Non ST Elevation-ACS

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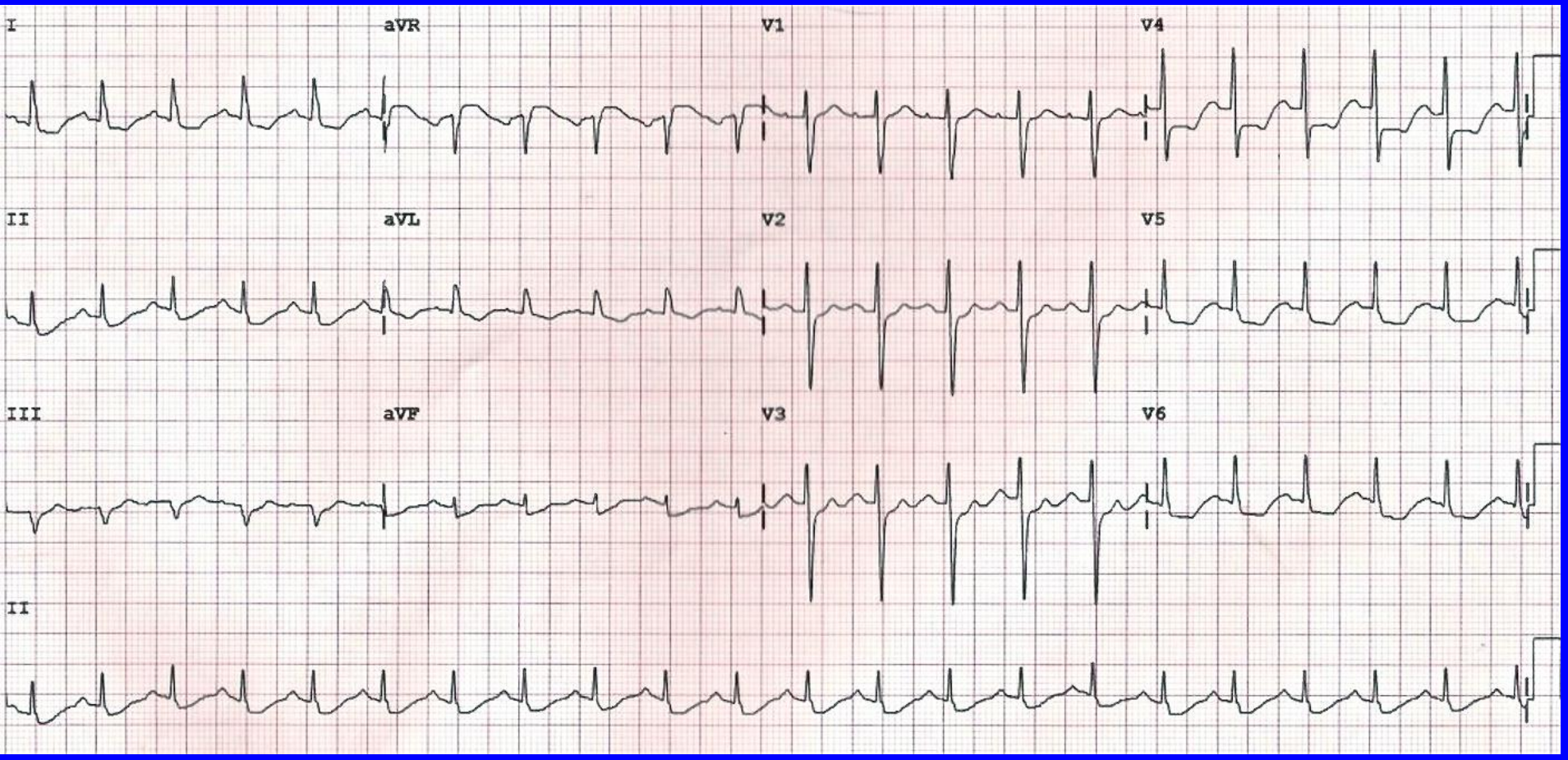
Case Presentation

- 65 year old man
  - PMH:
    - CAD s/p stent in 2008
    - HTN
    - HLD
  - Presents with chest pressure, substernally and radiating to the left arm and jaw, similar to prior MI.
  - He is seen in the ED and called to the floor for admission.

Case Presentation

- Patient was given nitroglycerin in the ED.
- Chest pain was relieved eventually by 4 mg IV morphine
- Troponin was 6.8 initially.

Case Presentation



Questions

- What anti-platelet therapy is most appropriate
- What could have been done better on the front end
- What anti-anginals should be used
- If the patients receives PCI should different anti-platelet agent be used?
- What do the guidelines say about manageing this patient?

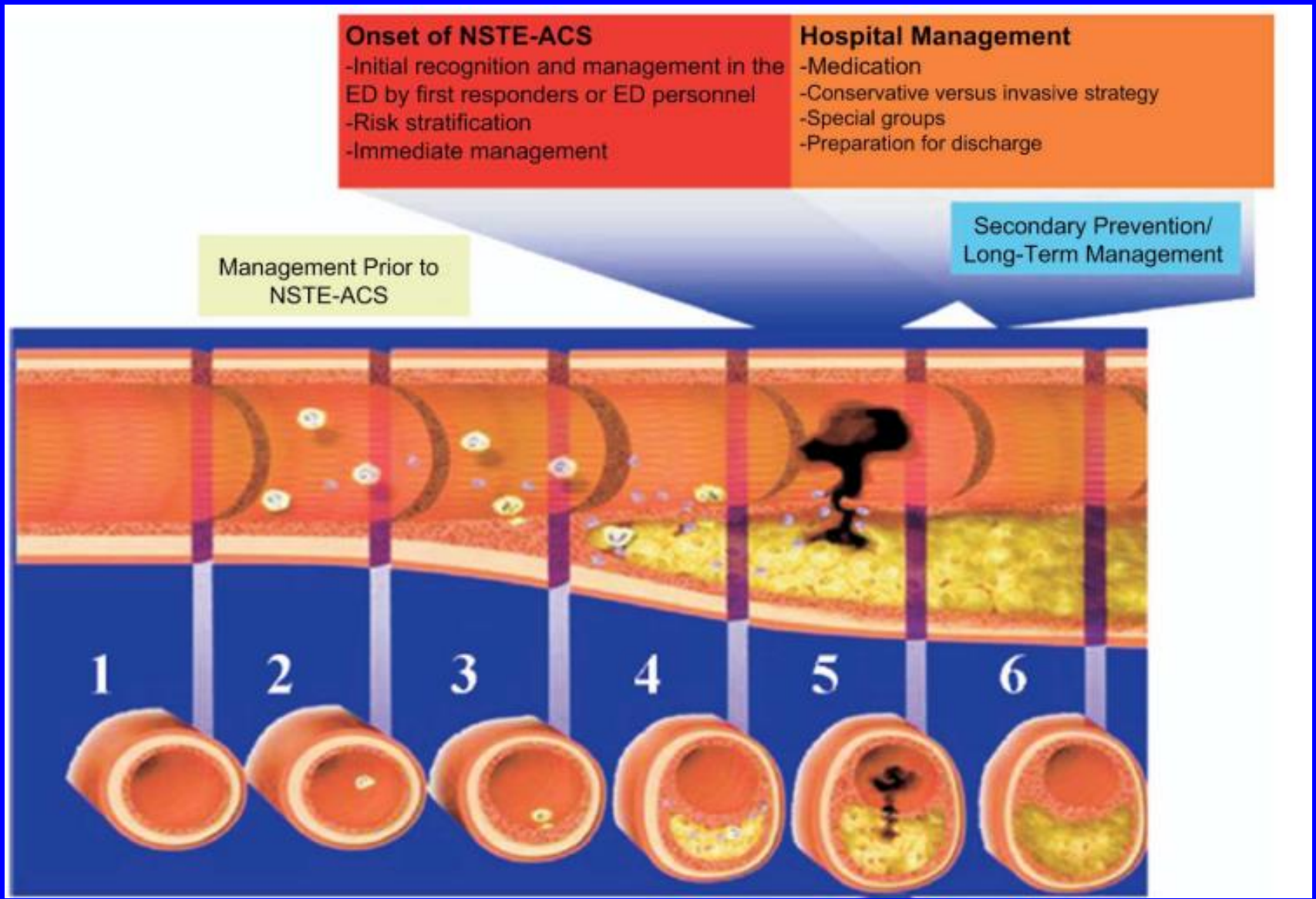
ACC/AHA Levels of Evidence

	CLASS I *Benefit >>> Risk* Procedure/Treatment **SHOULD** be performed/ administered	CLASS IIa *Benefit >> Risk* *Additional studies with focused objectives needed* **IT IS REASONABLE** to perform procedure/administer treatment	CLASS IIb *Benefit ≥ Risk* *Additional studies with broad objectives needed; additional registry data would be helpful* Procedure/Treatment **MAY BE CONSIDERED**	CLASS III *No Benefit* or CLASS III *Harm* Procedure/ Test / Treatment COR III: No benefit — Not Helpful — No Proven Benefit COR III: Harm — Excess Cost w/o Benefit or Harmful — Harmful to Patients
LEVEL A Multiple populations evaluated* Data derived from multiple randomized clinical trials or meta-analyses	■ Recommendation that procedure or treatment is useful/effective ■ Sufficient evidence from multiple randomized trials or meta-analyses	■ Recommendation in favor of treatment or procedure being useful/effective ■ Some conflicting evidence from multiple randomized trials or meta-analyses	■ Recommendation's usefulness/efficacy less well established ■ Greater conflicting evidence from multiple randomized trials or meta-analyses	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Sufficient evidence from multiple randomized trials or meta-analyses
LEVEL B Limited populations evaluated* Data derived from a single randomized trial or nonrandomized studies	■ Recommendation that procedure or treatment is useful/effective ■ Evidence from single randomized trial or nonrandomized studies	■ Recommendation in favor of treatment or procedure being useful/effective ■ Some conflicting evidence from single randomized trial or nonrandomized studies	■ Recommendation's usefulness/efficacy less well established ■ Greater conflicting evidence from single randomized trial or nonrandomized studies	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Evidence from single randomized trial or nonrandomized studies
LEVEL C Very limited populations evaluated* Only consensus opinion of experts, case studies, or standard of care	■ Recommendation that procedure or treatment is useful/effective ■ Only expert opinion, case studies, or standard of care	■ Recommendation in favor of treatment or procedure being useful/effective ■ Only diverging expert opinion, case studies, or standard of care	■ Recommendation's usefulness/efficacy less well established ■ Only diverging expert opinion, case studies, or standard of care	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Only expert opinion, case studies, or standard of care

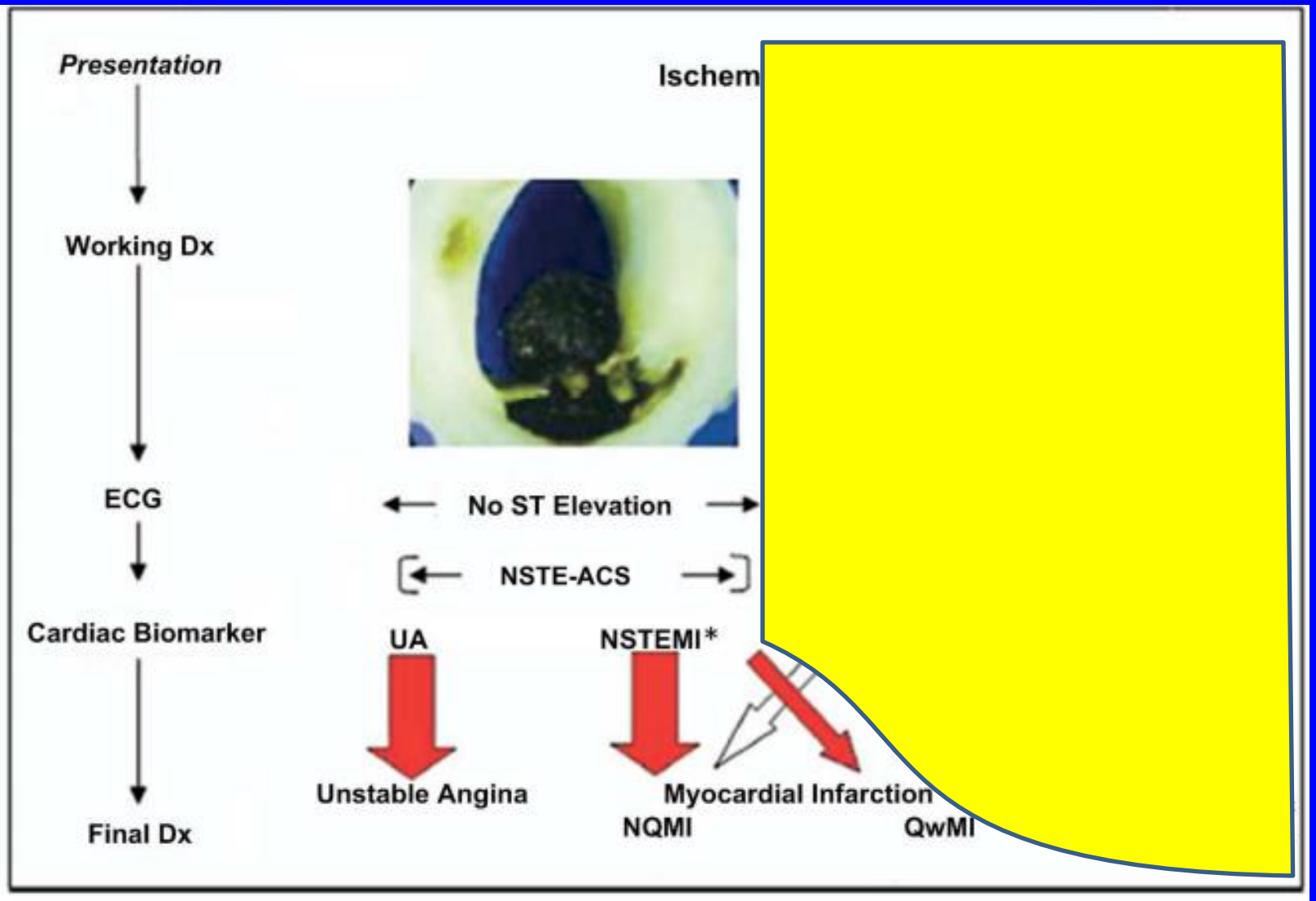
Definition

- Acute Coronary Syndrome (ACS)
  - A spectrum of conditions compatible with acute myocardial ischemia and/or infarction, usually due to an abrupt decrease in coronary blood flow
- Non ST Elevation ACS
  - ACS in the absence of persistent ST-elevation.
  - Encompasses both unstable angina and NSTEMI

NSTE-ACS pathogenesis



NSTE-ACS pathogenesis



Initial Evaluation and Management

- Class I
  - Patient with suspected ACS should be risk stratified based on likelihood of ACS and adverse outcomes to decide need for hospitalization.
    - HEART Score
    - Vancouver Rule
    - TIMI Risk Score
    - PURSUIT risk score
    - GRACE risk score

TIMI Score

- Age ≥65
- ≥3 CAD risk factors
  - Hypertension, hypercholesterolemia, diabetes, family history of CAD, or current smoker
- Known CAD (stenosis ≥50%)
- ASA use in past 7 days
- Severe angina (≥2 episodes in 24 hrs)
- EKG ST changes ≥0.5mm
- Positive cardiac marker

GRACE Score

- AGE
- HR
- SBP
- Creatinine
- Killip Class
- Cardiac arrest
- Elevated CBMs
- ST Deviation

HEART Score

- History
- EKG
- Age
- Risk Factors
- Initial Troponin

Third Universal Definition of MI

Definition of Myocardial Infarction

Criteria for acute myocardial infarction

The term acute myocardial infarction (MI) should be used when there is evidence of myocardial necrosis in a clinical setting consistent with acute myocardial ischemia. Under these conditions any one of the following criteria meets the diagnosis for MI:

- Detection of a rise and/or fall of cardiac biomarker values [preferably cardiac troponin (cTn)] with at least one value above the 99th percentile upper reference limit (URL) and with at least one of the following:
  - Symptoms of ischemia.
  - New or presumed new significant ST-segment–T wave (ST–T) changes or new left bundle branch block (LBBB).
  - Development of pathological Q waves in the ECG.
  - Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
  - Identification of an intracoronary thrombus by angiography or autopsy.
- Cardiac death with symptoms suggestive of myocardial ischemia and presumed new ischemic ECG changes or new LBBB, but death occurred before cardiac biomarkers were obtained, or before cardiac biomarker values would be increased.
- Percutaneous coronary intervention (PCI) related MI is arbitrarily defined by elevation of cTn values (>5 × 99th percentile URL) in patients with normal baseline values (≤99th percentile URL) or a rise of cTn values >20% if the baseline values are elevated and are stable or falling. In addition, either (i) symptoms suggestive of myocardial ischemia or (ii) new ischemic ECG changes or (iii) angiographic findings consistent with a procedural complication or (iv) imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality are required.
- Stent thrombosis associated with MI when detected by coronary angiography or autopsy in the setting of myocardial ischemia and with a rise and/or fall of cardiac biomarker values with at least one value above the 99th percentile URL.
- Coronary artery bypass grafting (CABG) related MI is arbitrarily defined by elevation of cardiac biomarker values (>10 × 99th percentile URL) in patients with normal baseline cTn values (≤99th percentile URL). In addition, either (i) new pathological Q waves or new LBBB, or (ii) angiographic documented new graft or new native coronary artery occlusion, or (iii) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

Criteria for prior myocardial infarction

Any one of the following criteria meets the diagnosis for prior MI:

- Pathological Q waves with or without symptoms in the absence of non-ischemic causes.
- Imaging evidence of a region of loss of viable myocardium that is thinned and fails to contract, in the absence of a non-ischemic cause.
- Pathological findings of a prior MI.

Third Universal Definition of MI

Table 2. Universal Classification of Myocardial Infarction

Type 1: Spontaneous myocardial infarction

Spontaneous myocardial infarction related to atherosclerotic plaque rupture, ulceration, Assuring, erosion, or dissection with resulting intraluminal thrombus in one or more of the coronary arteries leading to decreased myocardial blood flow or distal platelet emboli with ensuing myocyte necrosis. The patient may have underlying severe CAD but on occasion non-obstructive or no CAD.

Type 2: Myocardial infarction secondary to an ischemic imbalance

In instances of myocardial injury with necrosis where a condition other than CAD contributes to an imbalance between myocardial oxygen supply and/or demand, e.g. coronary endothelial dysfunction, coronary artery spasm, coronary embolism, tachy-/brady-arrhythmias, anemia, respiratory failure, hypotension, and hypertension with or without LVH.

Type 3: Myocardial infarction resulting in death when biomarker values are unavailable

Cardiac death with symptoms suggestive of myocardial ischemia and presumed new ischemic ECG changes or new LBBB, but death occurring before blood samples could be obtained, before cardiac biomarker could rise, or in rare cases cardiac biomarkers were not collected.

Type 4a: Myocardial infarction related to percutaneous coronary intervention (PCI)

Myocardial infarction associated with PCI is arbitrarily defined by elevation of cTn values >5 x 99th percentile URL in patients with normal baseline values (<99^{th} percentile URL) or a rise of cTn values >20% if the baseline values are elevated and are stable or falling. In addition, either (i) symptoms suggestive of myocardial ischemia, or (ii) new ischemic ECG changes or new LBBB, or (iii) angiographic loss of patency of a major coronary artery or a side branch or persistent slow- or no-flow or embolization, or (iv) imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality are required.

Type 4b: Myocardial infarction related to stent thrombosis

Myocardial infarction associated with stent thrombosis is detected by coronary angiography or autopsy in the setting of myocardial ischemia and with a rise and/or fall of cardiac biomarkers values with at least one value above the 99th percentile URL.

Type 5: Myocardial infarction related to coronary artery bypass grafting (CABG)

Myocardial infarction associated with CABG is arbitrarily defined by elevation of cardiac biomarker values >10 x 99th percentile URL in patients with normal baseline cTn values (<99th percentile URL). In addition, either (i) new pathological Q waves or new LBBB, or (ii) angiographic documented new graft or new native coronary artery occlusion, or (iii) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

Early Risk Stratification

Recommendations	COR	LOE
Perform rapid determination of likelihood of ACS, including a 12-lead ECG within 10 min of arrival at an emergency facility, in patients whose symptoms suggest ACS	I	C
Perform serial ECGs at 15- to 30-min intervals during the first hour in symptomatic patients with initial nondiagnostic ECG	I	C
Measure cardiac troponin (cTnI or cTnT) in all patients with symptoms consistent with ACS*	I	A
Measure serial cardiac troponin I or T at presentation and 3-6 h after symptom onset* in all patients with symptoms consistent with ACS	I	A
Use risk scores to assess prognosis in patients with NSTE-ACS	I	A
Risk-stratification models can be useful in management	IIa	B
Obtain supplemental electrocardiographic leads V_7 to V_9 in patients with initial nondiagnostic ECG at intermediate/high risk for ACS	IIa	B
Continuous monitoring with 12-lead ECG may be a reasonable alternative with initial nondiagnostic ECG in patients at intermediate/high risk for ACS	IIb	B
BNP or NT-pro-BNP may be considered to assess risk in patients with suspected ACS	IIb	B

Biomarker Use

Recommendations	COR	LOE
Diagnosis		
Measure cardiac-specific troponin (troponin I or T) at presentation and 3–6 h after symptom onset in all patients with suspected ACS to identify pattern of values	I	A
Obtain additional troponin levels beyond 6 h in patients with initial normal serial troponins with electrocardiographic changes and/or intermediate/high risk clinical features	I	A
Consider time of presentation the time of onset with ambiguous symptom onset for assessing troponin values	I	A
With contemporary troponin assays, CK-MB and myoglobin are not useful for diagnosis of ACS	III: No Benefit	A
Prognosis		
Troponin elevations are useful for short- and long-term prognosis	I	B
Remeasurement of troponin value once on d 3 or 4 in patients with MI may be reasonable as an index of infarct size and dynamics of necrosis	IIb	B
BNP may be reasonable for additional prognostic information	IIb	B

Immediate Management

- Class IIa recommendations
  - Observe patients with symptoms consistent with ACS without objective data of myocardial ischemia
  - To stress (treadmill EKG, MPI, echo) patient with possible ACS with normal EKG and negative CMBs, within 72 hours as inpatient or outpatient. (A)
  - CTA in low risk patients with no history of CAD (A)
  - DC with aspirin, NTG, beta blocker (C).

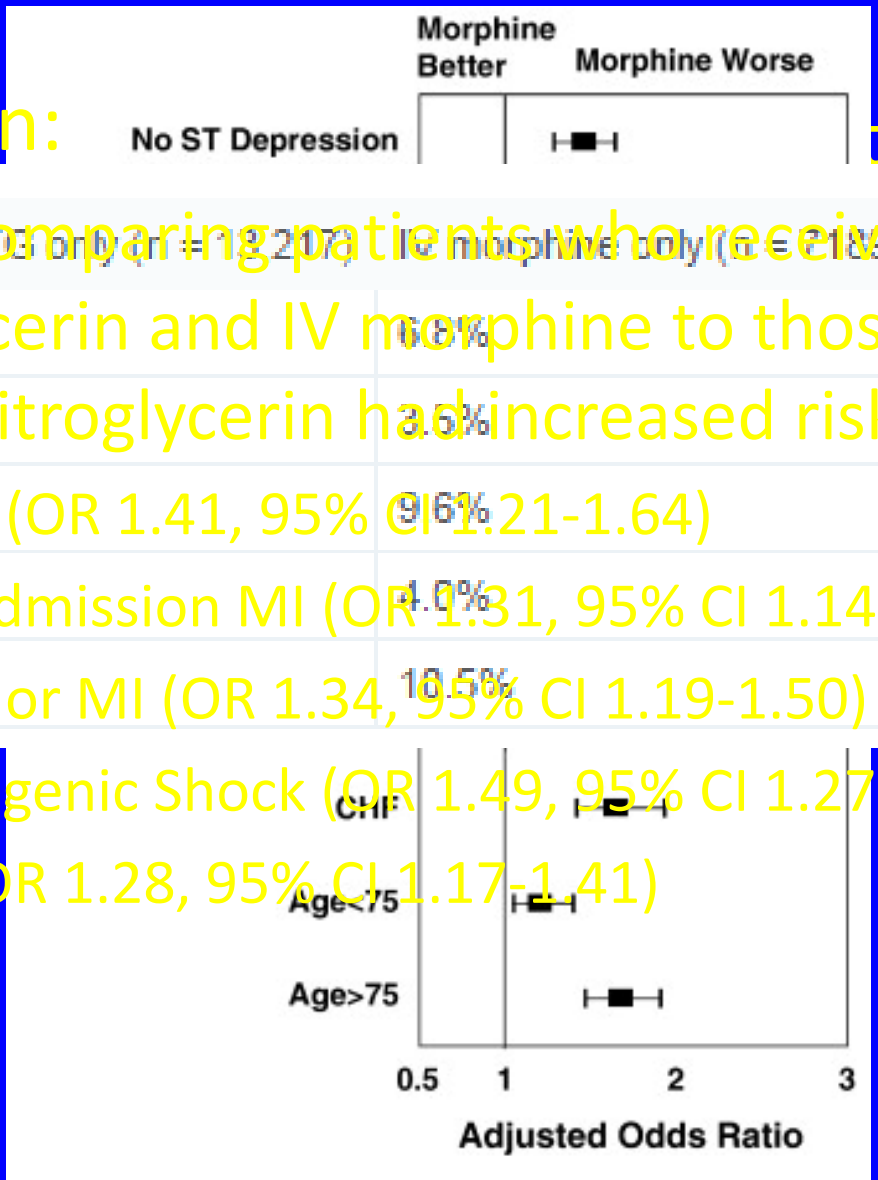
Initial Hospital Management

Recommendations	COR	LOE
CCBs		
Analgesic therapy		
IV morphine sulfate may be reasonable for continued ischemic chest pain despite maximally tolerated anti-ischemic medications	IIb	B
NSAIDs (except aspirin) should not be initiated and should be discontinued during hospitalization for NSTE-ACS because of the increased risk of MACE associated with their use	III: Harm	B
CCBs are recommended for ischemic symptoms when beta blockers are not successful, are contraindicated, or cause unacceptable side effects*	I	
Long-acting CCBs and nitrates are recommended for patients with coronary artery spasm	I	
Immediate-release nifedipine is contraindicated in the absence of a beta blocker	III: Harm	

Morphine and NSTE-ACS

- In addition: CRUSADE quantitative
  - When comparing patients who received both IV nitroglycerin and IV morphine to those receiving just IV nitroglycerin had increased risk of:
    - Death (OR 1.41, 95% CI 1.21-1.64)
    - Post-admission MI (OR 1.31, 95% CI 1.14-1.51)
    - Death or MI (OR 1.34, 95% CI 1.19-1.50)
    - Cardiogenic Shock (OR 1.49, 95% CI 1.27-1.74)
    - CHF (OR 1.28, 95% CI 1.17-1.41)

Outcomes	IV NTG only (n = 3,207)	IV morphine only (n = 2,184)	Adjusted OR (95% CI)*
Death	2.8%	6.8%	1.49 (1.25-1.77)
Post-admission MI	3.2%	5.6%	1.18 (0.99-1.41)
Death or MI	6.5%	9.6%	1.40 (1.22-1.62)
Cardiogenic shock	2.4%	4.0%	1.44 (1.19-1.74)
CHF	8.8%	10.5%	1.06 (0.93-1.20)



ACE Inhibitors/ARBs

- Class I (A)
  - ACE should be started in all patient's with LVEF <0.4 and in those with HTN, DM, or stable CKD
  - ARBs in above who are ACE intolerant
  - Aldosterone blockade in patient post MI without significant renal dysfunction (cr>2.5m or 2.0 w) who are receiving therapeutic doses of ACE and BB if EF <40, dm.
- Class IIa
  - ACE may be reasonable in all patient with cardiac or vascular disease. (B)

Clinical Treatment Pathways

- As of the 2014 guidelines these replaced previous guidelines for an initial invasive vs an initial conservative therapy, the guideline update changes the treatment pathways.
  - Urgent/Immediate invasive strategy
  - Early invasive strategy
  - Ischemia guided therapy.

Ischemia Guided Therapy Pathway

- This will tend to be your relatively low risk unstable angina patient.
- Invasive strategy will be implemented if initial medical therapy failure is determined
  - Refractory angina or angina at rest after therapy
  - Evidence of ischemia
    - EKG changes, +stress,
  - High TIMI or Grace scores.

Ischemia Guided Therapy Pathway

- Intended to avoid the routine use of early invasive procures unless patient require them.
  - This is an effort to save patients who might stabilize on medical therapy alone from having to undergo invasive therapy.
  - Severe ischemia on non-invasive studies warrants further evaluation with invasive strategies

Early Invasive Therapy Pathway

- High grade UA or NSTEMI
  - These patient are patient with high or intermediate scores on risk stratification methods.
  - Will be designated for cardiac catheterization.
  - Initial management is very similar to management for the ischemia guided therapy.

Ischemia Guided Therapy

TABLE 8 Factors Associated With Appropriate Selection of Early Invasive Strategy or Ischemia-Guided Strategy in Patients With NSTE-ACS

Immediate invasive (within 2 h)	Refractory angina
	Signs or symptoms of HF or new or worsening mitral regurgitation
	Hemodynamic instability
	Recurrent angina or ischemia at rest or with low-level activities despite intensive medical therapy
	Sustained VT or VF
Ischemia-guided strategy	Low-risk score (e.g., TIMI [0 or 1], GRACE [<109])
	Low-risk Tn-negative female patients
	Patient or clinician preference in the absence of high-risk features
Early invasive (within 24 h)	None of the above, but GRACE risk score >140
	Dynamic change in Tn (Section 3.4)
	New or presumably new ST depression
Delayed invasive (within 25–72 h)	None of the above but diabetes mellitus
	Renal insufficiency (GFR <60 mL/min/1.73 m^2)
	Reduced LV systolic function (EF <0.40)
	Early postinfarction angina
	PCI within 6 mo
	Prior CABG
	GRACE risk score 109–140; TIMI score ≥2

EMERGENT

LOW RISK

HIGH RISK

INTERMEDIATE RISK

Pathway Guidelines

- Class I
  - Urgent/Immediate invasive stratagy indicated in patient with NSTE-ACS with refractory angina or hemodynamic or electrical instability without serious comorbidities or contraindications (A)
  - Early invasive strategy is indicated in initally stabilized patient with NSTE-ACS who have elevated risk for clinical events (B)
    - Elevated troponin or elevated GRACE or TIMI score

Pathway Guidelines

- Class IIa
  - Early invasive strategy over delayed strategy for initially stabilized high risk patients. For those not at high/intermediate risk, delayed invasive approach is reasonable. (B)
- IIb – initially stabilized patients may be considered for ischemia-guided strategy if
  - Elevated risk for clinical events (B)
  - After considering clinical and patient preference (C)

Pathway Guidelines

- Class III – No benefit
  - Early invasive strategy not recommended in patients with
    - Extensive comorbidities (hepatic, renal, pulmonary failure, cancer) in whom the risk of revascularization and comorbid conditions are likely to outweigh the benefits of revascularization.
    - Acute chest pain and low likelihood of ACS who are troponin negative, especially women.

Lets Start Off Easy

Aspirin			
• Non-enteric-coated aspirin to *all* patients promptly after presentation	162 mg–325 mg	I	A
• Aspirin maintenance dose continued indefinitely	81 mg/d–325 mg/d*	I	A

Anti-Platelet Agents

$P2Y_{12}$ inhibitors			
• Clopidogrel loading dose followed by daily maintenance dose in patients unable to take aspirin	75 mg	I	B
• $P2Y_{12}$ inhibitor, in addition to aspirin, for up to 12 mo for patients treated initially with either an early invasive or initial ischemia-guided strategy: – Clopidogrel – Ticagrelor*	300-mg or 600-mg loading dose, then 75 mg/d 180-mg loading dose, then 90 mg BID	I	B
• $P2Y_{12}$ inhibitor therapy (clopidogrel, prasugrel, or ticagrelor) continued for at least 12 mo in post-PCI patients treated with coronary stents	N/A	I	B
• Ticagrelor in preference to clopidogrel for patients treated with an early invasive or ischemia-guided strategy	N/A	IIa	B

GP IIb/IIIa inhibitors			
• GP IIb/IIIa inhibitor in patients treated with an early invasive strategy and DAPT with intermediate/high-risk features (e.g., positive troponin)	Preferred options are eptifibatide or tirofiban	IIb	B

Anticoagulation

• SC enoxaparin for duration of hospitalization or until PCI is performed	• 1 mg/kg SC every 12 h (reduce dose to 1 mg/kg/d SC in patients with CrCl <30 mL/min) • Initial 30 mg IV loading dose in selected patients	I	A
• Bivalirudin until diagnostic angiography or PCI is performed in patients with early invasive strategy only	• Loading dose 0.10 mg/kg loading dose followed by 0.25 mg/kg/h • Only provisional use of GP IIb/IIIa inhibitor in patients also treated with DAPT	I	B
• SC fondaparinux for the duration of hospitalization or until PCI is performed	2.5 mg SC daily	I	B
• Administer additional anticoagulant with anti-IIa activity if PCI is performed while patient is on fondaparinux	N/A	I	B
• IV UFH for 48 h or until PCI is performed	• Initial loading dose 60 IU/kg (max 4,000 IU) with initial infusion 12 IU/kg/h (max 1,000 IU/ h) • Adjusted to therapeutic aPTT range	I	B
• IV fibrinolytic treatment not recommended in patients with NSTE-ACS	N/A	III: Harm	A

Why is Effient not preferred?

Table 2. Major Efficacy End Points in the Overall Cohort at 15 Months.*

End Point	Prasugrel (N=6813)	Clopidogrel (N=6795)	Hazard Ratio for Prasugrel (95% CI)	P Value†
	no. of patients (%)			
Death from cardiovascular causes, nonfatal MI, or nonfatal stroke (primary end point)	643 (9.9)	781 (12.1)	0.81 (0.73–0.90)	<0.001
Death from cardiovascular causes	133 (2.1)	150 (2.4)	0.89 (0.70–1.12)	0.31
Nonfatal MI	475 (7.3)	620 (9.5)	0.76 (0.67–0.85)	<0.001
Nonfatal stroke	61 (1.0)	60 (1.0)	1.02 (0.71–1.45)	0.93
Death from any cause	188 (3.0)	197 (3.2)	0.95 (0.78–1.16)	0.64
Death from cardiovascular causes, nonfatal MI, or urgent target-vessel revascularization	652 (10.0)	798 (12.3)	0.81 (0.73–0.89)	<0.001
Death from any cause, nonfatal MI, or nonfatal stroke	692 (10.7)	822 (12.7)	0.83 (0.75–0.92)	<0.001
Urgent target-vessel revascularization	156 (2.5)	233 (3.7)	0.66 (0.54–0.81)	<0.001
Death from cardiovascular causes, nonfatal MI, nonfatal stroke, or rehospitalization for ischemia	797 (12.3)	938 (14.6)	0.84 (0.76–0.92)	<0.001
Stent thrombosis‡	68 (1.1)	142 (2.4)	0.48 (0.36–0.64)	<0.001

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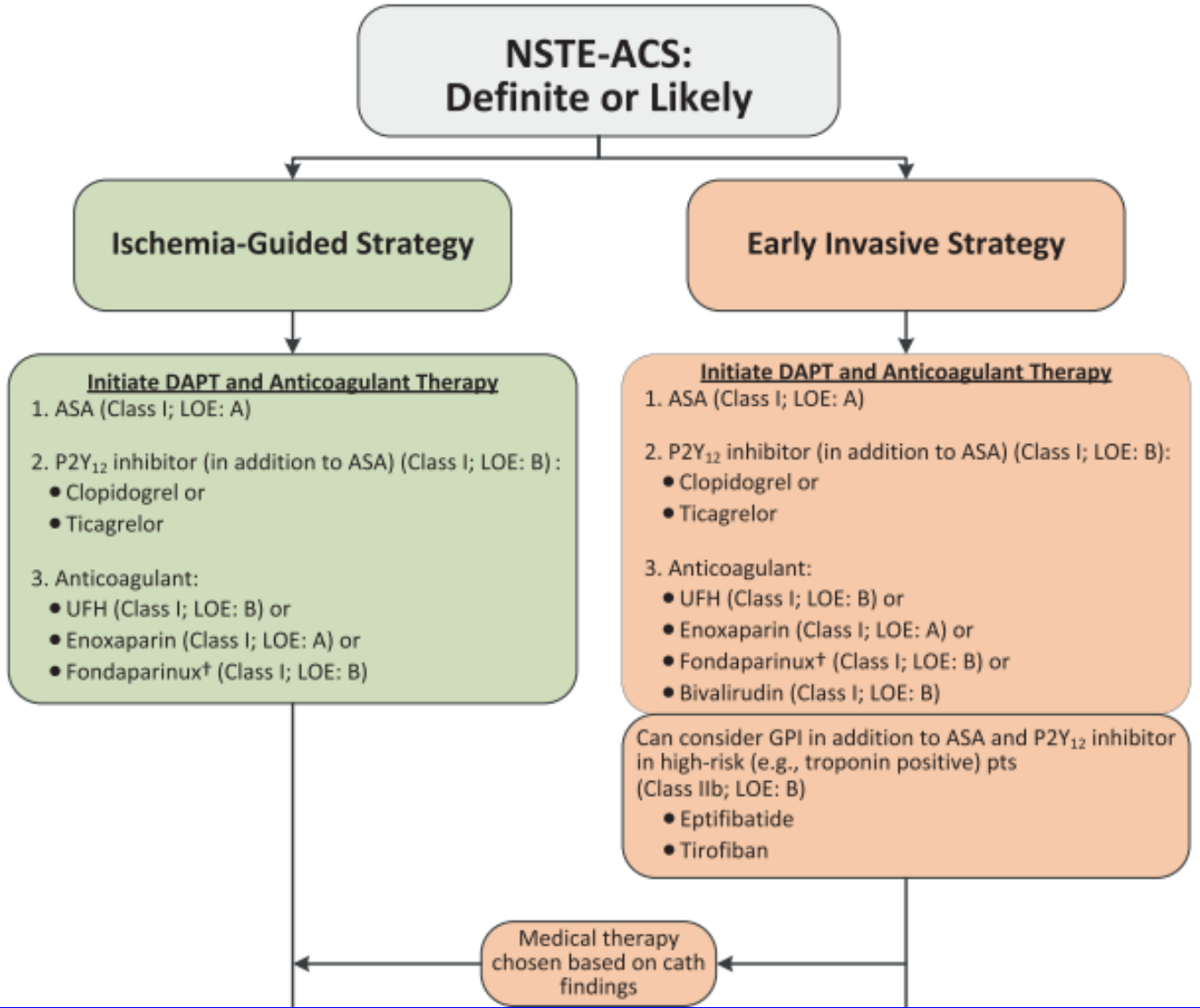
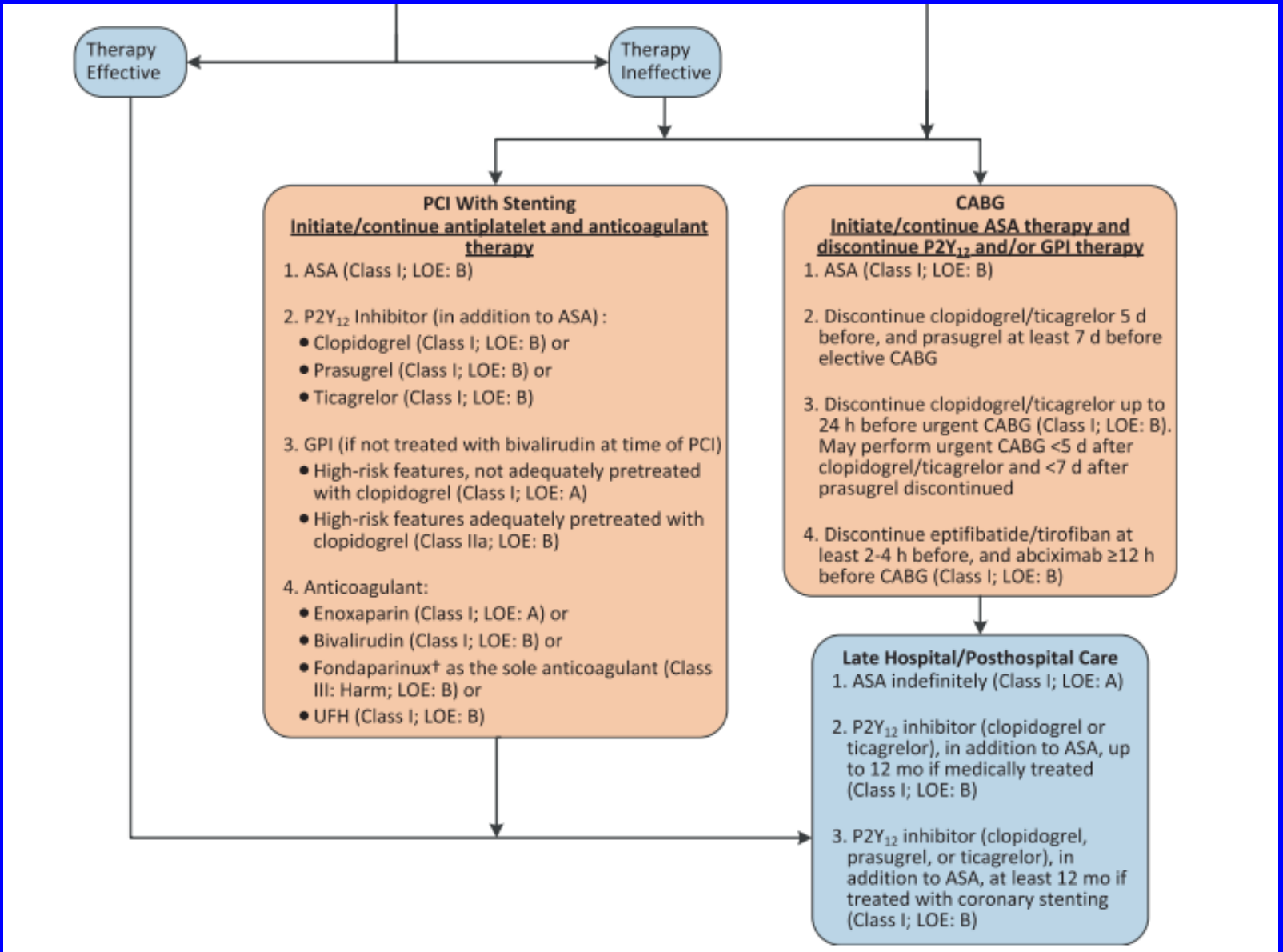
Table 3. Major Efficacy End Points at 12 Months.*

End Point	Ticagrelor Group	Clopidogrel Group	Hazard Ratio for Ticagrelor Group (95% CI)	P Value†
Primary end point: death from vascular causes, MI, or stroke — no./total no. (%)	864/9333 (9.8)	1014/9291 (11.7)	0.84 (0.77–0.92)	<0.001‡
Secondary end points — no./total no. (%)				
Death from any cause, MI, or stroke	901/9333 (10.2)	1065/9291 (12.3)	0.84 (0.77–0.92)	<0.001‡
Death from vascular causes, MI, stroke, severe recurrent ischemia, recurrent ischemia, TIA, or other arterial thrombotic event	1290/9333 (14.6)	1456/9291 (16.7)	0.88 (0.81–0.95)	<0.001‡
MI	504/9333 (5.8)	593/9291 (6.9)	0.84 (0.75–0.95)	0.005‡
Death from vascular causes	353/9333 (4.0)	442/9291 (5.1)	0.79 (0.69–0.91)	0.001‡
Stroke	125/9333 (1.5)	106/9291 (1.3)	1.17 (0.91–1.52)	0.22
Ischemic	96/9333 (1.1)	91/9291 (1.1)		0.74
Hemorrhagic	23/9333 (0.2)	13/9291 (0.1)		0.10
Unknown	10/9333 (0.1)	2/9291 (0.02)		0.04
Other events — no./total no. (%)				
Death from any cause	399/9333 (4.5)	506/9291 (5.9)	0.78 (0.69–0.89)	<0.001
Death from causes other than vascular causes	46/9333 (0.5)	64/9291 (0.8)	0.71 (0.49–1.04)	0.08
Severe recurrent ischemia	302/9333 (3.5)	345/9291 (4.0)	0.87 (0.74–1.01)	0.08
Recurrent ischemia	500/9333 (5.8)	536/9291 (6.2)	0.93 (0.82–1.05)	0.22
TIA	18/9333 (0.2)	23/9291 (0.3)	0.78 (0.42–1.44)	0.42
Other arterial thrombotic event	19/9333 (0.2)	31/9291 (0.4)	0.61 (0.34–1.08)	0.09
Death from vascular causes, MI, stroke — no./total no. (%)				
Invasive treatment planned§	569/6732 (8.9)	668/6676 (10.6)	0.84 (0.75–0.94)	0.003‡
Event rate, days 1–30	443/9333 (4.8)	502/9291 (5.4)	0.88 (0.77–1.00)	0.045
Event rate, days 31–360¶	413/8763 (5.3)	510/8688 (6.6)	0.80 (0.70–0.91)	<0.001
Stent thrombosis — no. of patients who received a stent/total no. (%)				
Definite	71/5640 (1.3)	106/5649 (1.9)	0.67 (0.50–0.91)	0.009
Probable or definite	118/5640 (2.2)	158/5649 (2.9)	0.75 (0.59–0.95)	0.02
Possible, probable, or definite	155/5640 (2.9)	202/5649 (3.8)	0.77 (0.62–0.95)	0.01

Inpatient Management Guidelines



Inpatient Management Guidelines



Risk Stratification In Ischemia Guided

- Class I
  - Noninvasive stress testing recommended in low and intermediate risk patient who have been free of ischemia at rest or with low level activity for 12-24 hours.
  - Treadmill testing useful in patients able to exercise who have EKG free of ST changes
  - Stress testing with imaging modality should be used in those who do not qualify for EKG stress testing
  - Pharm stress testing with imaging recommended when physical limitations preclude adequate exercise stress.
  - Noninvasive imaging test is recommended to evaluate LV function in patient with definite ACS.

PCI Recommendations

- Class IIb
  - A strategy of multivessel PCI, in contrast to culprit lesion only PCI may be reasonable in patients undergoing coronary revascularization as part of treatment for NSTE-ACS

PCI – Antiplatelet Agents

- Remain Essentially unchanged
  - Class I
    - Patient on ASA should be given 81-325 prior to PCI
    - Patient not on ASA should be given 325 prior to PCI
    - After PCI ASA at 81-325 daily indefinately
    - Loading dose of P2Y12 inhibitor
      - Ticagrelor
      - Prasugrel – may switch and reload at this time
      - Clopidogrel
    - IIb/IIIa ihibitor if NSTE-ACS and high risk not adequately treat with clopidogrel or ticagrelor
    - 12 months of DAPT if stent placement.

PCI – Antiplatelet Agents

- Class IIa – reasonable to choose:
  - Ticagrelor over clopidogrel in early invasive category and coronary stenting
  - Prasugrel if not at high risk of bleeding
  - 81 mg of ASA in preference to higher dose after PCI
  - Early discontinuation of P2Y12 antagonist if risk of morbidity from bleeding outweighs the anticipated risk

PCI – Antiplatelet Agents

- Class III- Harm
  - Effient should be avoided in patient with prior stroke or TIA.

DAPT study

Table 3. Bleeding End Point during Month 12 to Month 30.*

Bleeding Complications	Continued Thienopyridine (N=4710)	Placebo (N=4649)	Difference	Two-Sided P Value for Difference
	no. of patients (%)		*percentage points (95% CI)*	
GUSTO severe or moderate†	119 (2.5)	73 (1.6)	1.0 (0.4 to 1.5)	0.001
Severe	38 (0.8)	26 (0.6)	0.2 (–0.1 to 0.6)	0.15
Moderate	81 (1.7)	48 (1.0)	0.7 (0.2 to 1.2)	0.004
BARC type 2, 3, or 5	263 (5.6)	137 (2.9)	2.6 (1.8 to 3.5)	<0.001
Type 2	145 (3.1)	72 (1.5)	1.5 (0.9 to 2.1)	<0.001
Type 3	122 (2.6)	68 (1.5)	1.1 (0.6 to 1.7)	<0.001
Type 5	7 (0.1)	4 (0.1)	0.1 (–0.1 to 0.2)	0.38
Stro				0.32
				0.16
				0.68
				0.32

0 12 15 18 21 24 27 30 33

Months since Enrollment

No. at	No. at Risk							
Thieno	Thienopyridine	5020	4917	4840	4778	4702	4611	4554	3029
Placebo	Placebo	4941	4799	4715	4635	4542	4476	4412	2997

When Patient Needs CABG

- Class I:
  - ASA should be continued
  - Clopidogrel or ticagrelor should be discontinued 5 days prior to surgery
  - Prasugrel 7 days prior to surgery
- Urgent CABG
  - Clopidogrel and Ticagrelor should be discontinued at least 24 hours prior to surgery to reduce major bleeding
  - IIb/IIIa inhibitor should be stopped 2-4 hours prior to surgery
- Class IIb:
  - Urgent CABG may reasonably be performed less that 5 days after clopidogrel or ticagrelor or less than 7 days day after prasugrel discontinued.

Remainder of recommendations

- These regard discharge planning and follow-up and remain essentially unchanged from prior guideline sets
  - Cardiac rehab
  - Long term therapy

Cangrelor?

- Short lived.
- Only studied in ACS, no indication for NSTEMI prior to PCI.
- Leave this one to your interventionalist

Discussion

- What anti-platelet therapy is most appropriate?
- What could have been done better on the front end?
- What anti-anginals should be used?
- If the patients receives PCI should different anti-platelet agent be used?
- What are the new guidelines for caring for this patient?