Diagnosis and Management of Chest Pain and Acute Coronary Syndrome (ACS)

Unstable Angina/NSTEMI

1

Case # 1 Ms. S.A.

- 44 female with new onset RSCP
- When did pain begin?
  - 4/7 days
- What were you doing?-swimming
- How long did it last?-15minutes
- Recur?-yesterday, off and on for 3 hours
- Where?-R/S radiating to jaw
- What did it feel like?-tenseness,tightness

2

Case # 1 Ms. S.A.

- Aggravating factors?-walking
- Relieving factors?-rest/NTG at Urgi-centre
- Total duration?-3 hours
- Any similar pain before?-no
- Risks:
  - HPL, smoking 15 pack years, FH++++

3

Case # 1 Ms. S.A.

- Physical exam
  - S4 otherwise normal
- ECG-evolving inferior T changes
- CK 120 TnT 0.12
- Admit to telemetry
- Rx:
  - ASA,Plavix,Lovenox
  - β-blocker, long acting nitrate

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Case # 1 Ms. S.A.

- Cardiac catheterization:
  - LCA-small vessels, no discrete lesions
  - RCA-70% proximal & 95% mid stenosis, 3 non significant mid plaque ulcerations
- PCI & stenting
  - Proximal and mid lesions to 0%
  - More distal plaque ulcerations not touched

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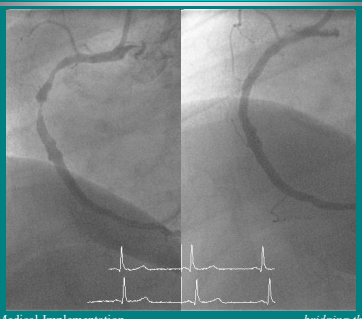
Ms. S.A. Ad Hoc PCI



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Initial Approach

1. Is the presenting symptom:
   - typical angina?
   - atypical angina?
   - non-anginal chest pain?
2. How do you define each of the above?
3. What is this patient's clinical likelihood of CAD?
4. Does the patient require immediate therapy?
5. What investigations are indicated? When?
6. Does the patient require coronary angiography?
7. Does the patient require revascularization?

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Chest Pain Differential

- Angina
- Pericarditis
- Pleurisy
- Pulmonary Embolism
- Aortic dissection
- Esophageal reflux/spasm
- Peptic/biliary/colonic referred pain
- Chest wall pain
- Neurogenic pain
  - C disc disease
  - Thoracic outlet
  - shingles

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Visceral chest pain

Cardiac until proven otherwise!!!!!

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Categorize the Chest Pain

Typical angina
1. Retrosternal component
2. Brought on by stress or exercise
3. Relieved promptly by rest or NTG

Atypical angina
- 2 of above 3 criteria

Non-anginal chest pain
- 1 of above 3 criteria

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Prevalence of CAD (%) in Symptomatic Patients According to Age and Sex

	Typical angina		Atypical angina		Non anginal chest pain	
AGE	Men	Women	Men	Women	Men	Women
30-39	69.7	25.8	21.8	4.2	5.2	0.8
40-49	87.3	55.2	46.1	13.3	14.1	2.8
50-59	92.0	79.4	58.9	32.4	21.5	8.4
60-69	94.3	90.6	90.6	54.6	28.1	18.6
	3 of 3 criteria		2 of 3 criteria		1 of 3 criteria	

1) Retrosternal discomfort.2) Provoked by exercise or stress.3) Relieved by rest or NTG

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Define the Chest Pain Syndrome

- Chronic stable angina
  - Grade CCS severity
- Unstable angina
  - Define syndrome
  - Assess short term risk of death or MI
- Non ST-elevation MI
- ST-elevation MI

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Unstable Anginal Syndromes

- New onset angina (1 month)
- Crescendo angina
  - Increased frequency,severity or duration
- Acute coronary syndrome (ACS)
  - Ischaemic chest pain > 20 minutes
- Post infarction angina
- Prinzmetal's (variant) angina

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ACS Nomenclature

- ACI (Acute coronary insufficiency)
  - Prolonged ischaemic chest pain > 20 min.
  - ± ST changes
  - Negative enzyme/biomarkers
- Non STEMI
  - Prolonged ischaemic chest pain > 20 min.
  - ± ST changes
  - Elevated enzyme/biomarkers

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ACS Nomenclature

- ST Elevation MI:
  - Prolonged R/S chest pain > 20 minutes with
  - Persisting ST elevation despite NTG
    - At least 1 mm in 2 adjacent limb leads
    - At least 2 mm in 2 adjacent precordial leads
    - LBBB (for purpose of thrombolysis)
  - Evolution of Q waves (excluding LBBB scenario)
  - Enzyme or bio-marker elevation

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Categorize the Severity of Angina

CCS Classification	
Class 0	asymptomatic
Class I	on strenuous activity
Class II	on moderate activity > 2 blocks or 2 flights of stairs
Class III	on mild activity < 2 blocks or 2 flights of stairs
Class IV	rest or minimal activity

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Categorize the Severity of Angina

CCS Classification	
Class IV A	Patient admitted to hospital and becomes relatively asymptomatic with aggressive medical therapy
Class IV B	Patient admitted to hospital and continues to experience angina on maximal medical therapy and cannot be safely discharged home, but does not require IV nitroglycerin.
Class IV C	Patient admitted to hospital and maximal medical therapy, including IV nitroglycerin, fails to control symptoms or there is hemodynamic instability.
Class IV D	Cardiogenic Shock

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Non ST Elevation MI

- Definition:
  - Prolonged ischaemic chest pain
  - ST-T changes
  - Positive serum markers

http://www.cybersessions.com/aventis/

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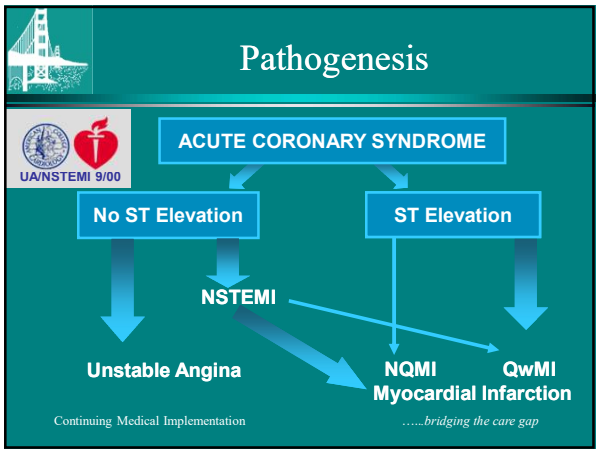
Pathogenesis
UA/NSTEMI 9/00
ACUTE CORONARY SYNDROME
No ST Elevation
ST Elevation
NSTEMI
Unstable Angina
NQMI
QwMI
Myocardial Infarction
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…..bridging the care gap

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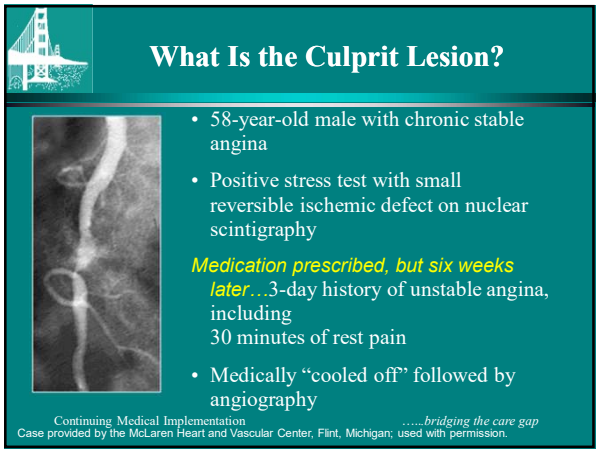
What Is the Culprit Lesion?
• 58-year-old male with chronic stable angina
• Positive stress test with small reversible ischemic defect on nuclear scintigraphy
Medication prescribed, but six weeks later…3-day history of unstable angina, including
30 minutes of rest pain
• Medically "cooled off" followed by angiography
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…..bridging the care gap
Case provided by the McLaren Heart and Vascular Center, Flint, Michigan; used with permission.

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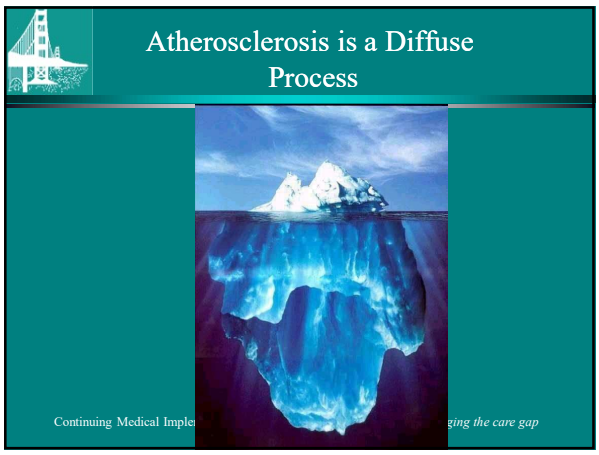
Atherosclerosis is a Diffuse Process
Continuing Medical Imple
ging the care gap

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Glagov's Model

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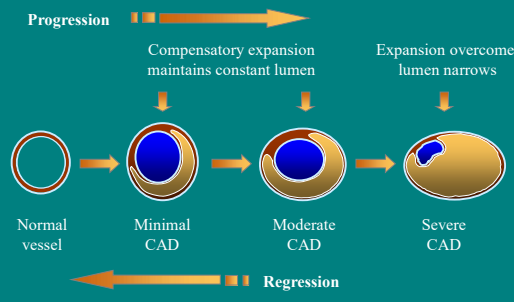
Glagov's Coronary Remodeling Hypothesis



Adapted from Glagov et al. *N Engl J Med* 1987; 316:1371-1375.

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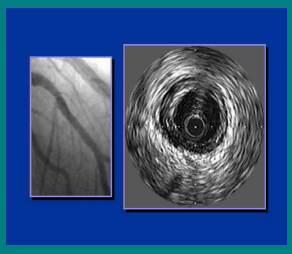
IVUS versus Angiography



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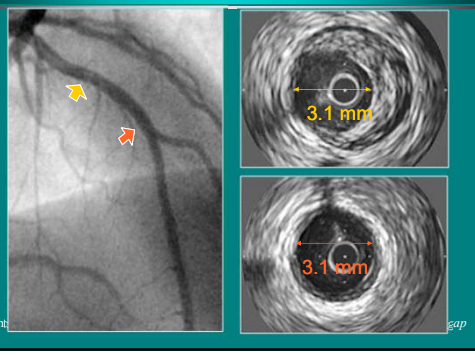
Angiography Fails to Depict Coronary Arterial Remodeling



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Pathogenesis

- Plaque rupture or erosion
- Thrombosis with/without occlusion
- Necrosis contingent on
  - Severity of plaque rupture
  - Duration of ischaemia
  - Lability of occlusive thrombus
  - Adequacy of collaterals
  - Vasoconstriction
  - Downstream platelet/fibrin emboli

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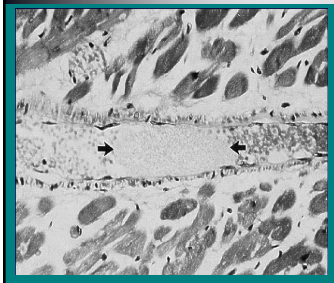
Unstable Angina: Platelet Plugging of the Capillaries



"The results support the view that platelet aggregates in the myocardium represent an embolic phenomenon and are a potential cause of unstable angina. The association of myocardial necrosis with such emboli could precipitate sudden death from ventricular fibrillation."

From: **Intramyocardial platelet aggregation in patients with unstable angina suffering sudden ischemic cardiac death Davies MJ, *Circulation* 1985**

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Plaque Transition to Acute Coronary Syndrome

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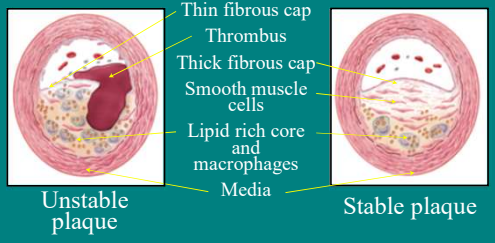
Pathophysiology of Stable and Unstable Plaques



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Unstable Angina-Triggers Systemic factors

- Hyper-coagulable state
- Increased vascular resistance
- Coronary spasm
- Increased cortisol & catecholamines
- Vasoconstriction
- Increased arterial pressure
- Circadian variation

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Unstable Angina Triggering Situations

- Awakening
- Excessive physical exertion
- Mental stress
- Anger
- Cigarette smoking
- Coffee & alcohol consumption
- Sexual activity

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Non ST Elevation MI

Ruptured Plaque

90% of acute MIs are caused by thrombus formation from rupture of unstable plaques

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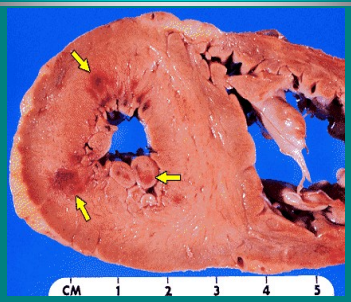
Sub-endocardial MI



Extensive myocardial infarction (arrows)

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Occlusive Thrombus

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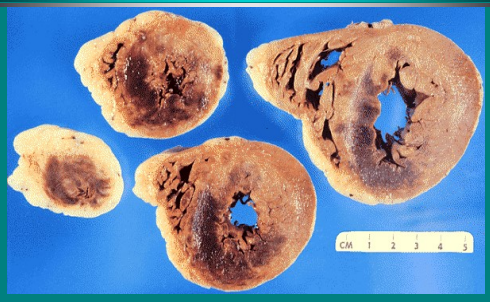
Transmural Infarction



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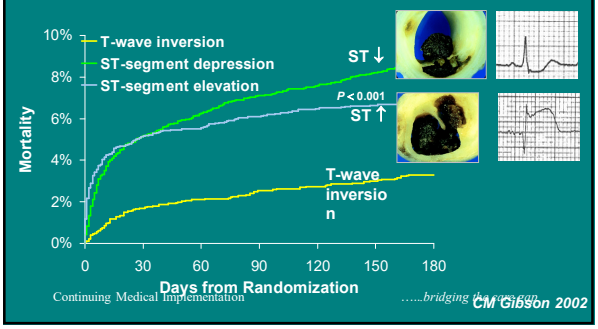
GUSTO 2B: ST Depression
A High-Risk Patient Population



Continuing Medical Implementation ……bridging the care gap CM Gibson 2002

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Risk Stratification of ACS/NSTEMI

- Contingent on
  - Unstable angina syndrome
  - ST/T changes
  - Elevated cardiac enzymes or positive biomarkers
  - Extent of ischaemia
  - Recurrent ischaemia

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Low Risk	Intermediate Risk	High Risk	Very High Risk
Non ST ↑ ACS 30 Day Death/MI Risk			
< 3 %	3-8 %	8-15 %	>15%
No higher risk features •Single short duration (<10 min.) rest pain •Crescendo angina/New onset angina (Mod severity) 6 Hour Observation •ECG X 2 normal, unchanged or non-specific ST Δ's •Negative biomarkers X 2	Rest pain < 20 min. New onset/ Crescendo angina (Low threshold severity) •ECG non-specific abnormalities or normal •Biomarkers normal or borderline ↑ Increased baseline risk •DM •Previous CABG/MI •Recent PCI	Rest pain > 20 min. •ECG ST depression < 2mm •Deep T inversion (e.g. > 5 mm) •T inversion > 2 mm –Especially in > 5 leads •Isolated biomarker clearly +ve	Prolonged recurrent pains •ST depression < 2mm With ↑ CK-MB or Tn •ST depression > 2mm –Multiple leads –With pain •Transient ST ↑ > 1 mm •Hemodynamic instability –↓ BP/CHF Refractory ischaemia with ST shift

D Fitchett, SG Goodman M Gupta, A Langer. Can J Card 2002; 18 (11):1179-1190.

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TIMI Risk Score for UA/NSTEMI

TIMI RISK SCORE for UA/NSTEMI

HISTORICAL	POINTS
Age ≥ 65	1
≥ 3 CAD risk factors (FHx, HTN, ↑ chol, DM, active smoker)	1
Known CAD (stenosis ≥ 50%)	1
ASA use in past 7 days	1
PRESENTATION	
Recent (≤24H) severe angina	1
↑ cardiac markers	1
ST deviation ≥ 0.5 mm	1
RISK SCORE = Total Points (0 - 7)	

RISK OF CARDIAC EVENTS (%) BY 14 DAYS IN TIMI 11B*

RISK SCORE	DEATH OR MI	DEATH, MI OR URGENT REVASC
0/1	3	5
2	3	8
3	5	13
4	7	20
5	12	26
6/7	19	41

*Entry criteria/UA or NSTEMI defined as ischemic pain at rest within past 24H, with evidence of CAD (ST segment deviation or +marker)

For more info go to www.timi.org

Antman et al JAMA 2000; 284: 835 - 842

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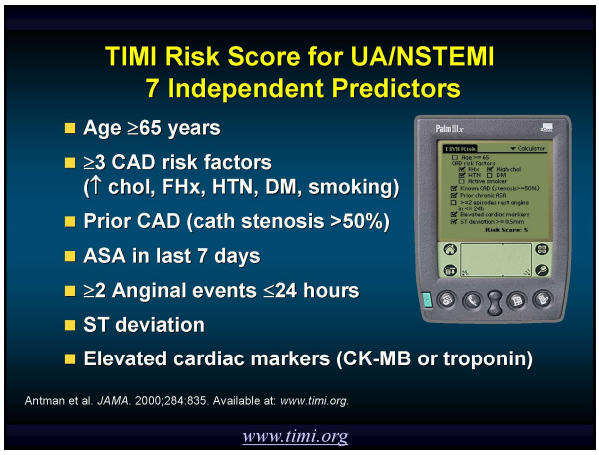
TIMI Risk Score for UA/NSTEMI
7 Independent Predictors
Age ≥65 years
≥3 CAD risk factors
(↑ chol, FHx, HTN, DM, smoking)
Prior CAD (cath stenosis >50%)
ASA in last 7 days
≥2 Anginal events ≤24 hours
ST deviation
Elevated cardiac markers (CK-MB or troponin)
Antman et al. JAMA. 2000;284:835. Available at: www.timi.org.
www.timi.org

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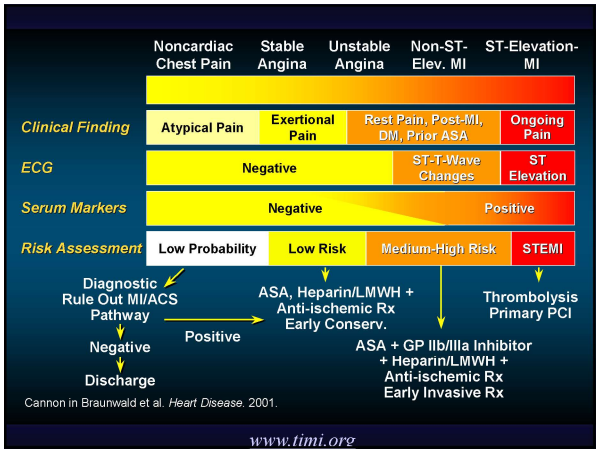
Noncardiac Chest Pain
Stable Angina
Unstable Angina
Non-ST-Elev. MI
ST-Elevation-MI
Clinical Finding
Atypical Pain
Exertional Pain
Rest Pain, Post-MI, DM, Prior ASA
Ongoing Pain
ECG
Negative
ST-T-Wave Changes
ST Elevation
Serum Markers
Negative
Positive
Risk Assessment
Low Probability
Low Risk
Medium-High Risk
STEMI
Diagnostic Rule Out MI/ACS Pathway
Negative
Discharge
Positive
ASA, Heparin/LMWH + Anti-ischemic Rx Early Conserv.
ASA + GP IIb/IIIa Inhibitor + Heparin/LMWH + Anti-ischemic Rx Early Invasive Rx
Thrombolysis Primary PCI
Cannon in Braunwald et al. Heart Disease. 2001.
www.timi.org

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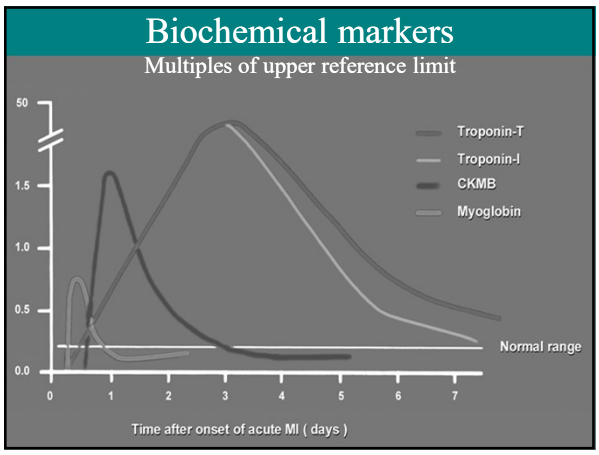
Biochemical markers
Multiples of upper reference limit
50
1.5
1.0
0.5
0.0
0
1
2
3
4
5
6
7
Troponin-T
Troponin-I
CKMB
Myoglobin
Normal range
Time after onset of acute MI (days)

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BIOCHEMICAL CARDIAC MARKERS IN PTS WITH SUSPECTED ACS WITHOUT STE

Advantages

CK-MB

1. Rapid, cost-efficient, accurate assays
2. Ability to detect early reinfarction

Myoglobin

1. High sensitivity
2. Useful in early detection of MI
3. Detection of reperfusion
4. Most useful in ruling out MI

Troponins

1. Powerful for stratification
2. Greater sensitivity and specificity than CK-MB
3. Detection of recent MI up to 2 weeks after onset
4. Useful for selection of therapy
5. Detection of reperfusion

UA/NSTEMI 9/00

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BIOCHEMICAL CARDIAC MARKERS IN PTS WITH SUSPECTED ACS WITHOUT STE

Disadvantages

CK-MB

1. Lack of specificity with skeletal muscle disease/injury
2. Low sensitivity during early MI (<6 h) or late (>36 h) after symptom onset and for minor myocardial damage

Myoglobin

1. Very low specificity with skeletal muscle injury or disease
2. Rapid return to normal

Troponins

1. Low sensitivity in early phase of MI (<6 h after symptom onset)
2. Limited ability to detect late minor reinfarction

UA/NSTEMI 9/00

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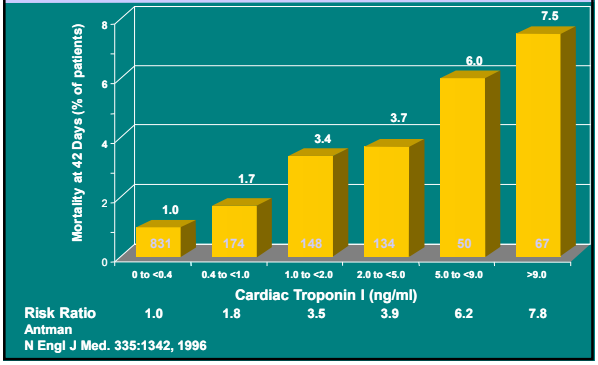
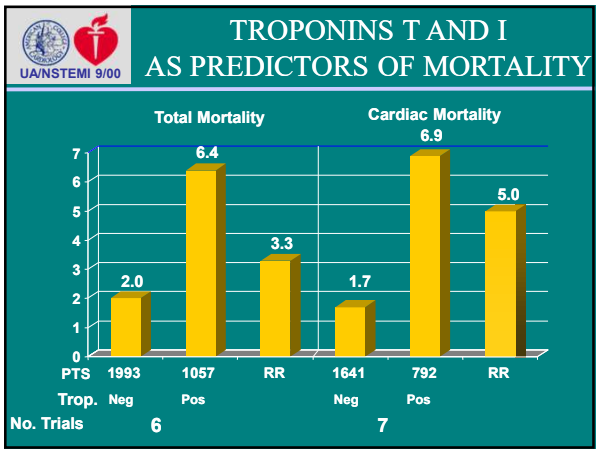
TROPONIN I LEVELS PREDICT RISK OF MORTALITY IN UA/NSTEMI

UA/NSTEMI 9/00



Antman
N Engl J Med. 335:1342, 1996

45


UA/NSTEMI 9/00
TROPONINS T AND I
AS PREDICTORS OF MORTALITY
Total Mortality
Cardiac Mortality
7
6
5
4
3
2
1
0
6.4
2.0
3.3
6.9
1.7
5.0
PTS 1993 1057 RR 1641 792 RR
Trop. Neg Pos Neg Pos
No. Trials 6 7

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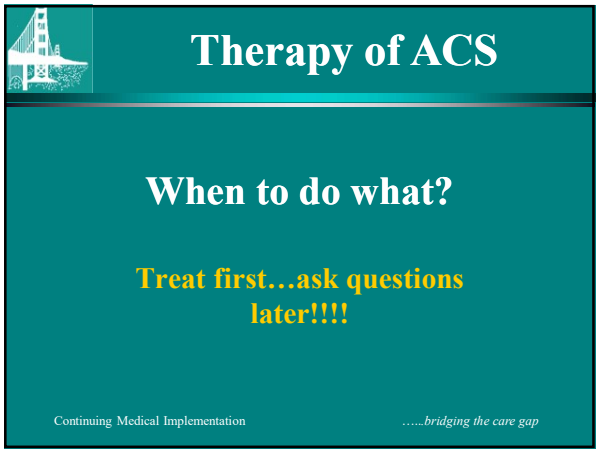
Therapy of ACS
When to do what?
Treat first…ask questions later!!!!
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…..bridging the care gap

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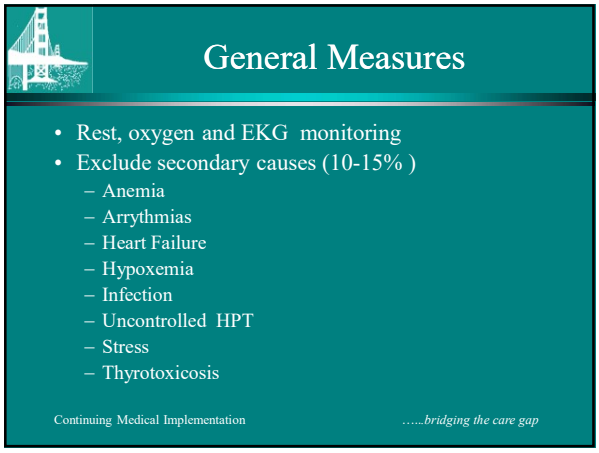
General Measures
• Rest, oxygen and EKG monitoring
• Exclude secondary causes (10-15%)
– Anemia
– Arrythmias
– Heart Failure
– Hypoxemia
– Infection
– Uncontrolled HPT
– Stress
– Thyrotoxicosis
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Unstable Angina/ACS Therapeutic Goals-1

- Prevent re-thrombosis & prevent downstream embolization
  - Anti-platelet therapy
    - ASA (65-75% ↓ early events;50%↓ death/MI 2-24 months)
    - Clopidogrel 300-600 mg → 75 mg OD
    - Glycoprotein IIB/IIIA inhibitors
  - Anti-coagulant therapy (↓ death MI additional 40%)
    - UFH or LMWH

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Unstable Angina/ACS Therapeutic Goals-1

- Control ischaemia
  - β-blockers
  - Nitrates
  - CCB's
- Relieve Obstruction
  - Cardiac cath
  - PCI
  - CABG

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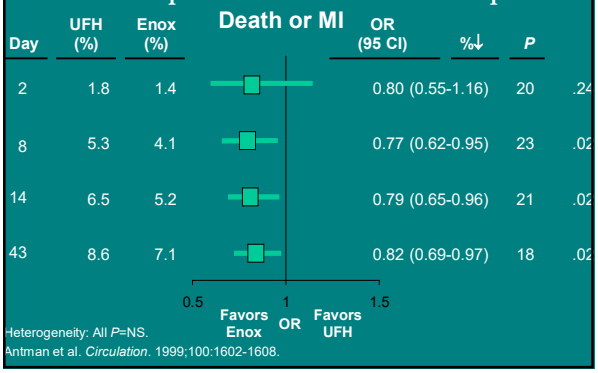
TIMI 11B/ESSENCE Meta-analysis: Enoxaparin vs Unfractionated Heparin



Heterogeneity: All *P*=NS.
Antman et al. *Circulation*. 1999;100:1602-1608.

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CURE Trial

CURE

Clopidogrel in Unstable Angina
to Prevent Recurrent Ischemic Events

Continuing Medical Implementation ……*bridging the care gap*

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Severity of Angina Determines the Therapy

CCS Angina Class	Definition: Angina with	Number of medications	Choice of Rx
I	Strenuous activity	**1**	ASA[1] + NTG prn
II	Moderate activity • > 2 blocks or 2 flights of stairs	**2**	ASA[1] + β-blocker or Rate limiting CCB[2,3]
III	Mild activity • < 2 blocks or 2 flights of stairs	**3**	ASA[1] + β-blocker or Rate limiting CCB[2,3] + Long acting nitrate
IV A	Rest or minimal activity • Patient admitted to hospital and becomes relatively asymptomatic with aggressive medical therapy	**4**	ASA[1] + β-blocker + CCB[3] (do not combine verapamil and β-blocker) + Long acting nitrate
IV B	Rest or minimal activity. • Patient admitted to hospital and continues to experience angina on maximal medical therapy and cannot be safely discharged home, but does not require IV nitroglycerin.	• For prolonged rest pain > 20 minutes Rx heparin LMWH > UFH Enoxaparin (Lovenox®) (ESSENCE / TIMI 11B / TIMI 11B-ESSENCE Metanalysis) • Add clopidogrel 300 mg stat 75 mg OD (CURE) where non-interventional approach is standard of care or early angiography not readily available. • For recurrent pain > 24 hours consider angiography • With planned angiography/PCI within 24-48 hours consider adding glycoprotein IIb/IIIa inhibitor/re-evaluate clopidogrel • tirofiban (Aggrastat®) ○ TACTICS-TIMI 18 ○ PRISM-PLUS • eptifibitide (Integrilin®) -PURSUIT • With ST elevation consider for thrombolytic protocol	
IV C	Rest or minimal activity. • Patient admitted to hospital and maximal medical therapy, including IV nitroglycerin, fails to control symptoms or there is hemodynamic instability.		
IV D	Cardiogenic Shock	Inotropic Agents IABP Angiography Revascularization	

[1]Consider clopidogrel 75mg OD with recent MI, CVA, PVD in ASA intolerant or allergic patients (CAPRIE).
[2]Rate limiting CCB(calcium channel blocker): diltiazem or verapamil (**do not combine verapamil and β-blocker**)
[3]For patients with chronotropic incompetence use long acting DHP- CCB (amlodipine, felodipine or nifedipine)

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Unstable Angina -Therapy

1. ASA-always!!!
2. Heparin-if rest component or ACI!! Lovenox > UFH
3. Add Clopidogrel (Plavix) for Troponin +/dynamic ST changes
4. Lytics-NO!!!(TIMI IIIB)
5. Add IIB/IIIA inhibitors if planned cath/PCI within 24-48 hours or transient ST elevation
6. Beta-Blockers-yes!!!exertional component
7. Calcium blockers-rest pain!!rate limiting CaB
8. Nitrates-yes!!!multiple routes(IV >rapidity)
9. IABP-Tertiary centre
10. Cardiac catheterization-if pain >48 hours

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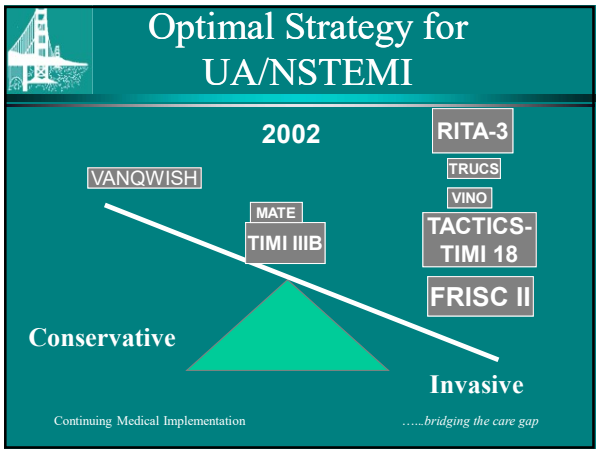
Optimal Strategy for UA/NSTEMI
2002
RITA-3
TRUCS
VINO
VANQWISH
MATE
TIMI IIIB
TACTICS-TIMI 18
FRISC II
Conservative
Invasive
Continuing Medical Implementation
.....bridging the care gap

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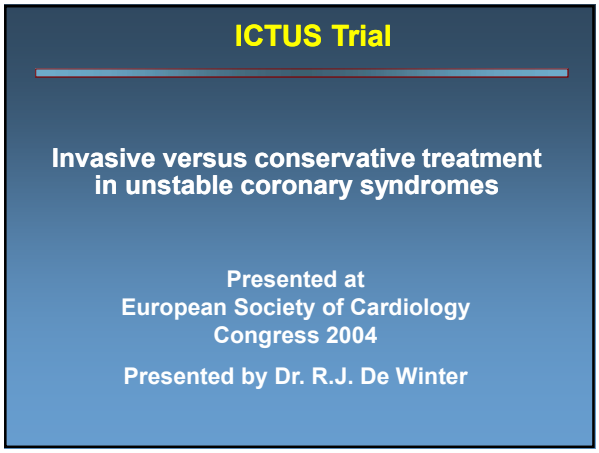
ICTUS Trial
Invasive versus conservative treatment in unstable coronary syndromes
Presented at
European Society of Cardiology
Congress 2004
Presented by Dr. R.J. De Winter

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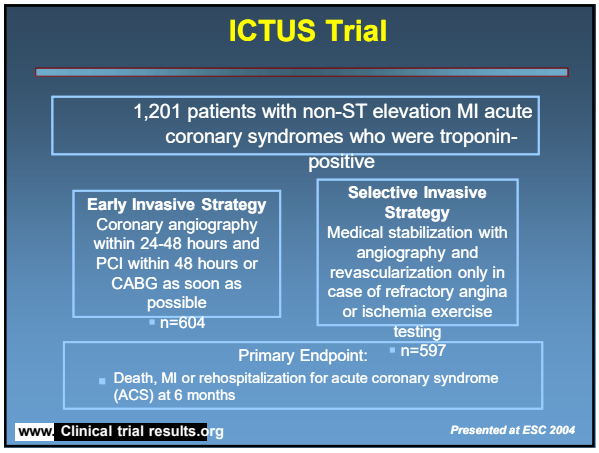
ICTUS Trial
1,201 patients with non-ST elevation MI acute coronary syndromes who were troponin-positive
Early Invasive Strategy
Coronary angiography within 24-48 hours and PCI within 48 hours or CABG as soon as possible
n=604
Selective Invasive Strategy
Medical stabilization with angiography and revascularization only in case of refractory angina or ischemia exercise testing
n=597
Primary Endpoint:
Death, MI or rehospitalization for acute coronary syndrome (ACS) at 6 months
www.Clinical trial results.org
Presented at ESC 2004

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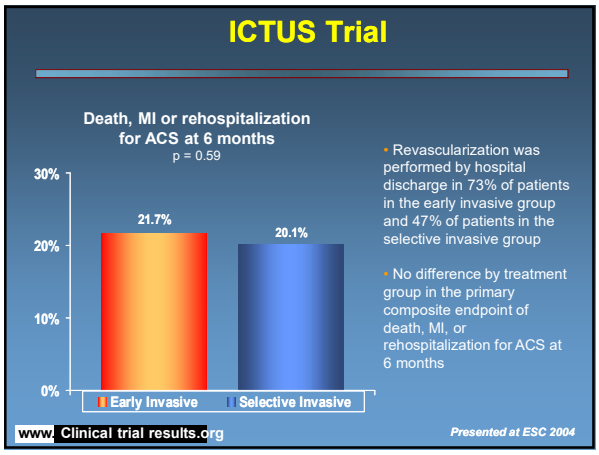
ICTUS Trial
Death, MI or rehospitalization for ACS at 6 months
p = 0.59
30%
20%
10%
0%
21.7%
20.1%
Early Invasive
Selective Invasive
• Revascularization was performed by hospital discharge in 73% of patients in the early invasive group and 47% of patients in the selective invasive group
• No difference by treatment group in the primary composite endpoint of death, MI, or rehospitalization for ACS at 6 months
www.Clinical trial results.org
Presented at ESC 2004

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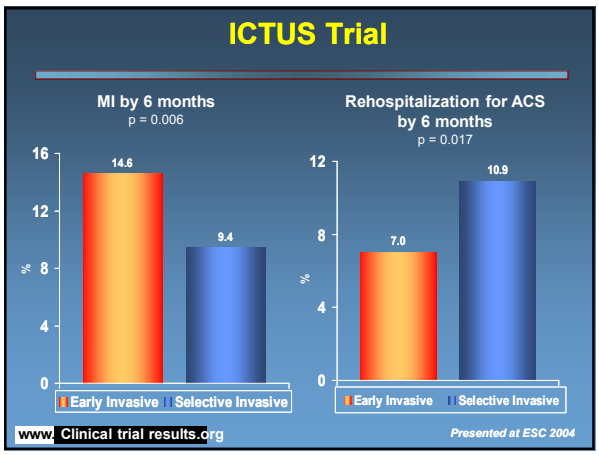
ICTUS Trial
MI by 6 months
p = 0.006
16
12
8
4
0
%
14.6
9.4
Early Invasive
Selective Invasive
Rehospitalization for ACS by 6 months
p = 0.017
12
8
4
0
%
7.0
10.9
Early Invasive
Selective Invasive
www.Clinical trial results.org
Presented at ESC 2004

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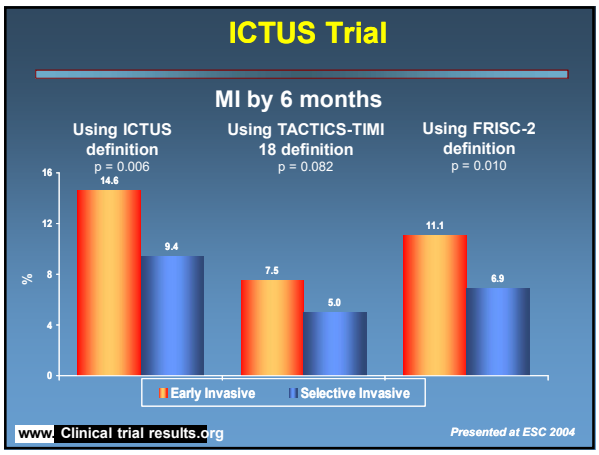
ICTUS Trial
MI by 6 months
Using ICTUS definition
p = 0.006
Using TACTICS-TIMI 18 definition
p = 0.082
Using FRISC-2 definition
p = 0.010
16
12
8
4
0
%
14.6
9.4
7.5
5.0
11.1
6.9
Early Invasive
Selective Invasive
www.Clinical trial results.org
Presented at ESC 2004

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ICTUS Trial

- Among troponin positive patients with a non-ST elevation ACS, treatment with an early invasive strategy was not associated with a difference in the primary endpoint compared with a selective invasive strategy
- However, two major components the primary endpoint, MI and rehospitalization for an ACS, show treatment differences in opposite direction
- Rate of MI in present trial notably higher than other similar trials, likely a reflection of peri-procedural MI given nonstringent definition of MI of CK-MB >1x ULN
- Primary endpoint and MI data in present trial differ from recent TACTICS-TIMI 18 trial and FRISC-2 trial, which showed benefit of an early invasive strategy over a conservative strategy in a similar patient population
- Additionally, larger percentage of patients in conservative strategy in present trial underwent early revascularization (47%) than in TACTICS-TIMI 18 (36%) or FRISC-2 (9%)

www.Clinical trial results.org

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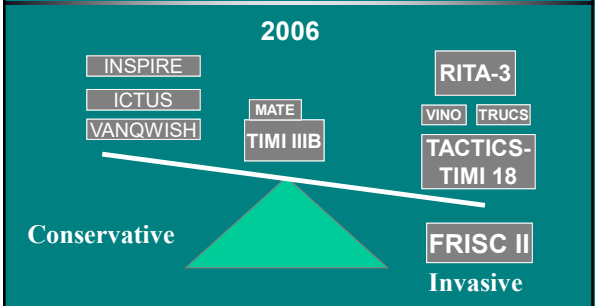
Optimal Strategy for UA/NSTEMI



Continuing Medical Implementation ……bridging the care gap

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Clinical Trial Links: Invasive vs Conservative Strategy for UAP/NSTEMI

Favors Conservative	No Difference	Favors Invasive
VANQUISH	TIMI III B	TACTICS-TIMI 18
INSPIRE	MATE	FRISC II
ICTUS		RITA 3
		TRUCS
		VINO

Continuing Medical Implementation ……bridging the care gap

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Management Strategy for ACS/NSTEMI

Non-cardiac exacerbation of known CAD
- Anemia
- Infection
- Hyperthyroidism
- Other medical causes

Patient preference
Stability
Normal LV Function
Good exercise tolerance
Relative C/I to CABG/PCI
Brief duration of ischaemia
No ST changes
Negative biomarkers

Conservative Therapy
(medical Rx/non-invasive evaluation)

Aggressive Therapy
(early cath/intervention)

Dynamic ST changes
Positive Enzymes or Biomarkers
Recurrent ischaemia on medical Rx
Prolonged ischaemic pain
PTCA < 6 months
Known severe CAD
LV dysfunction
- Pre-existing
- new MR
- new CHF

Intolerance to medical Rx
Recurrent ventricular arrythmia's/SCD
Early positive non-invasive test
Poor exercise tolerance

ACS/NSTEMI

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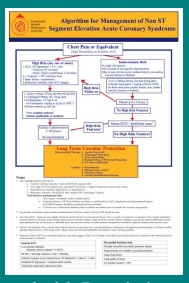
Algorithm for Management of Non ST Segment Elevation Acute Coronary Syndrome



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http://www.chrc.net/acsguidelines/ACS%20algorithm.pdf

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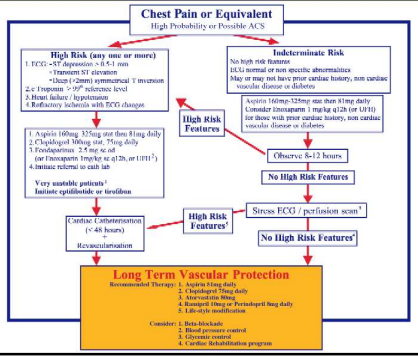
Algorithm for Management of Non ST Segment Elevation Acute Coronary Syndrome



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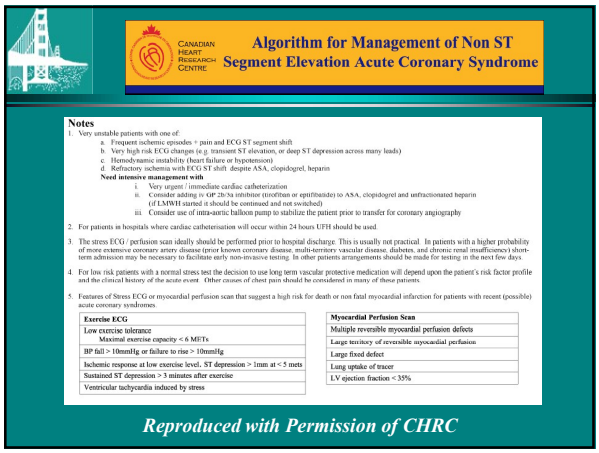
Canadian Heart Research Centre
Algorithm for Management of Non ST Segment Elevation Acute Coronary Syndrome
Notes
1. Very unstable patients with one of:
a. Frequent ischemic episodes + pain and ECG ST segment shift
b. Very high risk ECG changes (e.g. transient ST elevation, or deep ST depression across many leads)
c. Hemodynamic instability (heart failure or hypotension)
d. Refractory ischemia with ECG ST shift despite ASA, clopidogrel, heparin
Need intensive management with
i. Very urgent / immediate cardiac catheterization
ii. Consider adding iv GP 2b/3a inhibitor (tirofiban or eptifibatide) to ASA, clopidogrel and unfractionated heparin
(if LMWH started it should be continued and not switched)
iii. Consider use of intra-aortic balloon pump to stabilize the patient prior to transfer for coronary angiography
2. For patients in hospitals where cardiac catheterisation will occur within 24 hours UFH should be used.
3. The stress ECG / perfusion scan ideally should be performed prior to hospital discharge. This is usually not practical. In patients with a higher probability of more extensive coronary artery disease (prior known coronary disease, multi-territory vascular disease, diabetes, and chronic renal insufficiency) short-term admission may be necessary to facilitate early non-invasive testing. In other patients arrangements should be made for testing in the next few days.
4. For low risk patients with a normal stress test the decision to use long term vascular protective medication will depend upon the patient's risk factor profile and the clinical history of the acute event. Other causes of chest pain should be considered in many of these patients.
5. Features of Stress ECG or myocardial perfusion scan that suggest a high risk for death or non fatal myocardial infarction for patients with recent (possible) acute coronary syndromes.
Exercise ECG
Low exercise tolerance
Maximal exercise capacity < 6 METs
BP fall > 10mmHg or failure to rise > 10mmHg
Ischemic response at low exercise level, ST depression > 1mm at < 5 mets
Sustained ST depression > 3 minutes after exercise
Ventricular tachycardia induced by stress
Myocardial Perfusion Scan
Multiple reversible myocardial perfusion defects
Large territory of reversible myocardial perfusion
Large fixed defect
Lung uptake of tracer
LV ejection fraction < 35%
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