PRIMARY PCI IN STEMI

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- Given the progressive loss of functioning myocytes with persistent occlusion of the infarct-related artery in STEMI , initial management aims to restore blood flow to the infarct zone as rapidly as possible.

- Primary PCI is generally the preferred option, provided that an experienced operator and team.

- The “chain of survival” for STEMI involves a highly integrated strategy beginning with patient education about the symptoms of MI and early contact with the medical system, coordination of destination protocols in emergency medical service (EMS) systems, efficient practices in emergency departments to shorten door-to-reperfusion time, and expeditious implementation of the reperfusion strategy by a trained team

- Most deaths associated with STEMI occur within the first hour of its onset and usually result from ventricular fibrillation (VF).Therefore, immediate implementation of resuscitative efforts and rapid transportation of the patient to a hospital have prime importance.

- Major components of the time from the onset of ischemic symptoms to reperfusion include :

(1) the time for the patient to recognize the problem and seek medical attention;

(2) prehospital evaluation, treatment, and transportation;

(3) the time for diagnostic measures and initiation of treatment in the hospital (e.g., “door-to-needle” time for patients receiving a fibrinolytic agent and “door-to-device” time for patients undergoing a catheter-based reperfusion strategy)

(4) the time from initiation of treatment to restoration of flow

- Patient-related factors that correlate with a longer delay until deciding to seek medical attention include older age; female sex; black race; low socioeconomic or uninsured status; history of angina, diabetes, or both; consulting a spouse or other relative; and consulting a physician.

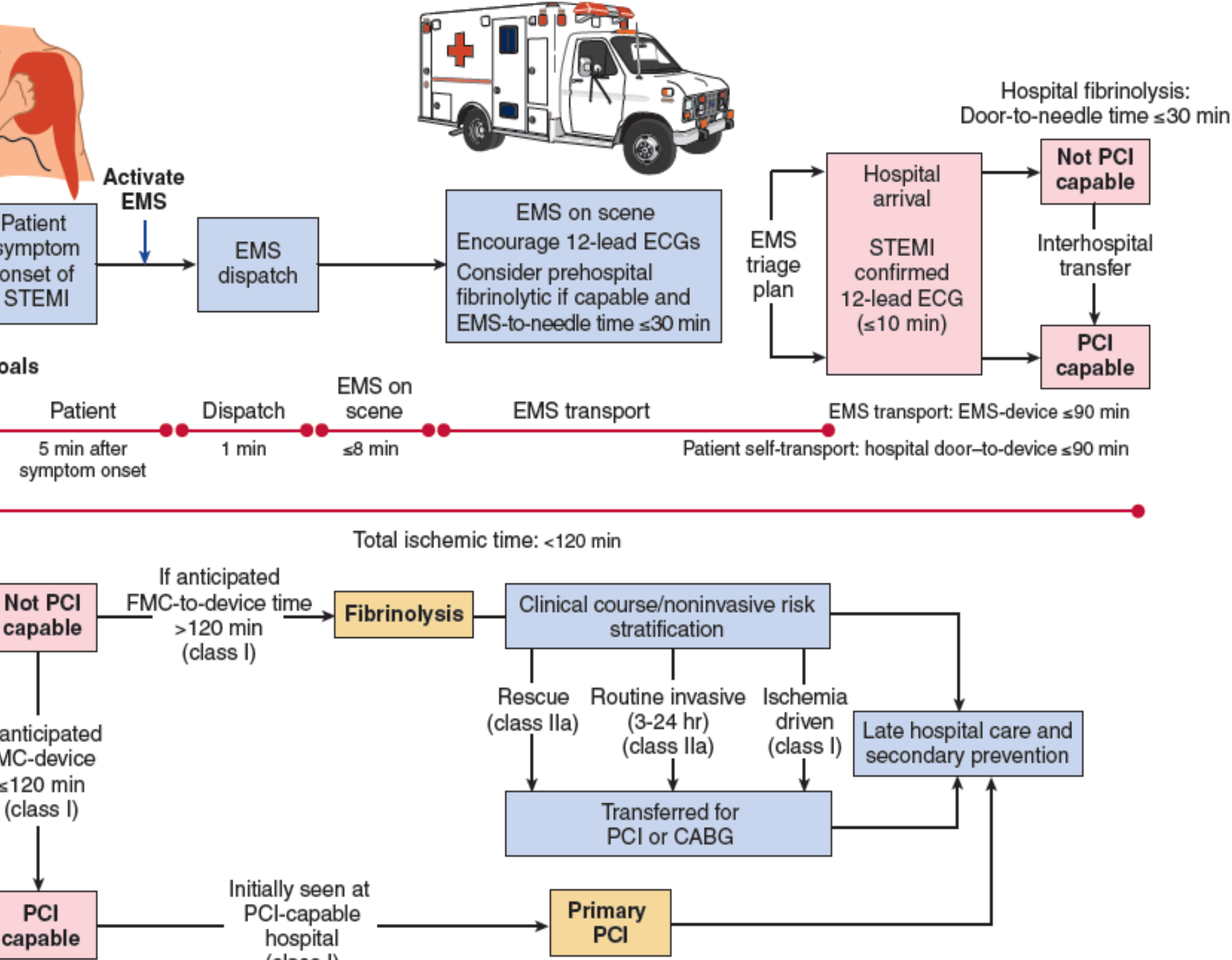

Activate EMS
Patient symptom onset of STEMI
EMS dispatch
EMS on scene
Encourage 12-lead ECGs
Consider prehospital fibrinolytic if capable and EMS-to-needle time ≤30 min
EMS triage plan
Hospital arrival
STEMI confirmed 12-lead ECG (≤10 min)
Hospital fibrinolysis: Door-to-needle time ≤30 min
Not PCI capable
Interhospital transfer
PCI capable
oals
Patient
5 min after symptom onset
Dispatch
1 min
EMS on scene
≤8 min
EMS transport
EMS transport: EMS-device ≤90 min
Patient self-transport: hospital door–to-device ≤90 min
Total ischemic time: <120 min
Not PCI capable
If anticipated FMC-to-device time >120 min (class I)
Fibrinolysis
Clinical course/noninvasive risk stratification
Rescue (class IIa)
Routine invasive (3-24 hr) (class IIa)
Ischemia driven (class I)
Late hospital care and secondary prevention
Transferred for PCI or CABG
anticipated MC-device ≤120 min (class I)
PCI capable
Initially seen at PCI-capable hospital
Primary PCI

TABLE 59.1 Criteria for a System of Care for ST-Elevation Myocardial Infarction (STEMI)

1. The system should be registered with Mission: Lifeline.
2. Ongoing multidisciplinary team meetings should occur, including EMS, non-PCI hospitals/STEMI referral centers, and PCI hospitals/STEMI receiving centers, to evaluate outcomes and quality improvement data. Operational issues should be reviewed, problems identified, and solutions implemented.
3. Each STEMI system should include a process for prehospital identification and activation, destination protocols to STEMI receiving centers, and transfer for patients who arrive at STEMI referral centers and are primary PCI candidates, are ineligible for fibrinolytic therapy, and/or are in cardiogenic shock.
4. Each system should have a recognized system coordinator, physician champion, and EMS medical director.
5. Each system component (EMS, STEMI referral centers, and STEMI receiving centers) should meet the appropriate criteria.

TABLE 59.2 Interventions to Improve Door-to-Device Times

1. A prehospital ECG for diagnosing STEMI is used to activate the PCI team while the patient is en route to the hospital.
2. Emergency physicians activate the PCI team.
3. A single call to a central page operator activates the PCI team.
4. A goal is set for the PCI team to arrive at the catheterization laboratory within 20 minutes after being paged.
5. Timely data feedback and analysis are provided to members of the STEMI care team.

- A history of ischemic-type discomfort and the initial 12-lead ECG (≤10 minutes after hospital arrival) are the primary tools for screening patients with possible acute coronary syndrome (ACS) for STEMI.
- Because lethal arrhythmias can occur suddenly in patients with STEMI, all patients should have bedside monitoring of the ECG and intravenous (IV) access.
- The presence of ST-segment elevation on the ECG in a patient with ischemic discomfort highly suggests thrombotic occlusion of an epicardial coronary artery and should trigger a well-rehearsed sequence of rapid assessment of the patient for initiation of a reperfusion strategy.

Pay Attention to figure of ECG in Post MI

- a door-to-needle time of 30 minutes or less for initiation of fibrinolytic therapy and a door-to-device time of 90 minutes or less for percutaneous coronary perfusion

Each 30-minute delay from symptom onset to PCI increases the relative risk (RR) for 1-year mortality by 8%.

TABLE 59.3 Contraindications to and Cautions in the Use of Fibrinolytics for Treating ST-Elevation Myocardial Infarction*

Absolute Contraindications
Any previous intracranial hemorrhage
Known structural cerebral vascular lesion (e.g., arteriovenous malformation)
Known malignant intracranial neoplasm (primary or metastatic)
Ischemic stroke within 3 months *except* acute ischemic stroke within 4.5 hours
Suspected aortic dissection
Active bleeding or bleeding diathesis (excluding menses)
Significant closed-head or facial trauma within 3 months
Intracranial or intraspinal surgery within 2 months
Severe uncontrolled hypertension (unresponsive to emergency therapy)
For streptokinase, previous treatment within the previous 6 months
Relative Contraindications
History of chronic, severe, poorly controlled hypertension
Significant hypertension at initial evaluation (SBP >180 mm Hg or DBP >110 mm Hg)†
History of previous ischemic stroke >3 months
Dementia
Known intracranial pathology not covered in Absolute Contraindications
Traumatic or prolonged (>10 minutes) cardiopulmonary resuscitation
Major surgery (<3 weeks)
Recent (within 2 to 4 weeks) internal bleeding
Noncompressible vascular punctures
Pregnancy
Active peptic ulcer
Oral anticoagulant therapy

CATHETER-BASED REPERFUSION STRATEGIES

- Catheter-based strategies can also achieve reperfusion of the infarct artery. This approach has evolved from passage of a balloon catheter over a guide wire in the culprit vessel only to now include potent oral antiplatelet therapy, multiple options for anticoagulants, and coronary stents, with the possibility of multi vessles revascularization.

- PCI used as primary reperfusion therapy in patients with STEMI.

Antiplatelet Therapy

Aspirin

• 162- to 325-mg loading dose before the procedure	I
• 81- to 325-mg daily maintenance dose (indefinite)*	I
• 81 mg daily is the preferred maintenance dose*	IIa

$P2Y_{12}$ Inhibitors

Loading Doses

• Clopidogrel: 600 mg as early as possible or at the time of PCI	I
• Prasugrel: 60 mg as early as possible or at the time of PCI	I
• Ticagrelor: 180 mg as early as possible or at the time of PCI	I

Maintenance Doses

Drug-eluting stents (DESs) placed: continue therapy for 1 yr with:

• Clopidogrel: 75 mg daily	I
• Prasugrel: 10 mg daily	I
• Ticagrelor: 90 mg twice a day	I

Bare-metal stents (BMSs)† placed: continue therapy for 1 yr with:

• Clopidogrel: 75 mg daily	I
• Prasugrel: 10 mg daily	I
• Ticagrelor: 90 mg twice a day	I

DESs placed

• Patients with STEMI and previous stroke or TIA: prasugrel	III: Harm

Intravenous Glycoprotein IIb/IIIa Receptor Antagonists in Conjunction with Unfractionated Heparin or Bivalirudin in Selected Pa

• Abciximab: 0.25-mg/kg IV bolus, then 0.125 µg/kg/min (maximum, 10 µg/min)	IIa
• Tirofiban (high bolus dose): 25-µg/kg IV bolus, then 0.15 µg/kg/min • In patients with CrCl <30 mL/min, reduce the infusion by 50%	IIa

• Eptifibatide (double bolus): 180-µg/kg IV bolus, then 2 µg/kg/min; a second 180-µg/kg bolus is administered 10 min after the first bolus
  • In patients with CrCl <50 mL/min, reduce the infusion by 50%
  • Avoid in patients on hemodialysis
	IIa

• Pre–catheterization laboratory administration of IV GP IIb/IIIa receptor antagonist	IIb
• Intracoronary abciximab: 0.25-mg/kg bolus	IIb

Anticoagulant Therapy

• Unfractionated heparin (UFH) • With a GP IIb/IIIa receptor antagonist planned: 50- to 70-unit/kg IV bolus to achieve therapeutic ACT‡	I
   • With no GP IIb/IIIa receptor antagonist planned: 70- to 100-unit/kg bolus to achieve a therapeutic ACT§	I
• Bivalirudin: 0.75-mg/kg IV bolus, then 1.75-mg/kg/hr infusion with or without previous treatment with UFH. An additional bolus of 0.3 mg/kg may be given if needed • Reduce the infusion to 1 mg/kg/hr with estimated an CrCl <30 mL/min	I
   • Preferred over UFH with a GP IIb/IIIa receptor antagonist in patients at high risk for bleeding	IIa
• Fondaparinux: not recommended as the sole anticoagulant for primary PCI	III: Harm

TABLE 59G.1 Primary Percutaneous Coronary Intervention (PCI) for ST-Elevation Myocardial Infarction

	COR	LOE
Ischemic symptoms <12 hr	I	A
Ischemic symptoms <12 hr and contraindications to fibrinolytic therapy irrespective of delay in time after FMC	I	B
Cardiogenic shock or acute severe heart failure irrespective of delay in time after the onset of myocardial infarction	I	B
Evidence of ongoing ischemia 12-24 hr after the onset of symptoms	IIa	B
PCI on a noninfarct artery at the time of primary PCI in patients without hemodynamic compromise	III: Harm	B

TABLE 59G.3 Indications for Fibrinolytic Therapy When the Delay from First Medical Contact to Primary Percutaneous Intervention Is Longer than 120 Minutes

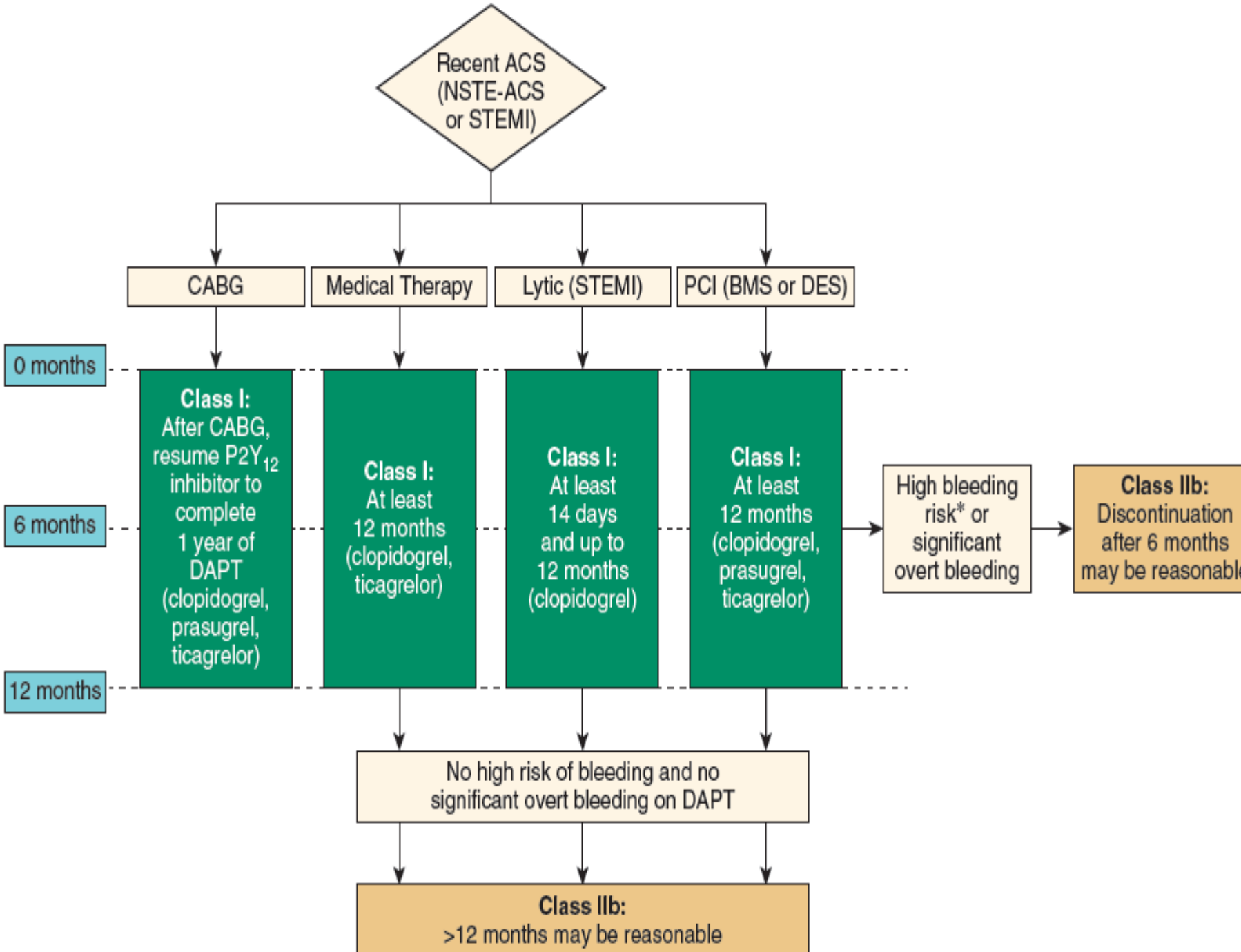
	COR	LOE
Ischemic symptoms <12 hr	I	A
Evidence of ongoing ischemia 12-24 hr after the onset of symptoms and a large area of myocardium at risk or hemodynamic instability	IIa	C
ST-segment depression except if true posterior (inferobasal) MI is suspected or when associated with ST-segment elevation in lead aVR	III: Harm	B

TABLE 59G.6 Indications for Transfer for Angiography after Fibrinolytic Therapy

	COR	LOE
Cardiogenic shock or acute severe heart failure that develops after initial evaluation	I	B
Intermediate- or high-risk findings on predischarge noninvasive ischemia testing	I	B
Spontaneous or easily provoked myocardial ischemia	I	C
Failed reperfusion or reocclusion after fibrinolytic therapy	IIa	B
Stable* patients after successful fibrinolysis, before discharge and ideally between 3 and 24 hr	IIa	B

TABLE 59G.7 Indications for Percutaneous Coronary Intervention (PCI) on an Infarct Artery in Patients Who Were Managed with Fibrinolytic Therapy or Who Did Not Receive Reperfusion Therapy

	COR	LOE
Cardiogenic shock or acute severe heart failure	I	B
Intermediate- or high-risk findings on predischarge noninvasive ischemia testing	I	C
Spontaneous or easily provoked myocardial ischemia	I	C
Patients with evidence of failed reperfusion or with reocclusion after fibrinolytic therapy (as soon as possible)	IIa	B
Stable* patients after successful fibrinolysis, ideally between 3 and 24 hr	IIa	B
Stable* patients >24 hr after successful fibrinolysis	IIb	B
Delayed PCI on a totally occluded infarct artery >24 hr after STEMI in stable patients	III: No benefit	B


Recent ACS (NSTE-ACS or STEMI)
CABG
Medical Therapy
Lytic (STEMI)
PCI (BMS or DES)
0 months
6 months
12 months
Class I: After CABG, resume P2Y12 inhibitor to complete 1 year of DAPT (clopidogrel, prasugrel, ticagrelor)
Class I: At least 12 months (clopidogrel, ticagrelor)
Class I: At least 14 days and up to 12 months (clopidogrel)
Class I: At least 12 months (clopidogrel, prasugrel, ticagrelor)
High bleeding risk* or significant overt bleeding
Class IIb: Discontinuation after 6 months may be reasonable
No high risk of bleeding and no significant overt bleeding on DAPT
Class IIb: >12 months may be reasonable

CARDIAC REHABILITATION

- Contemporary exercise-based cardiac rehabilitation after STEMI aims to increase functional capacity, reducing disability, improving quality of life, modifying coronary risk factors, and limit morbidity and mortality. The key components of cardiac rehabilitation include patient assessment; ongoing medical surveillance; nutritional counseling; management of hypertension, lipids, and diabetes mellitus; cessation of smoking; psychosocial counseling; physical activity counseling; exercise training; and pharmacologic treatment, as appropriate. When compared with usual care, cardiac rehabilitation is associated with lower total and cardiac mortality .

LIFESTYLE MODIFICATION

- Efforts to improve survival and quality of life after MI are related to lifestyle modification of known risk factors. Of these, cessation of smoking and control of hypertension are probably the most important. Use of hospital-based smoking cessation programs and referral to cardiac rehabilitation programs have led to successful smoking cessation.

DEPRESSION

- Physicians caring for patients following STEMI need to acknowledge the prevalence of major depression after infarction. This problem is associated independently with higher mortality.
- Therefore, a comprehensive cardiac rehabilitation program that includes primary health care personnel who counsel patients and make home visits can reduce the rate of rehospitalization for recurrent ischemia and infarction.

MODIFICATION OF LIPID PROFILE

- Obtaining a lipid profile on admission is reasonable in all patients admitted with acute MI. We continue to ascribe to a long-term target low-density lipoprotein (LDL) cholesterol level of less than 70 mg/dL for patients who experience an ACS.
- High-intensity statin therapy should be initiated or continued in all patients with STEMI and no contraindications to its use.

BLOOD PRESSURE CONTROL

- In patients with STEMI and HTN , blood pressure should be well controlled . (SBP < 140 mmhg but not < 110 mmhg)
- Drug therapy (beta blocker , ACE inh , ARB) with life style modification

SMOKING CESSATION

- Observational studies show that patients who stop smoking reduce their mortality in the succeeding years compared with continued smokers. Stopping smoking is potentially the most effective of all secondary prevention measures.
- Nicotine replacement, buproprione and antidepressants may be useful. Nicotine patches have been demonstrated to be safe in ACS patients .

DIET AND WEIGHT CONTROL

- Current guidelines on prevention recommend:
- (i) eating a wide variety of foods;
- (ii) adjustment of calorie intake to avoid obesity;
- (iii) increased consumption of fruit and vegetables, along with wholegrain cereals and bread, fish (especially oily varieties), lean meat and low-fat dairy products;
- (iv) replacing saturated and trans fats with monounsaturated and polyunsaturated fats from vegetable and marine sources, and to reduce total fats (of which less than one-third should be saturated) to ,30% of total calorie intake, and
- (v) to reduce salt intake if blood pressure is raised

- There is no evidence for the use of antioxidant supplements, low glycaemic index diets or homocysteine- lowering therapies following STEMI.
- Obesity is an increasing problem in patients with STEMI. Current ESC Guidelines define a body mass index (BMI) ,25 kg/m2 as optimal, and recommend weight reduction when the BMI is 30 kg/m2 or more, and when waist circumference is .102 cm in men or .88 cm in women, because weight loss can improve many obesity-related risk factors

PHYSICAL ACTIVITY

- Exercise therapy has long been used for rehabilitation purposes following STEMI and the benefit of regular physical exercise in stable CAD patients is also well established. It can reduce the anxiety associated with the life-threatening illness and improve patient self-confidence. Four mechanisms are considered to be important mediators of a reduced cardiac event rate: (i) improvement of endothelial function; (ii) reduced progression of coronary lesions; (iii) reduced thrombogenic risk and (iv) improved collateralization. In a large meta-analysis, exercise training as part of coronary rehabilitation programmes was associated with a 26% reduction in cardiac mortality rate in patients with CAD.

THANKS FOR YOUR ATTENTION