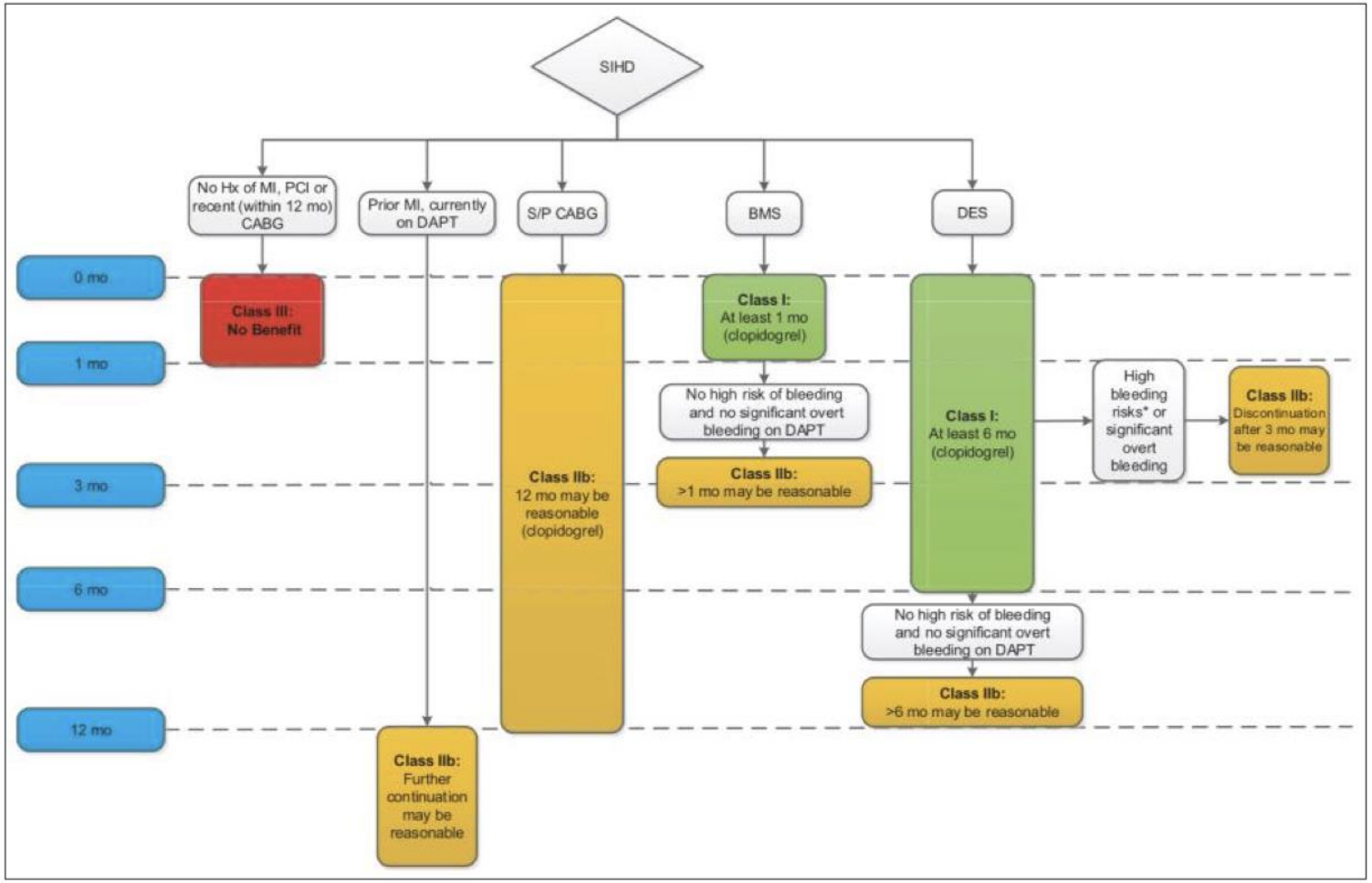
CAD

Khal Salem

Stable Chronic CAD

- A 55-year-old complains of chest pain while playing hockey.
- No ACS.
- Then he had PCI of RCA.
- For how long should he stay on DAPT?

DAPT in SIHD



Source: Levine GN et al. Circulation. 2016;134:e123-55.

- What are the Angina classes?

CCS Angina Class

SEVERITY OF ANGINA

CCS 1/4	Ordinary activity does not cause angina	**≥ 7 METs** • Angina occurs with strenuous or rapid or prolonged exertion only
CCS 2/4	Slight limitation of ordinary activity	**5–7 METs** • ≥ 2 flights of stairs • Walking ≥ 3 blocks on the level (or walking uphill) • Exertion after meals or in cold weather
CCS 3/4	Marked limitation of ordinary activity	**2–5 METs** • ≤ 1 flight of stairs • Walking one or two blocks on the level
CCS 4/4	Inability to carry out any physical activity without discomfort or angina at rest	**< 2 METs** • Any activity • Walking several steps

Definitions of typical and atypical angina. 2021 guidelines

COR	LOE	RECOMMENDATIONS
1	B-NR	1. An initial assessment of chest pain is recommended to triage patients effectively on the basis of the likelihood that symptoms may be attributable to myocardial ischemia (1-7).
1	C-LD	2. Chest pain should not be described as atypical, because it is not helpful in determining the cause and can be misinterpreted as benign in nature. Instead, chest pain should be described as cardiac, possibly cardiac, or noncardiac because these terms are more specific to the potential underlying diagnosis.

Pretest Probability

Age, Years	Chest Pain Criteria 1. Substernal chest discomfort with characteristic quality and duration 2. Provoked by exertion or emotional stress 3. Relieved promptly by rest or nitroglycerin					
	Nonanginal Chest Pain 1 of 3 Criteria		Atypical Angina 2 of 3 Criteria		Typical Angina 3 of 3 Criteria	
	Male	Female	Male	Female	Male	Female
30 – 39	4%	2%	34%	12%	76%	26%
40 - 49	13%	3%	51%	22%	87%	55%
50 - 59	20%	7%	65%	33%	93%	73%
60 - 69	27%	14%	72%	51%	94%	86%

- **Cardiac (Typical)– 3/3 of above**
- **Possibly cardiac (formerly atypical))– 2/3 of above characteristics**
- **Non-cardiac – 1/3 of above characteristics**

CV Risk based on angina classification

Pre-Test Probability of CAD: Stratification by Age/Gender/Symptoms

Age	Non-Anginal Chest Pain		Atypical Angina		Typical Angina	
	Men	Women	Men	Women	Men	Women
30–39	Low	Very Low	Intermediate	Low	Intermediate	Intermediate
40–49	Intermediate	Very Low	Intermediate	Intermediate	High	Intermediate
50–59	Intermediate	Low	Intermediate	Intermediate	High	Intermediate
60–69	Intermediate	Intermediate	Intermediate	Intermediate	High	High
NB: Stress testing most useful when the cause of chest pain is truly uncertain, i.e., pre-test probability 20%–80%						

Source: Fihn SD, Gardin JM, Abrams J, et al. *J Am Coll Cardiol*. 2012;60(24):e44-e164.

- 35-year-old male with 1/3 criteria: 4 % risk
- 35-year-old with 3/3 criteria: 76 % risk
- 55-year-old female with 1/3 criteria: 7 % risk.
- 55 YO Female with 3/3 criteria: 73 % risk

- He exercised on the Treadmill for 6 minutes and had no ST depression. He developed mild angina pain.
- How can risk stratify using a stress test?

Duke Test Score: Risk calculation using treadmill stress test data

DTS: = Exercise time – [5x max ST change] – [4x angina score]

DTS : **6 –(5X 0) – (4 X 1) = 6- 4 = 2**

- **The risk is:**

≥5+ Low Risk: 1 % one-year CV mortality risk.

+4 to -10 Moderate Risk: 1-5% one-year CV mortality risk.

≤ -11 High Risk (5% one-year CV mortality risk)

ACS

ACS

- Three years later, he presented to the ER with chest pain and rise and fall in serum Troponin and pathological Q waves in V5 and V6. His BP was 190/95.
- Scenario 1: An angiogram showed non-occlusive disease. Did he have an MI? If so, what type?
- Scenario 2: If angiogram showed LCX occlusion and patient had DES, and he came back 14 days later with stent thrombosis. Did he have an MI? If so, what type?

Fourth Universal Definition of MI: Criteria for Acute MI

- The term acute MI should be used when there is acute myocardial injury with clinical evidence of acute myocardial ischemia and with detection of a rise and/or fall of cTn values with at least 1 value above the 99th percentile URL and at least 1 of the following:
  - Symptoms of myocardial ischemia
  - New ischemic ECG changes
  - Development of pathological Q waves
  - Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischemic etiology
  - Identification of a coronary thrombus by angiography or autopsy

Source: Thygesen K *et al. Circulation* 2018;138:e618-e651.

Table. Universal Definition of MI

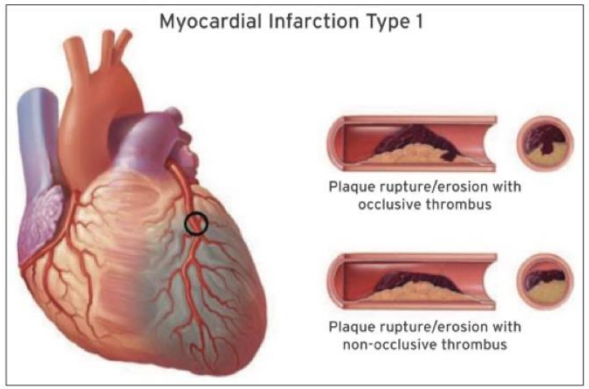
Type	Classification	Clinical and Diagnostic Criteria
1	Spontaneous MI	Plaque rupture, ulceration, fissuring, erosion, or dissection resulting in coronary thrombosis
2	Supply/demand mismatch	Mismatch between myocardial oxygen supply and demand driven by a secondary process other than coronary artery disease
3	Suspected MI-related death	Cardiac death in a setting suggestive of ischemic process without definitive cardiac biomarker evidence of MI
4a	PCI-related MI	Rise in cardiac biomarkers accompanied by symptoms, electrocardiographic, angiographic, or imaging evidence of ischemia after PCI
4b	Stent thrombosis	Confirmed stent thrombosis in context of ischemia and dynamic cardiac biomarker changes
5	CABG-related MI	Rise in cardiac biomarkers accompanied by electrocardiographic, angiographic, or imaging evidence of ischemia after CABG

Abbreviations: CABG, coronary artery bypass graft; MI, myocardial infarction; PCI, percutaneous coronary intervention.

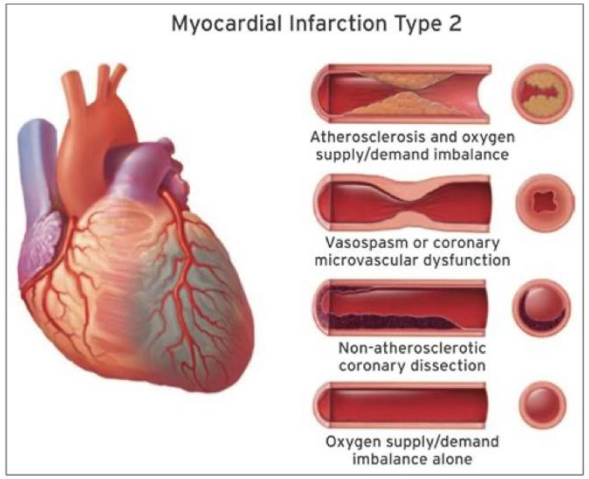
Typ1 and type 2 MI

Fourth Universal Definition of MI: Criteria for Acute MI Type 1 and 2

- *Type 1 MI.* Plaque disruption or coronary thrombosis



- *Type 2 MI.* Imbalance between myocardial oxygen supply and demand unrelated to acute atherothrombosis



Source: Thygesen K *et al. Circulation* 2018;138:e618-e651.

- He was admitted to the hospital.
- How to calculate the risk of hospital mortality and the 14-day risk of complications and revascularization.
- Consideration for early or delayed invasive intervention.

Invasive startegy

- AHA/ACC Coronary revasc 2021

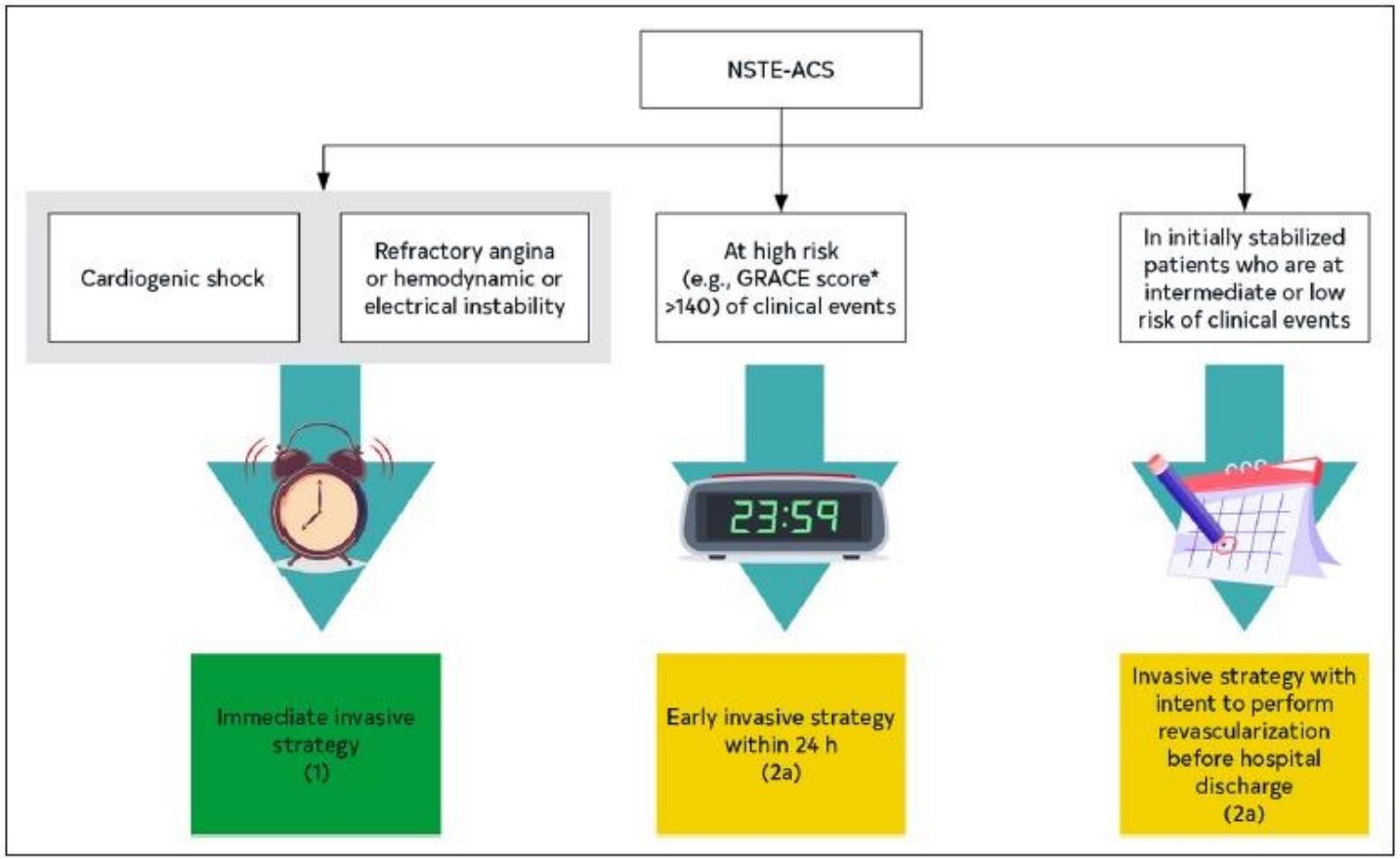
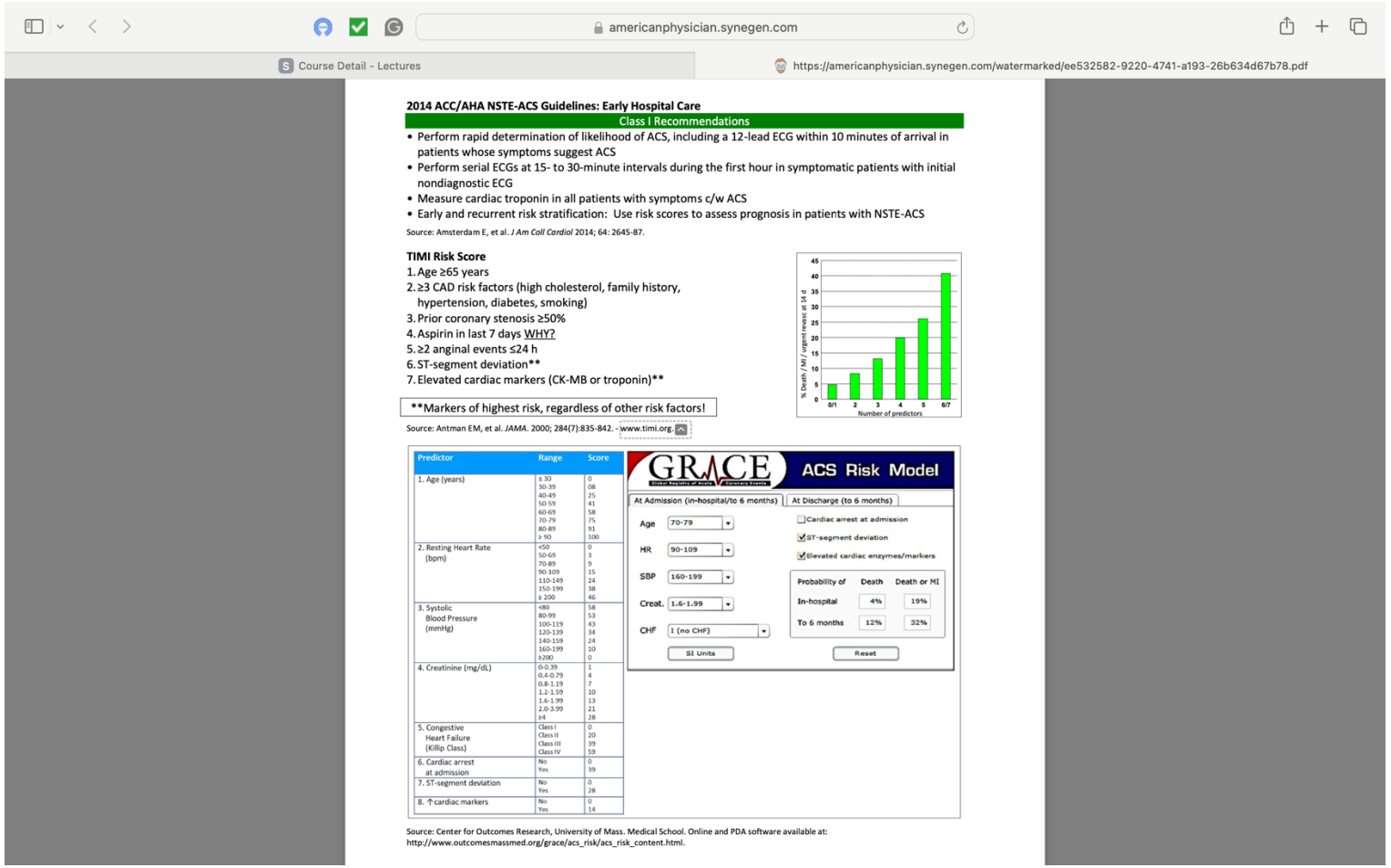
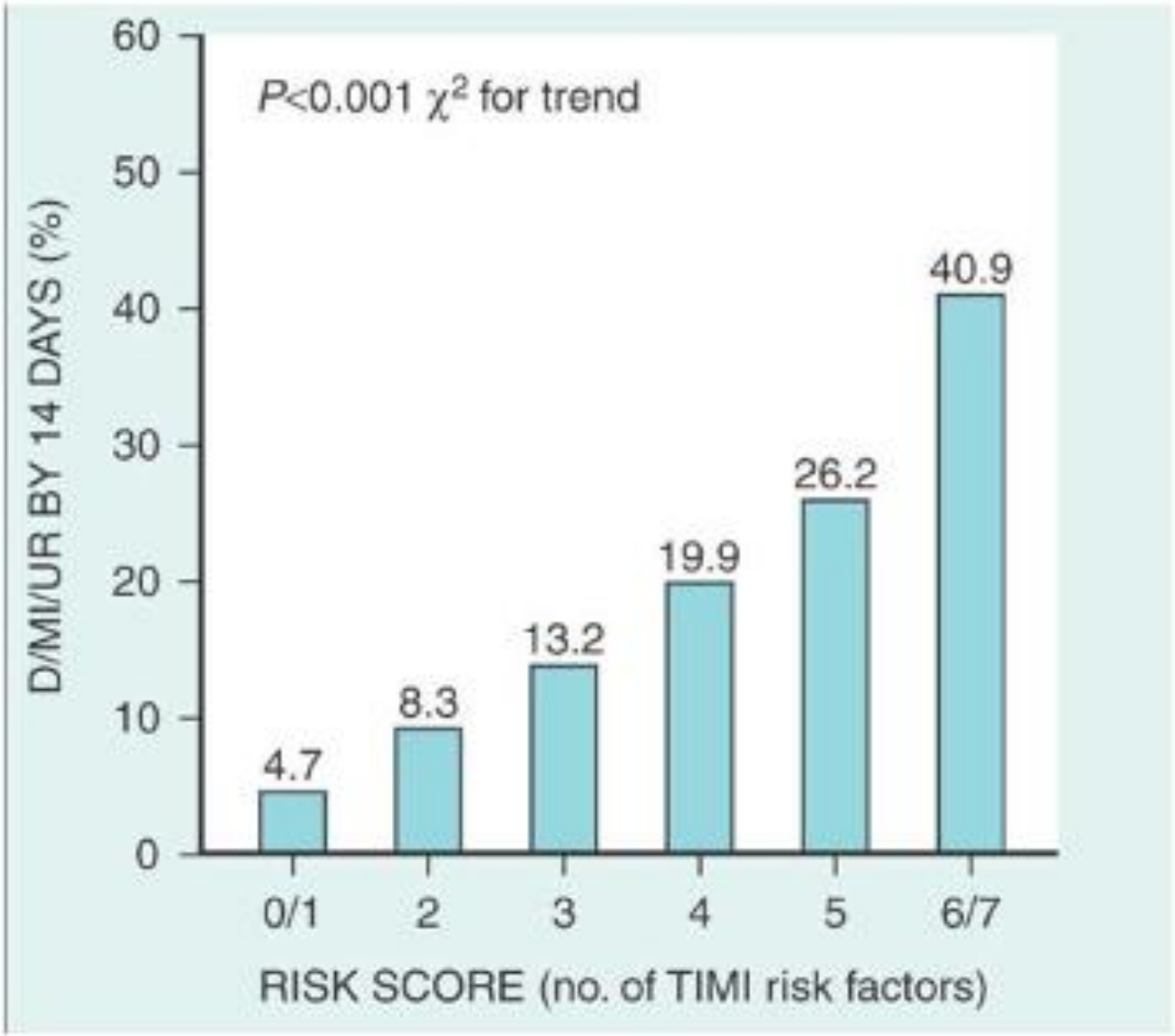


Figure 5. Recommendations for the Timing of Invasive Strategy in Patients With NSTE-ACS.
Colors correspond to Table 2.
GRACE indicates Global Registry of Acute Coronary Events; and NSTE-ACS, non–ST-segment–elevation acute coronary syndrome.
*https://www.mdcalc.com/grace-acs-risk-mortality-calculator.[31] This algorithm summarizes the recommendations in this guideline for coronary artery angiography with the intent to perform revascularization in NSTE-ACS. It is not meant to encompass every patient scenario or situation, and clinicians are encouraged to use a Heart Team approach when care decisions are unclear and to see the accompanying supportive text for each recommendation. Additionally, in situations that lack sufficient data to make formal recommendations for care, please see Section 17, "Unanswered Questions and Future Directions."

Hospital care



TIMI risk score



Grace and TIMI in NTEMI

TIMI SCORE	GRACE SCORE
1) Age > 65 years 2) ≥ 3 risk factors for CAD 3) Known stenosis > 50% 4) ASA < 7 days 5) ≥ 2 episodes of retrosternal chest pain x 24 h 6) ST depression ≥ 0.5 mm 7) Biomarkers **Death / MI / Urgent revascularization within 14 days** 0-1 point.....4.7 % 2 points .....8.3 % 3 points .....13.2 % 4 points .....19.9 % 5 points .....26.2 % 6-7 points.....40.9 %	www.gracescore.org 1) Age 2) SBP 3) HR 4) Killip Class 5) Heart failure 6) Cardiac arrest at presentation 7) ST segment depression 8) Biomarkers 9) Creatinine **In–hospital mortality** GRACE ≤ 108 points.....< 1 % GRACE 109-140 points .....1-3 % GRACE > 140 points .....> 3 %

Grace Risk Score

Risk category (tertile)	GRACE risk score	In-hospital death (%)
Low	≤108	<1
Intermediate	109–140	1–3
High	>140	>3

Risk category (tertile)	GRACE risk score	Post-discharge to 6-month death (%)
Low	≤88	<3
Intermediate	89–118	3-8
High	>118	>8

Grace Score and Early invasive decision

2014 ACC/AHA NSTE-ACS Guidelines

Timing of Angiography with an Early Invasive Strategy

CLASS 1, LOA A. An urgent/immediate invasive strategy is indicated in patients with NSTE-ACS with refractory angina or hemodynamic or electrical instability.

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
Early invasive (within 24 h)	None of the above, but GRACE risk score >140
	Temporal 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

Source: Amsterdam E, et al. *J Am Coll Cardiol* 2014; 64: 2645-87.

TIMI in STEMI

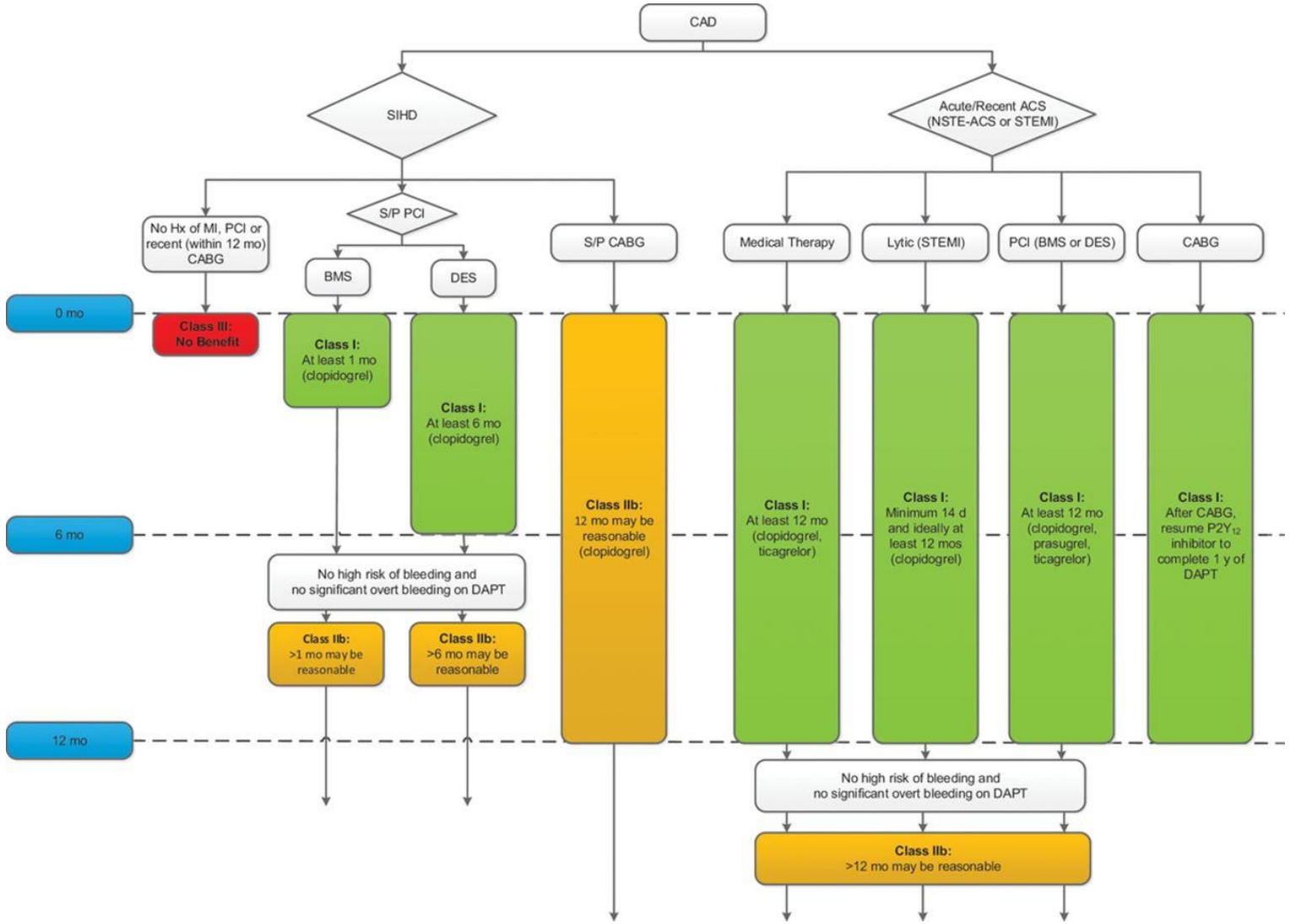
KILLIP CLASSIFICATION

Killip I	No crackles; No S3
Killip II	Crackles (< 50% of lung fields) ± S3
Killip III	Crackles > 50% of lung fields
Killip IV	Cardiogenic shock

TIMI SCORE IN STEMI

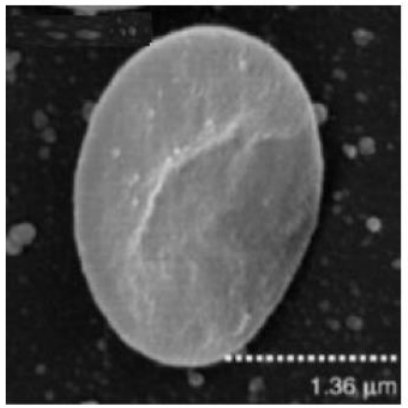
CRITERIA	# POINTS	30-DAY MORTALITY
Age 65-74 years or ≥ 75 years	2 or 3 points	**0 : 0.8 %**
SBP < 100	3 points	**1 : 1.6 %**
HR > 100 bpm	2 points	**2 : 2.2 %**
Killip II-III-IV	2 points	**3 : 4.4 %**
History: DM or HTN or Angina	1 point	**4 : 7.3 %**
< 67 kg	1 point	**5 : 12.4 %**
Time to treatment > 4 h	1 point	**6 : 16.1 %**
Anterior STEMI or LBBB	1 point	**7 : 23.4 %**
		8 : 26.8 %
		> 8 : 35.9 %

Antiplatelet therapy post PCI Vs SIHD



Platelet Activation

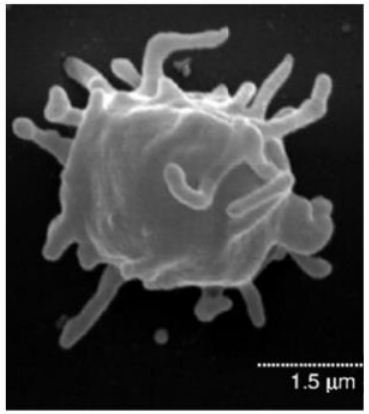
Plaque Rupture Releases Potent Platelet Activators That Produce a Dramatic Shape Change



Quiescent Platelet

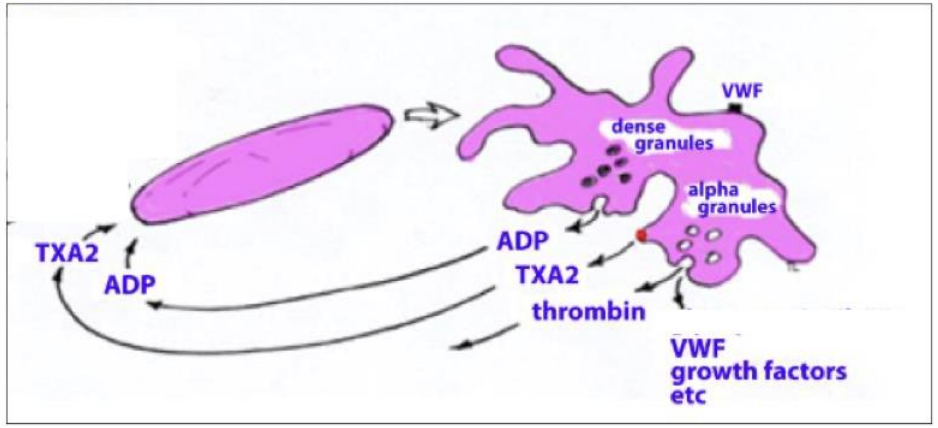
Platelet Activators

ADP
Collagen
Tissue Factor
Thrombin
Thromboxane A_2



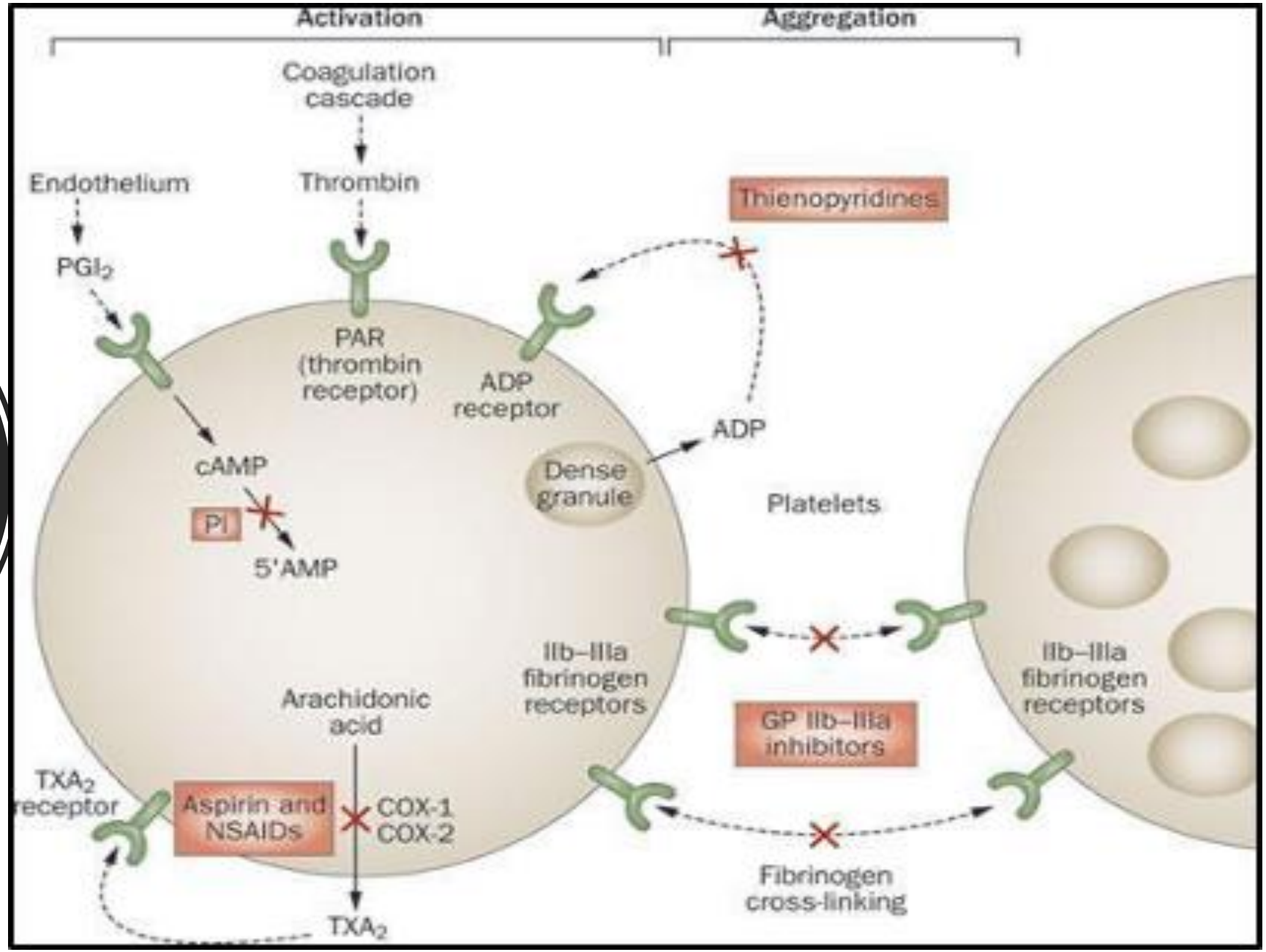
Activated Platelet

Amplification of Platelet Activation



Activated platelets release granule contents (ADP, thrombin, thromboxane) that amplify the process

Antiplatelets



ANTIPLATELET DRUG COMPARISON CHART

Drug	ASA	Clopidogrel (Plavix®)	Prasugrel (Effient®)	Ticagrelor (Brilinta®)
Indications	- 1°and 2°prevention of stroke and MI - ACS - PCI with stent - PVD	- ASA intolerance or failure - 1°and 2°prevention of stroke and MI (+/- ASA) - ACS (+ ASA) - PCI (+ ASA) - PVD	- With ASA, for treatment of ACS in patients treated with PCI **Contraindicated if:** age > 75 years; **OR** wt < 60 kg; **OR** history of stroke **NON-FORMULARY**	- With ASA, for treatment of ACS See BCHA restrictions below[1]
Dose and Duration	Load: 160-325 mg Maintenance: 80 or 81 mg daily Duration: Indefinite	Load: 300-600 mg Maintenance: 75 mg daily Duration: ACS: up to 1 year BMS: minimum 30 days DES: minimum 1 year	Load: 60 mg Maintenance: 10 mg daily Duration: up to 1 year	Load: 180 mg Maintenance: 90 mg BID Duration: up to 1 year
Class	Non-Steroidal Anti-Inflammatory Agent	Second generation thienopyridine (Prodrug)	Third-generation thienopyridine (Prodrug)	Cyto-pentyl-triazolo-pyrimidine
Mechanism of Platelet Inhibition	Irreversible inhibitor of COX-1 causing decrease in thromboxane A_2	Irreversible inhibitor of $P2Y_{12}$ component of ADP receptor (preventing ADP binding and activation of platelets)	Irreversible inhibitor of $P2Y_{12}$ component of ADP receptor (preventing ADP binding and activation of platelets)	Reversibly modifies $P2Y_{12}$ component of ADP receptor (preventing ADP binding and activation of platelets)
Oral Bioavailability	50-75%	> 50% (active metabolite)	> 78% (active metabolite)	30-42%
Peak Effect	1-3 hours	6 hours (after load)	4 hours (after load)	2 hours (after load)
Half-life (active metabolite)	3 hrs (salicylate)	0.5 hrs	7 hrs (range 2-15 hrs)	9 hrs (range 6.7-9.1 hrs)
Elimination	Hydrolyzed by esterases; Hepatic conjugation	Esterases; Metabolism by CYP-450 enzymes	Esterases; Metabolism by CYP-450 enzymes	Metabolism by CYP-450 enzymes
CYP Metabolism	No	CYP2C19	CYP3A4, CYP2B6	CYP3A4/5
When to Hold Dose Prior to Surgery	7 days (optional)	5-7 days	7 days	5 days

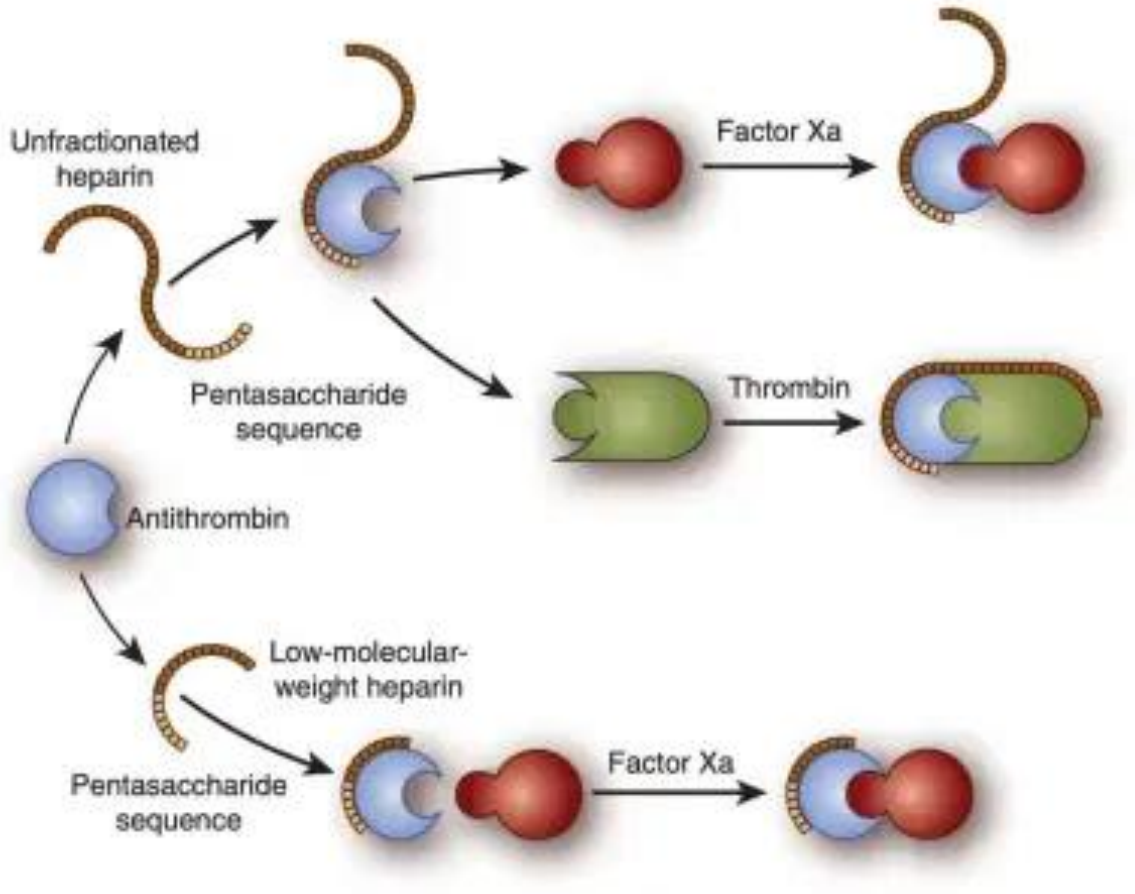
[1] Ticagrelor restricted to patients on prior to admission **or** those on ASA with ACS i.e. STEMI, non-STEMI, unstable angina (UA) **AND one of the following:**
Failure on optimal doses of clopidogrel and ASA therapy **or** recurrent ACS after revascularization with PCI; **OR**
STEMI **and** undergoing revascularization via PCI; **OR**
Non-STEMI or UA **and** high risk angiographic anatomy **and** undergoing revascularization via PCI

Abbreviations:
ACS = Acute Coronary Syndrome; PCI = Percutaneous Coronary Intervention; PVD = Peripheral Vascular Disease;
BMS = Bare Metal stent; DES = Drug-Eluting Stent; ADP = Adenosine Diphosphate

Routine therapies in the acute, subacute, and long-term phases: beta-blockers, angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, mineralocorticoid receptor antagonists, and lipid-lowering treatments after ST-elevation myocardial infarction

Recommendations	Class[a]	Level[b]
Beta-blockers		
Oral treatment with beta-blockers is indicated in patients with heart failure and/or LVEF ≤40% unless contraindicated.[357–361]	I	A
Intravenous beta-blockers should be considered at the time of presentation in patients undergoing primary PCI without contraindications, with no signs of acute heart failure, and with an SBP >120 mmHg.[346–348,350,403]	IIa	A
Routine oral treatment with beta-blockers should be considered during hospital stay and continued thereafter in all patients without contraindications.[344,354–356,404,405]	IIa	B
Intravenous beta-blockers must be avoided in patients with hypotension, acute heart failure or AV block, or severe bradycardia.[344]	III	B
Lipid lowering therapies		
It is recommended to start high-intensity statin therapy[c] as early as possible, unless contraindicated, and maintain it long-term.[364,366,368]	I	A
An LDL-C goal of < 1.8 mmol/L (70 mg/dL) or a reduction of at least 50% if the baseline LDL-C is between 1.8–3.5 mmol/L (70–135 mg/dL) is recommended.[367,369,376,382]	I	B
It is recommended to obtain a lipid profile in all STEMI patients as soon as possible after presentation.[369,406]	I	C
In patients with LDL-C ≥1.8 mmol/L (≥70 mg/dL) despite a maximally tolerated statin dose who remain at high risk, further therapy to reduce LDL-C should be considered.[376,382]	IIa	A
ACE inhibitors/ARBs		
ACE inhibitors are recommended, starting within the first 24 h of STEMI in patients with evidence of heart failure, LV systolic dysfunction, diabetes, or an anterior infarct.[383]	I	A
An ARB, preferably valsartan, is an alternative to ACE inhibitors in patients with heart failure and/or LV systolic dysfunction, particularly those who are intolerant of ACE inhibitors.[396,407]	I	B
ACE inhibitors should be considered in all patients in the absence of contraindications.[394,395]	IIa	A
MRAs		
MRAs are recommended in patients with an LVEF ≤40% and heart failure or diabetes, who are already receiving an ACE inhibitor and a beta-blocker, provided there is no renal failure or hyperkalaemia.[397]	I	B

Heparin and LMWH

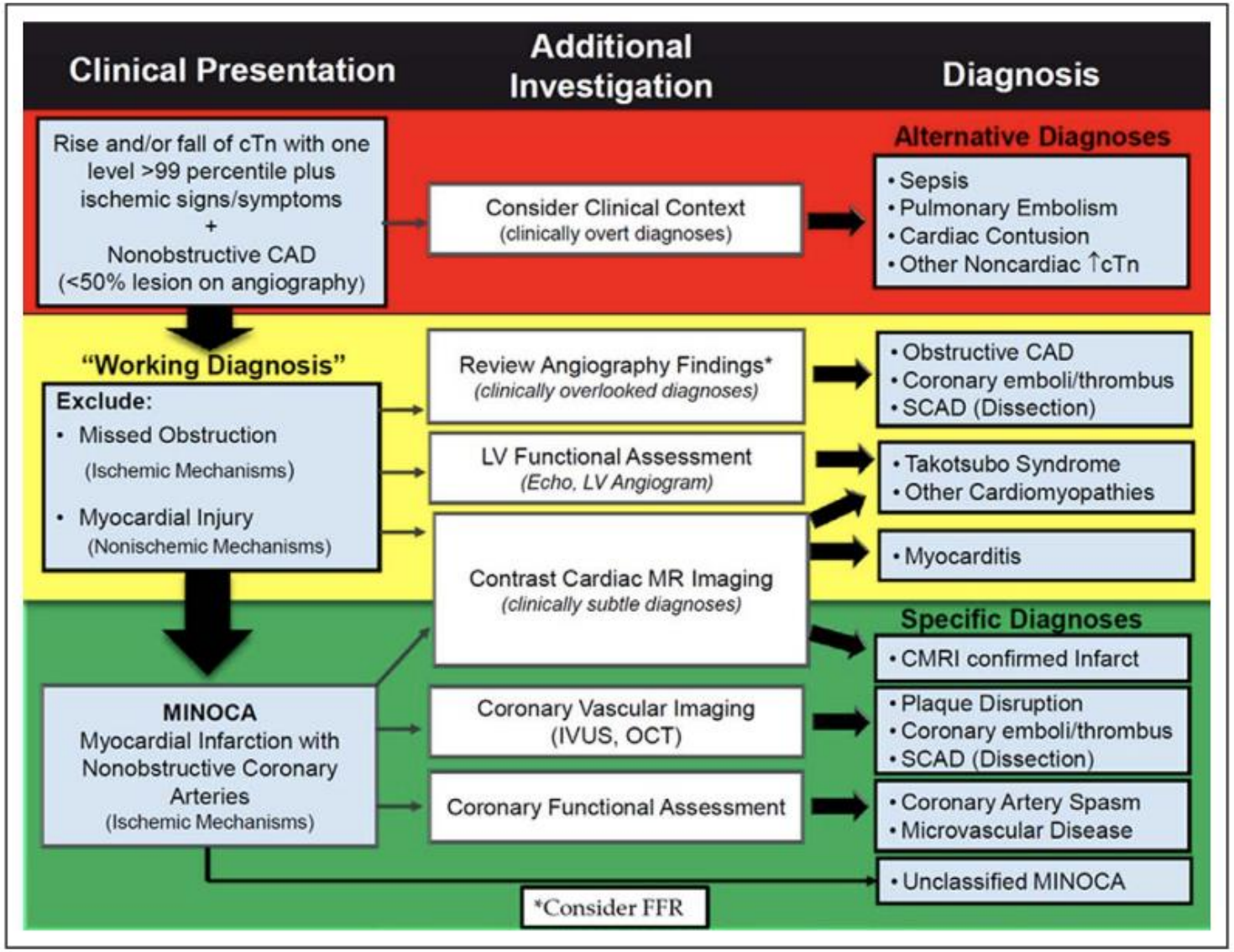


Heparin functions by activating antithrombin-III to bind to serine proteases involved in coagulation, blocking their function (predominantly Factor Xa and Factor IIa a.k.a. thrombin). Low molecular heparin predominantly facilitates inhibition of Xa, whereas unfractionated heparin inhibits Xa and thrombin. This reaction consumes antithrombin-III (which remains permanently stuck to factor Xa and/or thrombin).

Lai S et al. Kidney International 2013

Non obstructive CAD

MINOCA



1. Clinical algorithm for the diagnosis of MINOCA.

SCAD

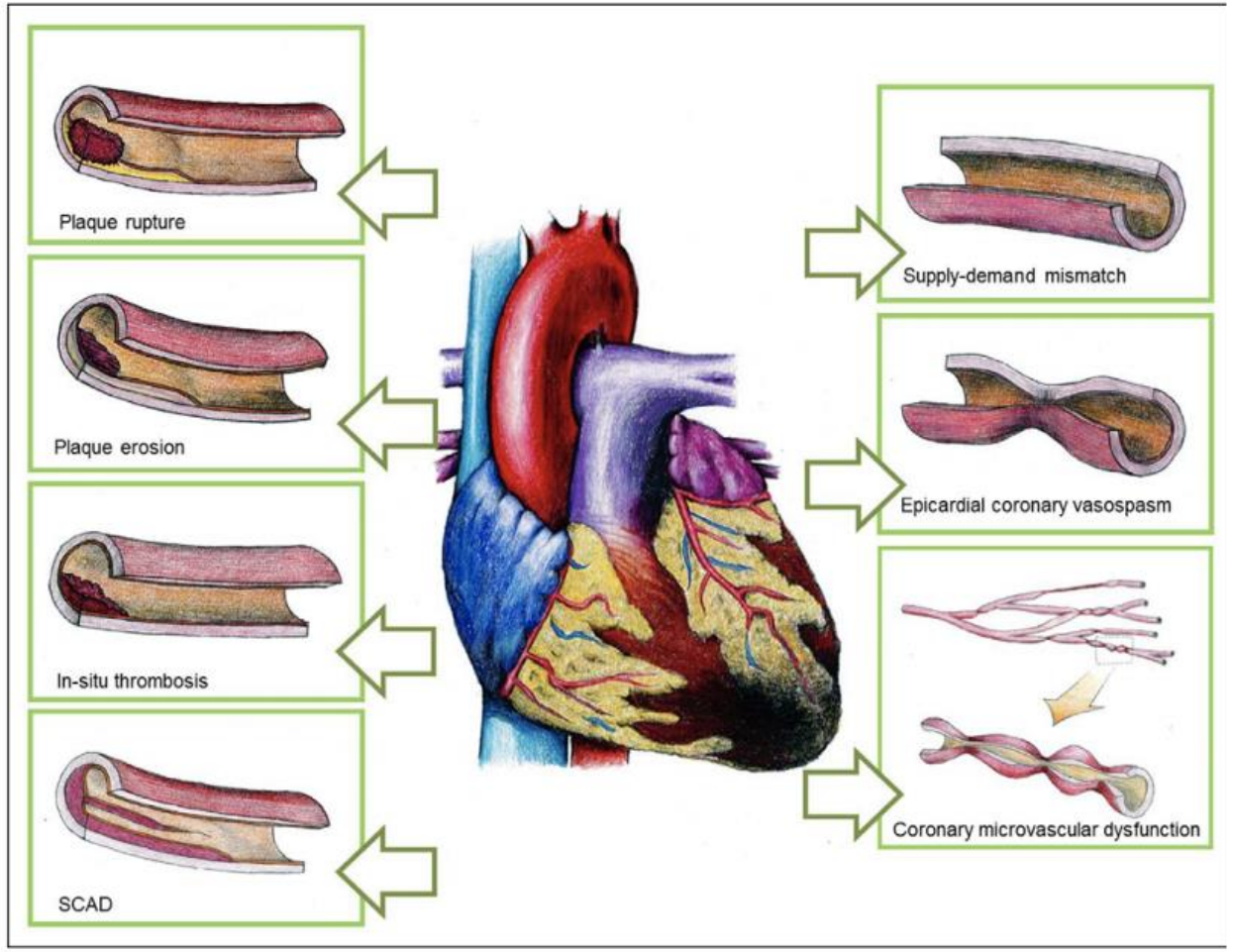


Figure 2. Specific causes.
SCAD indicates spontaneous coronary artery dissection.

SCAD

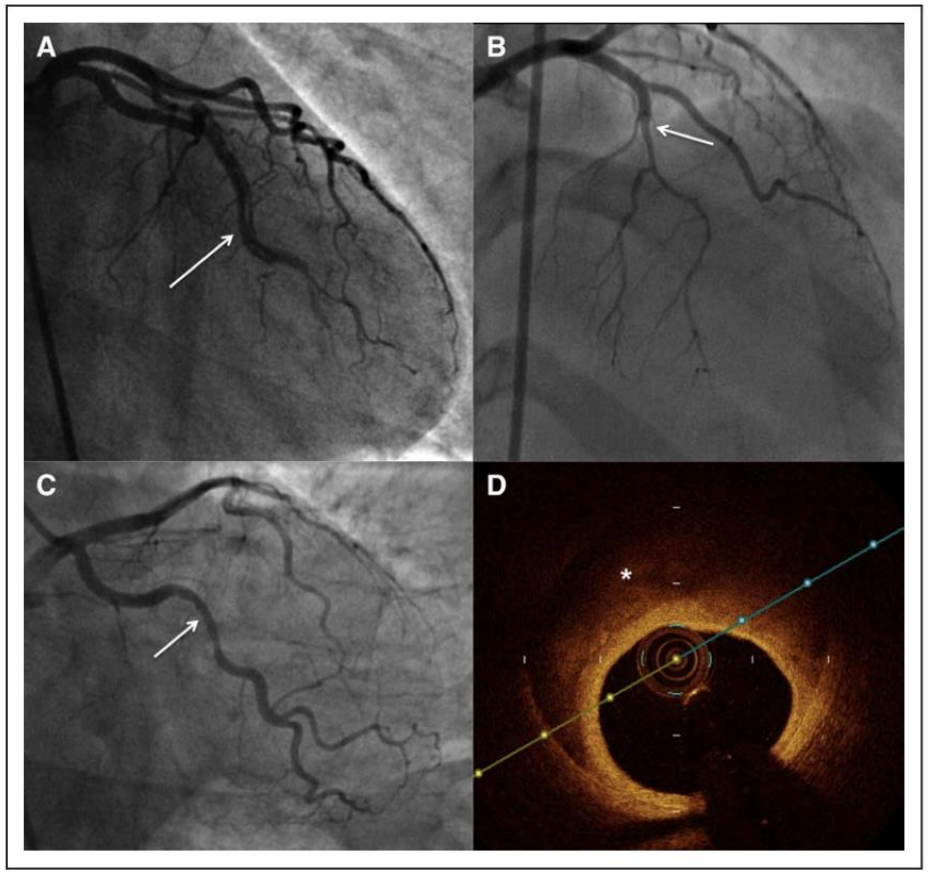
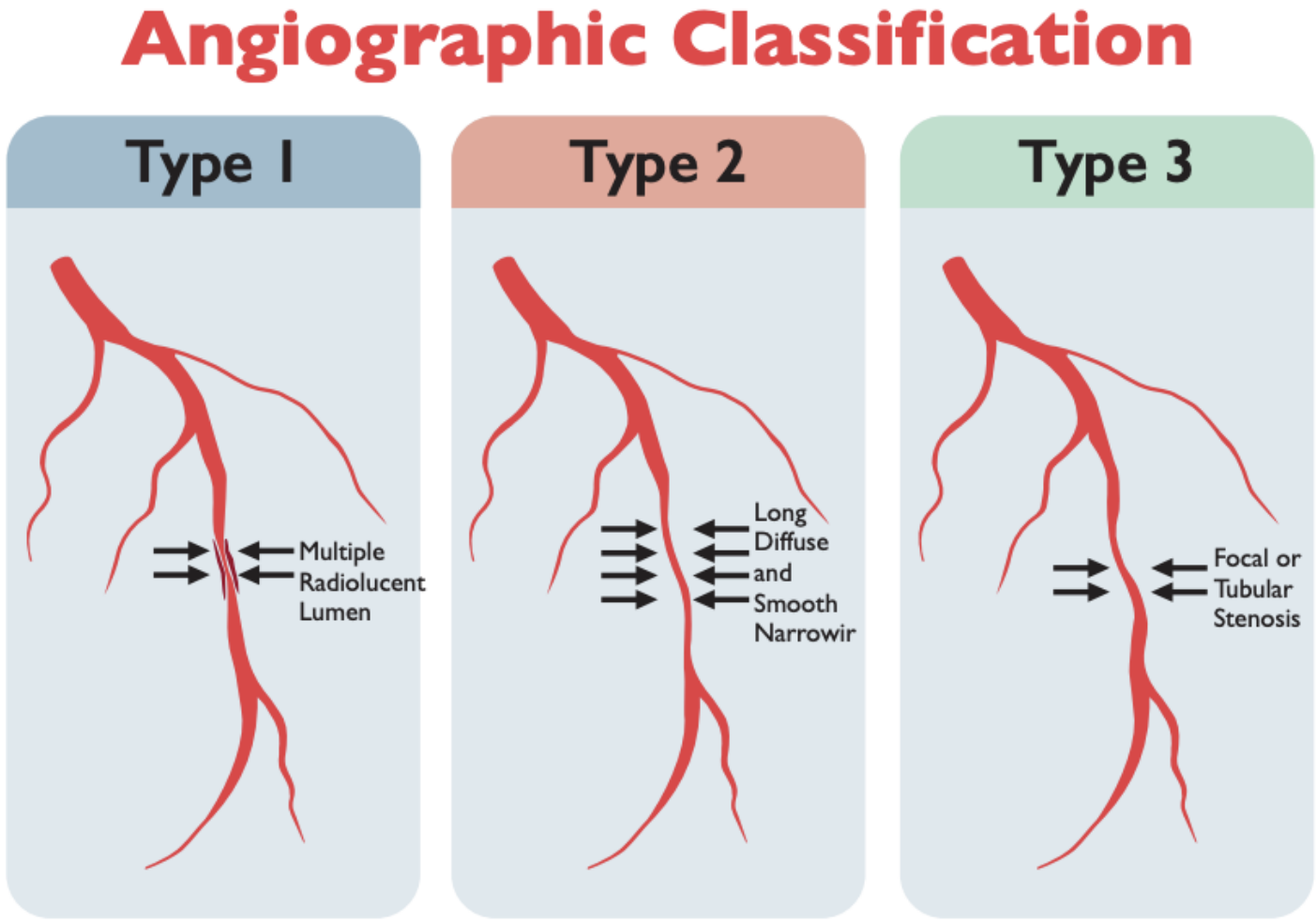


Figure 5. Angiographic features of spontaneous coronary artery dissection.
A, Type 1, multiple radiolucent lumens (arrow) or arterial wall contrast staining. **B**, Type 2, diffuse stenosis that can be of varying severity and length (dissection starting from arrow). **C**, Type 3: focal or tubular stenosis (arrow), usually <20 mm in length, that mimics atherosclerosis. Intracoronary imaging should be performed to confirm the presence of intramural hematoma or multiple lumens. **D**, Optical coherence tomography in type 3 (**C**) shows intramural hematoma (asterisk).

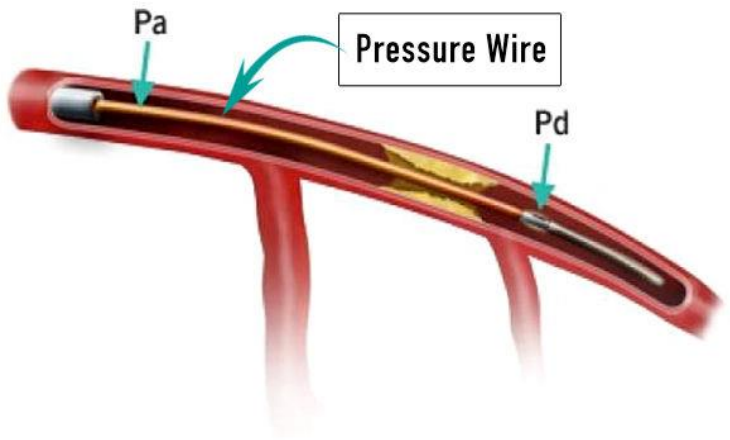
SCAD


Angiographic Classification
Type I
Multiple
Radiolucent
Lumen
Type 2
Long
Diffuse
and
Smooth
Narrowir
Type 3
Focal or
Tubular
Stenosis

FFR

$$FFR = \frac{\text{Distal Coronary Pressure (Pd)}}{\text{Proximal Coronary Pressure (Pa)}}$$

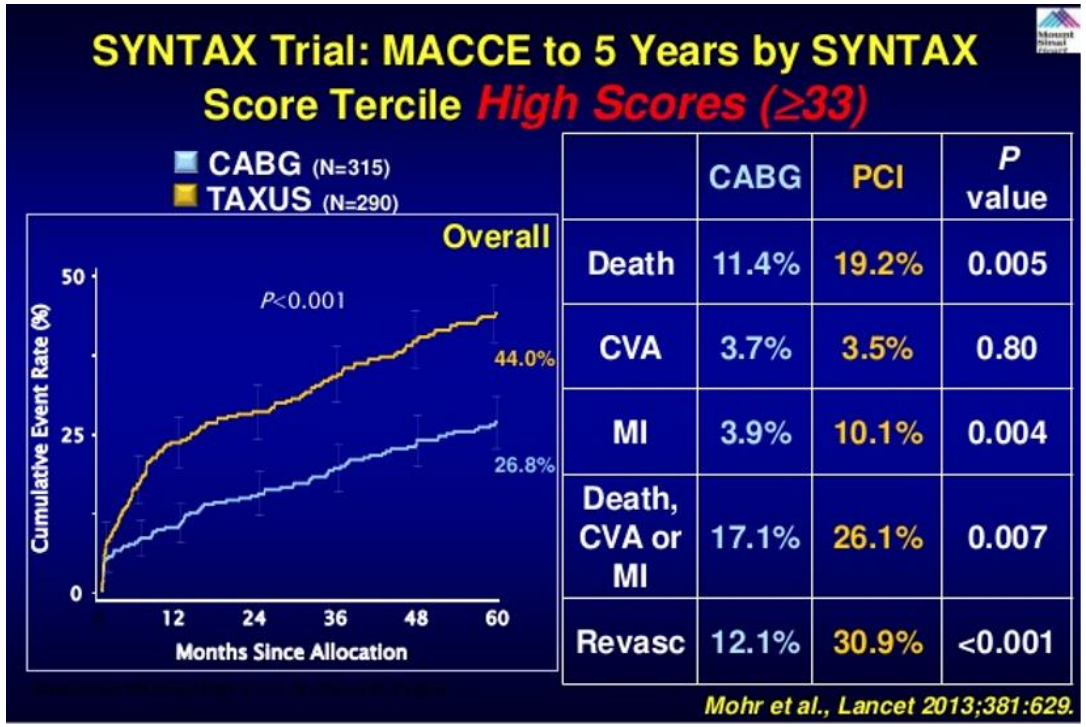
(During Maximum Hyperemia)



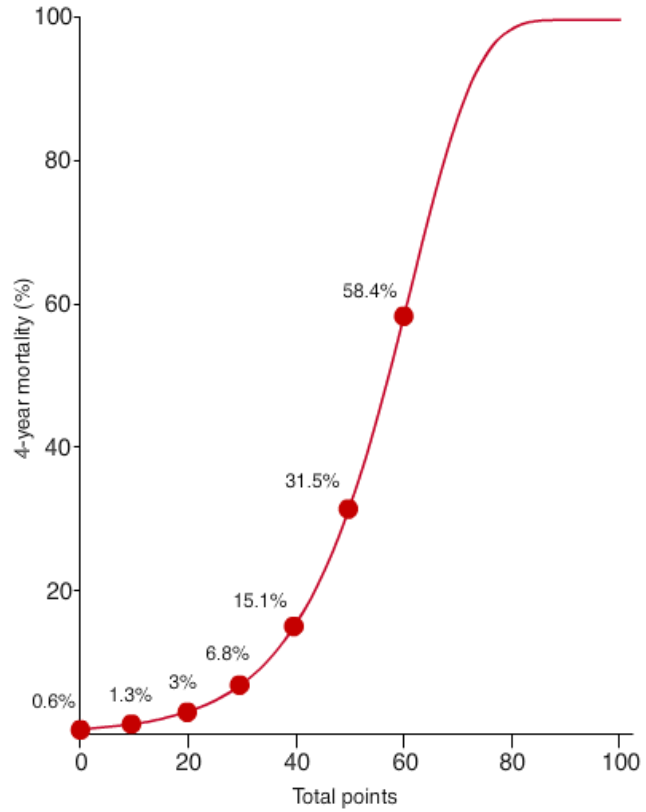
- What is The Syntax score?

Syntax score

Syntax Score



	CABG	PCI	*P* value
Death	11.4%	19.2%	0.005
CVA	3.7%	3.5%	0.80
MI	3.9%	10.1%	0.004
Death, CVA or MI	17.1%	26.1%	0.007
Revasc	12.1%	30.9%	<0.001



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THANK YOU