leads
 - New left bundle branch block (LBBB)
 - True posterior change

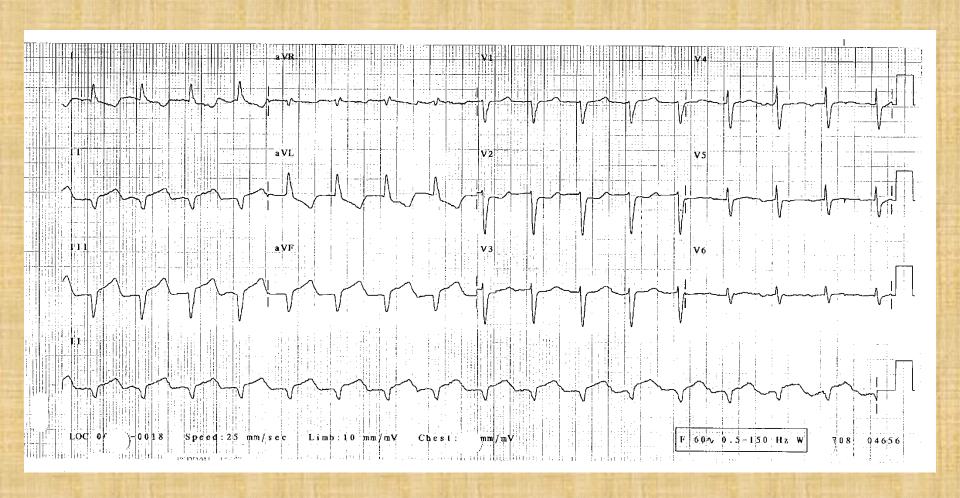


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ELECTROCARDIOGRAM INJURY





ACUTE CORONARY SYNDROMES ELECTROCARDIOGRAPHY

• Infarction:

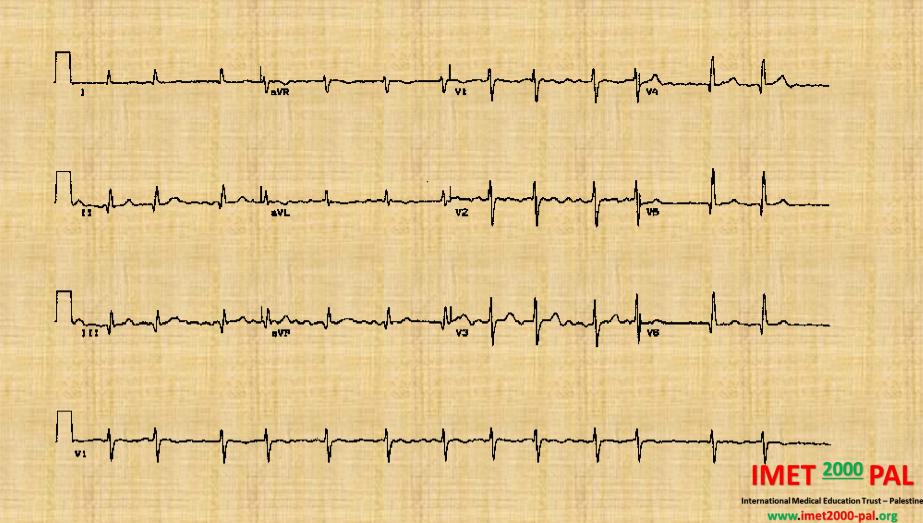
- Myocardial cell necrosis
 - Leaking of intracellular components

EKG Changes:

- Abnormal Q waves
 - >2 hours after symptoms
 - > 1 mm wide
 - Height > 25% R wave



ELECTROCARDIOGRAM INFARCTION



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THE ELECTROCARDIOGRAM WELLEN'S SYNDROME

• Clinical UA with:

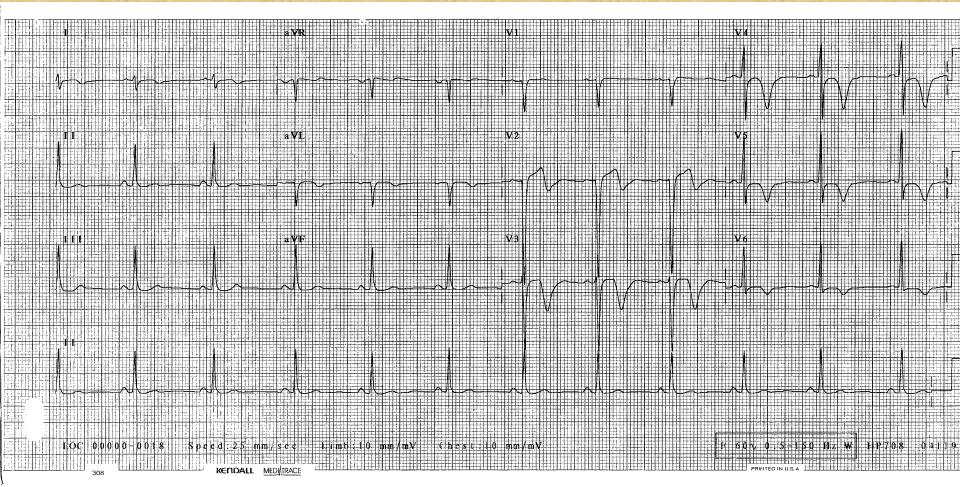
- Inverted or biphasic T-waves in V2 and V3
- T wave changes may also be present in V1, V4-V6
- Changes appear when pain free
- Little to no ST change
- No loss of precordial R waves
- No pathologic Q waves

• Concern:

- Highly specific for LAD lesions
- At risk for extensive AMI or sudden death



THE ELECTROCARDIOGRAM WELLEN'S SYNDROME



CARDIAC RISK FACTORS

TRADITIONAL

NOT PREDICTIVE ACUTE ISCHEMIA

- AGE
- FAMILY HISTORY
- HYPERTENSION
- DYSLIPIDEMIA
- DIABETES MELLITUS
- SMOKING HISTORY



TIMI RISK SCORES

• Thrombolysis In Myocardial Infarction

- Clinical risk algorithms
 - Risk stratification in ACS
- Retrospectively derived/ Prospectively validated

TIMI RISK SCORE FOR UA/NSTEMI

• HISTORICAL POIN	TS
• age >/= 65 y	1
• >/= 3 CAD risk factors	1
• known CAD (stenosis >/= 50%)	1
ASA use in past 7 days	1
• PRESENTATION	
• severe angina = 24 hours</td <td>1</td>	1
elevated cardiac markers	1
• ST deviation >/= 0.5 mm	1

RISK SCORE: /7



TIMI RISK SCORE FOR UA/NSTEMI

RISK OF CARDIAC EVENT IN 14 DAYS

RISK SCORE	0-1	2	3	4	5	6-7
DEATH OR AMI (%)	3	3	5	7	12	19
DEATH, AMI OR PTCA/ CABG	5	8	13	20	26	41
(%)						

Antman et.al. JAMA 2000; 284: 835-42



TIMI RISK SCORE FOR STEMI

• HISTORICAL	POINTS
• age >/= 75 y or 65-74 y	3 or 2
• DM, HTN, Angina	1
• EXAM	
• SBP < 100 mmHg	3
• HR > 100	2
Killip Score II-IV	2
• weight < 67 kg (150lbs)	1
• PRESENTATION	
anterior ST elevation or LBBB	1
• time to Rx > 4 hours	1
RISK SCORE: /14	



KILLIP SCORE

SEVERITY CLASS

I

lla

Ilb

Ш

IV

LV FUNCTION IN AMI

No crackles, no S3

Crackles < 50 % lung fields, no S3

Crackles < 50 % lung fields, S3 present

Crackles > 50 % lung fields, pulmonary edema Cardiogenic Shock



TIMI RISK SCORE FOR STEMI

30 DAY MORTALITY (%)

RISK SCORE 0 1 2 3 4 5 6 7 8 >8

30 DAY 0.8 1.6 2.2 4.4 7.3 12 16 23 27 36 **MORTALITY**

Morrow et.al Circulation; 102:2031-7



AGENTS USED IN ACUTE CORONARY SYNDROMES

- WHY
- WHEN
- HOW
- PRECAUTIONS



THE EVIDENCE

Level of Evidence

Grading

A

B

C

Well designed, randomized, controlled trials OR meta-analyses involving large number of patients

Smaller radomized trials OR reviews of observational, retrospective or nonrandomized trials

Expert consensus, primary nonrandomized OR observational data

THE EVIDENCE

Class I

Class II

lla

IIb

Class III

Evidence or general agreement that a specific procedure or treatmetn is useful and effective

Conflicting evidence or divergence of opinion about the utility or efficacy of a procedure or treatment

Weight of evidence or opinion is in favour of utility-efficacy

Utility-efficacy is less well established by evidence or opinion Evidence or general agreement that a specific procedure or treatment is neither useful n effective and may be harmful

AGENTS USED IN ACUTE CORONAY SYNDROMES OXYGEN

• WHY Level C evidence

- Increase supply to ischemic tissue

WHEN

- Suspect ACS

HOW

- Start with nasal cannula at 4L/min

PRECAUTIONS

- COPD



AGENTS USED IN ACUTE CORONAY SYNDROMES ASPIRIN

Class I, Level A evidence

WHY

- Mortality reduction
- Blocks synthesis of thromboxane A2
 - Inhibits platelet aggregation
 - Relaxes arterial tone

WHEN

- Suspected ACS

• HOW

- 160 mg chewed slowly, then 81-325 mg daily or pr

CONTRAINDICATION

- True allergy
- No GI tract!



AGENTS USED IN ACUTE CORONAY SYNDROMES UNFRACTIONATED HEPARIN

WHY

Class I, Level A evidence + ASA

- Inhibits thrombin > IXa, Xa
- Prevent thrombus formation over ruptured plaque
- Prevent recurrence of thrombosis
- Prevent mural thrombus

WHEN

- UA/NSTEMI
- With tPA
- With PTCA/surgical revascularization

HOW

- IV bolus (60 units/kg iv to maximum 5000 units), then
- Infusion (1000 units/hr)



AGENTS USED IN ACUTE CORONAY SYNDROMES UNFRACTIONATED HEPARIN

PRECAUTIONS

- Active bleeding
- Recent intracranial, intraspinal, eye surgery
- Severe hypertension
- Bleeding disorders



AGENTS USED IN ACUTE CORONAY SYNDROMES LOW MOLECULAR WEIGHT HEPARIN

E.g. Enoxaparin (Lovenox), Dalteparin

Class I, Level A evidence + ASA

• WHY

- Antithrombotic, anti Xa
- Predictable
- Do not require coagulation test monitoring
- Lower incidence of thrombocytopenia
- No platelet activation
- Binds clot bound thrombin

WHEN

Suspected ACS



AGENTS USED IN ACUTE CORONAY SYNDROMES LOW MOLECULAR WEIGHT HEPARIN

HOW

- Subcutaneously

PRECAUTIONS

- Renal insufficiency
- Weight > 150 kg



AGENTS USED IN ACUTE CORONAY SYNDROMES BETA-BLOCKERS (Bb)

E.g. Metoprolol, Bisoprolol, Atenolol, etc.

Level A evidence

• WHY

- Anti-arrhythmic
- Anti-ischemic
- Anti-hypertensive
- Decreased myocardial rupture at one week in STEMI

• WHEN

- Within 12 hours of AMI
- ACS and excess sympathetic activity

HOW

- Intravenous (Metoprolol)
- Oral



AGENTS USED IN ACUTE CORONAY SYNDROMES BETA-BLOCKERS (Bb)

CONTRAINDICATIONS

• ABSOLUTE

- Shock
- Bradycardia
- Hypotension
- Severe asthma
- Acute CHF/pulmonary edema

RELATIVE

- Asthma / severe COPD
- Heart blocks
- Severe PVD
- IDDM
- Extreme age



AGENTS USED IN ACUTE CORONAY SYNDROMES ADP ACTIVATION INHIBITORS

Class I, Level A evidence, if cannot take ASA Class I, Level B evidence, otherwise

E.g. Clopidogrel (Plavix), Ticlodipine

• WHY

Irreversible inhibitor of ADP-receptor mediated platelet aggregation

WHEN

- STEMI, ASA sensitivity
- UA/NSTEMI
- High risk patient characteristics



AGENTS USED IN ACUTE CORONAY SYNDROMES <u>CLOPIDOGREL (Plavix)</u>

HOW

- 300mg po load, then 75 mg po qd

PRECAUTIONS

- Allergy
- Thrombocytopenia
- High risk GI bleed
- (CV surgical procedure anticipated)
 - Stop minimum 5 days prior



AGENTS USED IN ACUTE CORONAY SYNDROMES GPIIb/IIIa INHIBITORS

Class I Level A evidence

in patients with planned PCI in 12-24 hours, ASA and heparin Class IIa, Level A evidence

In high risk patients without planned PCI, ASA and heparin

E.g. Eptifibatide (Integrelin), Abciximab Tirofiban

- WHY
- Competitive inhibition of fibrinogen binding between platelets
- WHEN
 - ACS, refractory symptoms
 - Urgent PCI
 - With ASA and UFH +/- PCI
- HOW
 - Bolus: 180 mcg/kg iv (maximum weight 120kg)
 - Infusion: 2 mcg/kg/min (half with renal insufficiency)



AGENTS USED IN ACUTE CORONAY SYNDROMES EPTIFIBATIDE (Integrelin)

No established role with thrombolysis or LMWH

PRECAUTIONS

- Active bleeding within 30 days
- Stroke or head injury within 30 days
- Bleeding diathesis
- INR > 2.0
- Platelets < 100,000
- Major surgery or trauma within 6 weeks
- Uncontrolled HTN (SBP > 200, DBP > 110)
- Hypersensitivity



AGENTS USED IN ACUTE CORONAY SYNDROMES NITROGLYCERINE

Does not reduce mortality

Level C evidence

• WHY

- Decreases ischemic pain
 - Venodilation/decreased preload
 - Dilates coronary arteries (eliminates vasospasm)
 - Increases coronary collateral flow

WHEN

- Ischemic chest pain
- For 24-48 hr after AMI
 - Recurrent pain
 - Hypertension
 - CHF



AGENTS USED IN ACUTE CORONAY SYNDROMES NITROGLYCERINE

HOW

- Sublingual
 - Tablets: 0.3mg q 5 minutes
 - Spray: 0.4 mg q 5 minutes
- IV Infusion
 - Start 10-20 mcg/min
 - Increase by 5-10 mcg/min q5-10 minutes

PRECAUTIONS

- Avoid hypotension
- Extreme caution with RV infarction
- Interaction with sildenafil (Viagra)



AGENTS USED IN ACUTE CORONAY SYNDROMES MORPHINE

• WHY

Level C evidence

- Reduce ischemic pain
- Reduce anxiety
- Reduce extension
 - Reduction of sympathetic tone and oxygen demands

WHEN

- Ongoing pain of infarction
- Acute pulmonary edema
- SBP > 90 mmHg



AGENTS USED IN ACUTE CORONAY SYNDROMES MORPHINE

HOW

- Small increments IV
 - 1 3 mg prn, to eliminate pain

PRECAUTIONS

- Allergy
- Nausea and vomiting
- Hypotension
- Respiratory depression



AGENTS USED IN ACUTE CORONAY SYNDROMES ACE-INHIBITORS

E.g. Ramipril, Enalapril, Captopril

• WHY

Level A evidence

(Anterior infarct, EF< 40%)

- Reduce
 - Left ventricular dysfunction and dilation
- Remodeling
- Decrease afterload and preload
- Reduction in mortality

• WHEN

- Within 24 hours AMI
- Suspected or known CAD



AGENTS USED IN ACUTE CORONAY SYNDROMES ACE-INHIBITORS

• HOW

- Oral

PRECAUTIONS

- Pregnancy
- Symptomatic hypotension
- Bilateral renal artery stenosis
- Angioedema
- Allergy



Very High Risk (>15% 30 day AMI/Mortality)

PRESENTATION

- Prolonged/recurrent pain
- >2mm ST depression
- Positive cardiac markers
- >1mm transient ST elevation
- Hemodynamic instability
- Refractory ischemia

• TREATMENT

- ASA
- Clopidogrel
- Heparin
- Eptifibatide
- Urgent angiography
- Urgent revascularization

Can. J Cardiol. 18(11), 2002



coronary

High Risk (8-15% 30 day AMI/Mortality)

PRESENTATION

- Rest pain> 20 minutes
- >2mm ST depression
- Deep T wave inversion (>5mm)
- >2mm T wave inversion in >5 leads
- Positive cardiac markers

TREATMENT

- ASA
- Clopidogrel
- Heparin
- +/- Eptifibatide (in consultation with cardiology)
- Early coronary angiography

Can. J Cardiol. 18(11), 2002



Intermediate Risk (3-8% 30 day AMI/Mortality)

PRESENTATION

- Rest pain
- New onset or crescendo pain
- Nonspecific or normal EKG
- Normal or borderline positive cardiac markers
- Increased baseline risk (DM, previous AMI, CABG, recent PCI)

TREATMENT

- ASA
- +/-Clopidogrel
- Heparin
- EST or myocardial perfusion scan
- Coronary angiography

Can. J Cardiol. 18(11), 2002



Low Risk (<3% 30 day AMI/Mortality)

PRESENTATION

- Single short duration pain
- New onset or crescendo pain
- Nonspecific/normal EKG X 2
- Normal cardiac markers X 2
- No high risk features

• TREATMENT

- ASA
- EST within 48 hours

Can. J Cardiol. 18(11), 2002



ACUTE CORONAY SYNDROMES PERCUTANEOUS TRANSLUMUNAL ANGIOPLASTY

Level A evidence

 "early angiography and directed revascularization (within 7 days), when combined with optimal medical pretreatment, is the preferred strategy for patients with and ACS who present with signs of ischemia on EKG or

raised biochemical markers at admission"

FRISC II



• Urgent reperfusion:

- FIBRINOLYSIS



- PERCUTANEOUS CORONARY INTERVENTION



AGENTS USED IN ACUTE CORONAY SYNDROMES FIBRINOLYTICS

WHY

Level A evidence

- Plasminogen activators
- Degrade the occlusive thrombus

WHEN

- WITHIN 30 MINUTES OF PRESENTATION
- Ischemic type chest pain
- EKG compatible
 - ST elevation > 2mm in 2+ contiguous leads
 - new LBBB
 - true posterior infarct
- Pain </= 6 hours (< 12 hours)</pre>
- No contraindications



CONTRAINDICATIONS TO THROMBOLYSIS

CONTRAINDICATIONS

• ABSOLUTE

- Lack of clear indications
- Active internal bleeding
- Recent trauma, major surgery, internal bleeding (within 2weeks)
- Suspected aortic dissection
- Pericarditis
- Previous hemorrhagic stroke
- Other strokes within one year
- Known intracranial neoplasm



CONTRAINDICATIONS TO THROMBOLYSIS

• RELATIVE:

- Recent trauma, major surgery, internal bleeding (2-4 weeks)
- Severe uncontrolled hypertension (> 180/110 mmHg)
- Current use of anticoagulants (INR > 2-3)
- Intracerebral pathology (other than stroke)
- Known bleeding diathesis
- Active peptic ulcer disease
- Pregnancy
- Noncompressible vascular punctures
- Known hypersensitivity to agent
- *Age* > 75 *years*
- Prolonged (> 10 minutes) traumatic CPR





AGENTS USED IN ACUTE CORONAY SYNDROMES FIBRINOLYTICS

HOW

- Streptokinase
 - » Derived from beta-hemolytic Streptococcus cultures
 - » Smaller infarcts, elderly, underweight
 - » 1.5 million units over 1 hour
- Tissue Plasminogen Activator tPA
 - » Naturally occurring enzyme
 - » Better with large infarct
 - » Highest incidence of ICH
 - » 15 mg IV bolus
 - » 0.75 mg/kg over next 30 min (50 mg)
 - » 0.50 mg/kg over next 60 min (35 mg/FT 20

AGENTS USED IN ACUTE CORONAY SYNDROMES FIBRINOLYTICS

- Tenecteplase (TNK)

- Single bolus
- Weight based dosing

- Reteplase

- Genetically modified t-PA
- Not weight based
- Two boluses of 10 units, 30 minutes apart

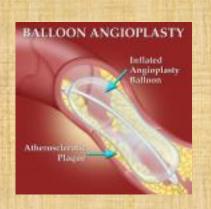


ACUTE CORONAY SYNDROMES PERCUTANEOUS TRANSLUMUNAL ANGIOPLASTY

Emergent PTCA:

Level A/B evidence

- Consider:
 - Ongoing symptoms of >12 hour duration
 - Contraindications to thrombolysis
 - Failure of thrombolytics
 - Cardiogenic shock and AMI
 - Previous CABG
 - "Stuttering Infarction"
 - Access to lab



ACUTE CORONARY SYNDROMES

PUTTING IT ALL TOGETHER



CASE ONE

- Frail 67 year old hypertensive male
- 8/10 substernal chest pain
- Radiation down left arm, into jaw
- Diaphoresis, tachypnea, nausea
- Onset within past four hours
- No relief with nitro
- T 37.1 C HR 112/min BP 150/100 RR 22/min

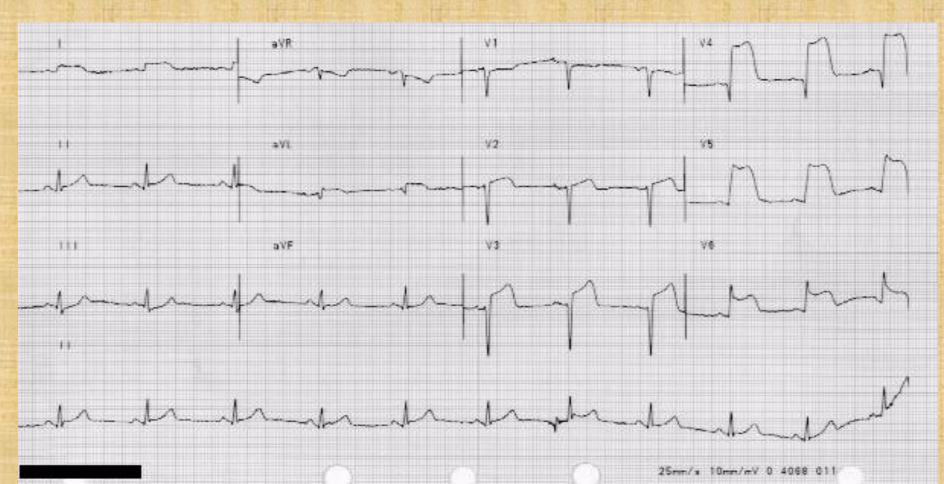
CASE ONE

Immediate Assessment:

- IV access Oxygen Monitors
- EKG
- Targeted history and exam
- CXR
- Eligibility for thrombolysis/PCI
- Labs



CASE ONE ELECTROCARDIOGRAM



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CASE ONE

Risk stratify:

- STEMI, TIMI score >8 (VERY HIGH RISK)

• Immediate Treatment:

- ASA 160 mg po
- Oxygen
- +/- nitro sl
- Metoprolol
- Heparin
- Emergent revascularization strategy



CASE ONE

Adjunctive Treatment:

- Clopidogrel po
- Nitroglycerine iv
- Morphine iv
- Consider IIb/IIIa agents if primary PCI

- 65 year old diabetic female
- Retrosternal/epigastric pressure with no radiation
- Occurs at rest, duration </= 15minutes
- Associated with nausea and diaphoresis
- Pain free currently
- Onset 1/12 ago but increasing 4/7

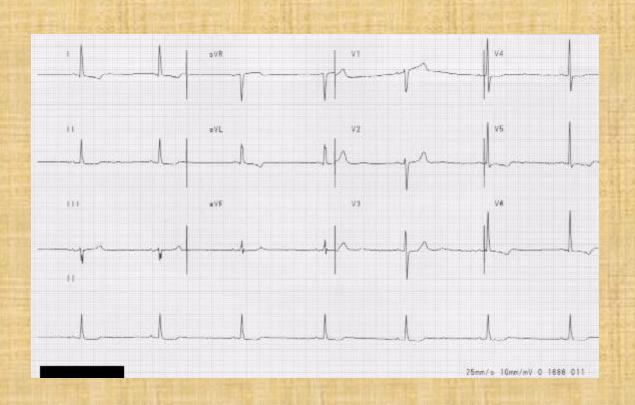


Immediate Assessment:

- IV access Oxygen Monitors
- EKG
- Targeted history and exam
 - smoker, dyslipidemic, hypertension, proteinuria
 - on ASA, HCTZ, metformin, glyburide, celexa
 - normal cardiac exam
- CXR
- Labs



CASE TWO ELECTROCARDIOGRAM



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Risk stratify:

- UA/NSTEMI, TIMI score >4 (INTERMEDIATE RISK)

• Immediate Treatment:

- ASA 160 mg po
- Heparin (LMWH > UFH)
- +/- Clopidogrel
- Coronary angiogram



• Adjunctive Treatment:

- Beta Blockers
- ACE Inhibitors
- -+/- Nitrates

CASE THREE

- 37 year old male complains of a retrosternal dull ache for 3 hours
- No radiation of pain
- No associated symptoms
- Smoker, significant family history



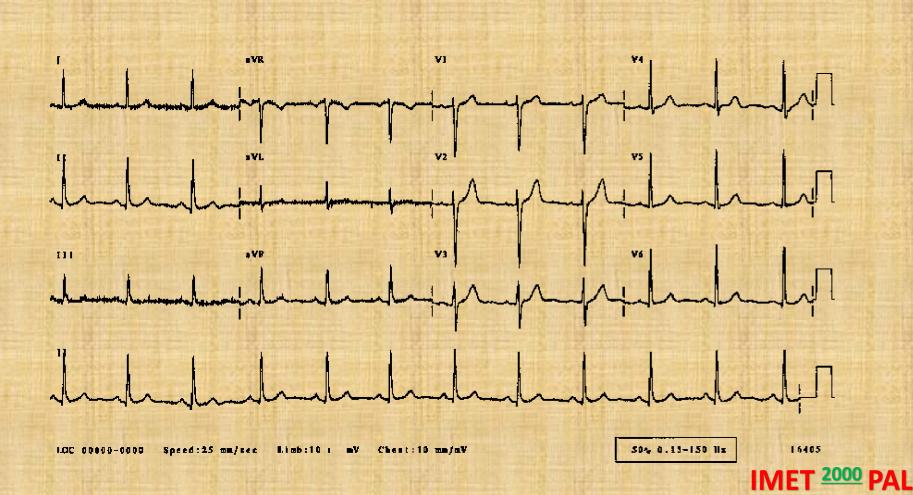
CASE THREE

Immediate Assessment:

- IV access Oxygen Monitors
- EKG
- Targeted history and exam
- CXR
- Labs



CASE THREE ELECTROCARDIOGRAM



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CASE THREE

Risk stratify:

- UA/NSTEMI, TIMI score 1 (LOW RISK)

• Immediate Treatment:

- ASA 160 mg po
- Monitor
- Serial EKG and enzymes (X2)
- Exercise Stress Test



SUMMARY

- Suspected Ischemic Chest Pain Needs:
 - Urgent +/- serial EKGs
 - Monitoring
 - Cardiac Biomarkers
 - Targeted History and Physical Examination to:
 - Define ACS
 - Risk stratify (e.g. TIMI Scores)
 - Appropriate management
 - Antiplatelet, antithrombotic, anti-ischemic, +/- revasculariization

SUMMARY

• STEMI

- complete thrombotic occlusion of a major epicardial artery
- GOAL = establish patency and preserve myocardial function

• UA/NSTEMI

- partially occluded culprit artery, or fully occluded with collaterals
- ulcerated plaque and associated thrombus
- significant risk of of thrombotic reocclusion
- THERAPY = antithrombotic and antiplatelet

SUMMARY THE ELECTROCARDIOGRAM

1. ST segment elevation 2mm (2 contiguous leads), new LBBB, true posterior ischemia

STEMI

EMERGENT REPERFUSION

2. ST depression >1mm, marked symmetrical T wave inversions >2 mm or Wellen's pattern, dynamic ST-T changes with pain

UA/NSTEMI LIKELY

MEDICAL MANAGEMENT +/- URGENT IMAGING

3. Non-diagnostic or normal ECG

ACS LESS LIKELY

RISK STRATIFY



SUMMARY

- Goal of ACS Management:
 - REDUCE PATIENT SYMPTOMS
 - REDUCE MORTALITY
 - LIMIT MYOCARDIAL DAMAGE
 - PRESERVE LV FUNCTION

"TIME IS MUSCLE"



Thank You for Your Attention

IMET 2000 PAL

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Raed Abu Sham'a, M.D.