

Percutaneous coronary intervention

Produced by :

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Introduction

- Percutaneous Coronary Intervention is a minimally invasive nonsurgical procedure performed to improve blood flow in one or more segments of the coronary circulation.
- The first percutaneous coronary angioplasty was performed by the German Cardiologist Andreas Gruentzig on 1977 , but the first angioplasty in lower limb arteries was done with Dr. Dotter on 1964.
- Coronary revascularization with PCI primarily involves the use of balloon angioplasty and intracoronary stenting with DES or BMS.

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- The first coronary angioplasty with a drug delivery stent system was performed in Buenos Aires in 1999.
 - DES reduced the rate of restenosis and target lesion revascularization compared with BMS
 - DES : 1. metallic alloy 2.polymer coating (durable or bioabsorbable)
3.antirestenotic drug

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- Benefit of Stenting : prevent recoil & negative remodeling
 - DES reduced local neointimal hyperplasia.
 - DES significantly lower the rate of target lesion revascularization compared BMS.

Type of DES

First generation DES

Sirolimus-eluting stents

Cypher[®] Stent (Medtronic,
Inc, Minneapolis, MN)

Paclitaxel-eluting stents

Taxus[®]

Second generation DES

Zotarolimus-eluting stents

Endeavor[®]

Everolimus-eluting stents

Xience V[®] Everolimus Eluting

Coronary Stent (Abbott

Laboratories, Abbott Park, IL)

Promus[®] Everolimus-Eluting

Coronary Stent System (Boston

Scientific Corporation, Natick, MA)

TABLE 1

Evolution of drug-eluting stents

First generation

Nonbiodegradable (ie, durable) polymer-based thick strut
Sirolimus- or paclitaxel-eluting stents

Second generation

Nonbiodegradable (ie, durable) polymer-based thin strut
"Limus"-eluting stent (eliminated paclitaxel)

Third generation

Biodegradable polymer-based thick or thin strut
"Limus"-eluting stent

Third generation "B"

Polymer-free strut
"Limus"-eluting stents

Fourth generation

Bioresorbable, thick/thin strut
"Limus"-eluting vascular scaffolds (PLLA or magnesium)

"Limus" drugs: biolimus, everolimus, myolimus, novolimus, sirolimus, zotarolimus.

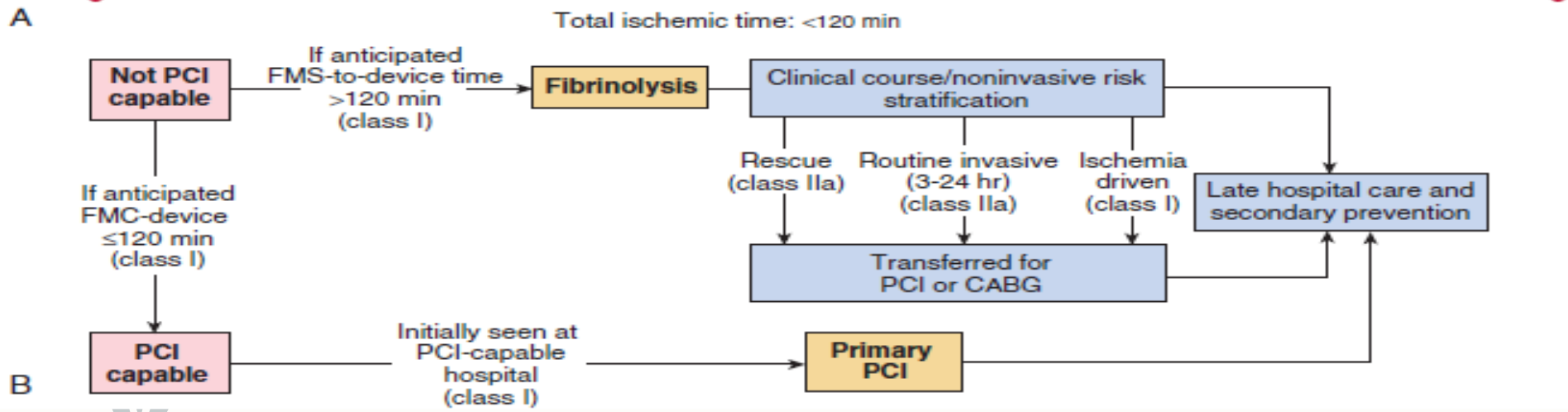
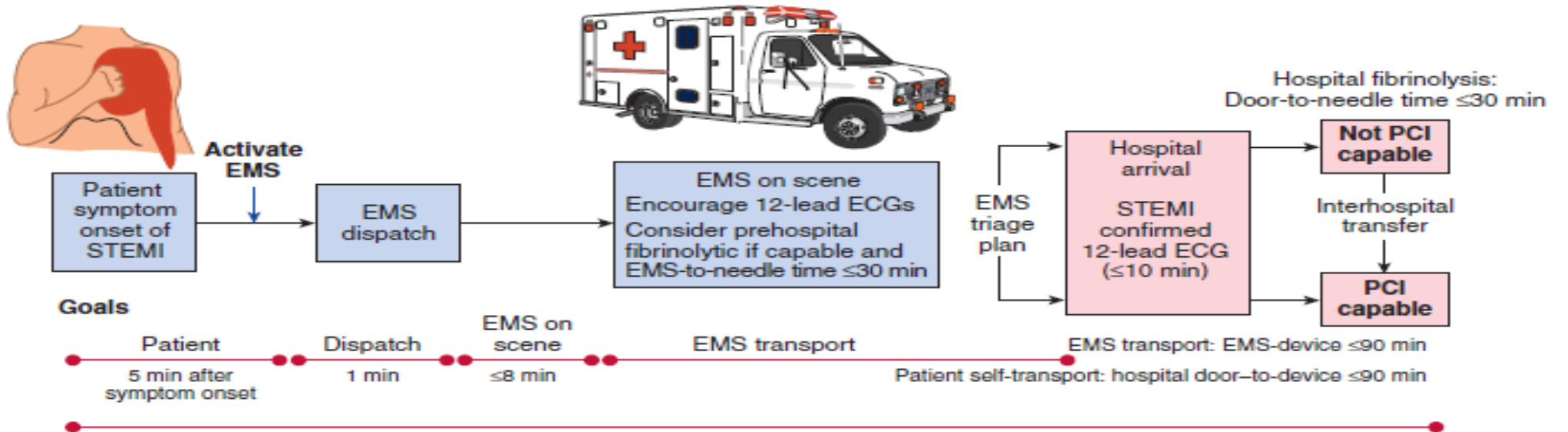
PLLA = poly-L-lactic acid

Primary PCI

- STEMI is defined by symptoms of myocardial ischemia associated with persistent electrocardiographic evidence of ST elevation and subsequent elevation of biologic markers of myocardial necrosis.
- ST elevation in the absence of either left bundle branch block (LBBB) or left ventricular (LV) hypertrophy is defined as new ST elevation of at least 2 mm in men or 1.5 mm in women in at least two contiguous leads.
- STEMI is inexorably linked to sudden cardiac death. Indeed, some 70% of deaths attributable to coronary heart disease occur with out-of-hospital arrest.

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- Primary PCI is generally preferable to fibrinolytic therapy.
 - Primary PCI, when successful, also results in early hospital discharge and return to activities.
 - Potential adverse effects of primary PCI include arterial access site complications and contrast agent– and antithrombotic-related complications.

Primary PCI



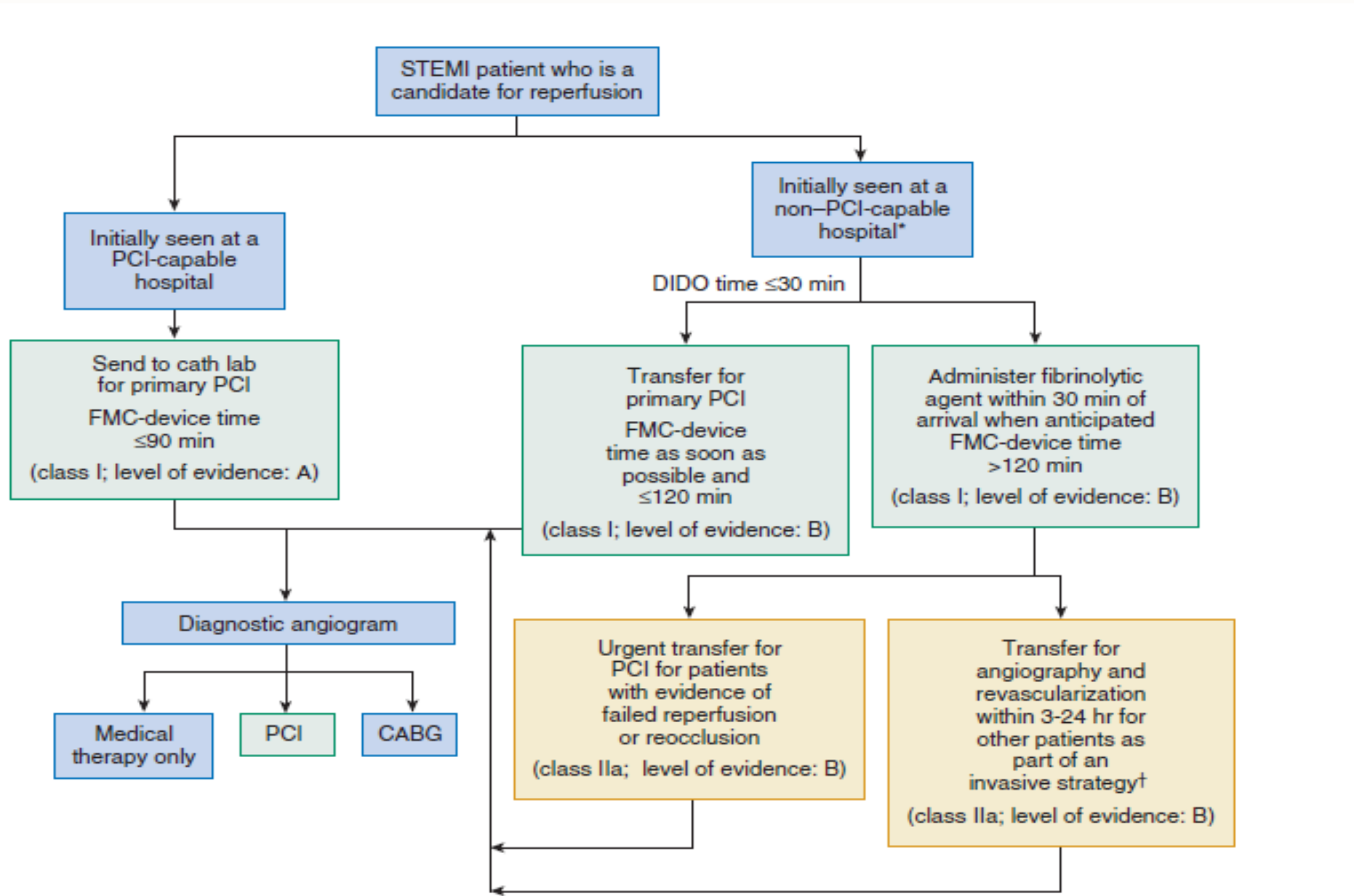


TABLE 52G-1 Primary Percutaneous Coronary Intervention for ST-Elevation Myocardial Infarction

| | COR | LEVEL OF EVIDENCE |
|--|-----|-------------------|
| 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 HF irrespective of delay in time after the onset of MI | I | B |
| Evidence of ongoing ischemia 12-24 hr after the onset of symptoms | IIa | B |

Absolute Contraindications

Any previous intracranial hemorrhage
Known structural cerebral vascular lesion
Known malignant intracranial neoplasm
Ischemic stroke within 3 months (except ischemic stroke within 4.5 hours)
Suspected aortic dissection
Active bleeding or bleeding diathesis
Significant closed-head or facial trauma within 3 months
Intracranial or intraspinal surgery within 2 months
Severe uncontrolled hypertension (not responsive to emergency therapy)
For streptokinase, previous treatment within 6 months

Relative Contraindications

History of chronic, severe, poorly controlled hypertension
Significant hypertension at initial evaluation (systolic blood pressure >180 mm Hg, diastolic blood pressure >110 mm Hg)
History of ischemic stroke >3 months
Dementia
Known intracranial pathology not covered in Absolute Contraindications
Traumatic or prolonged cardiopulmonary resuscitation (>10 minutes)
Major surgery within 3 weeks
Recent internal bleeding (within 2-4 weeks)
Noncompressible vascular puncture
Pregnancy
Active peptic ulcer
Oral anticoagulant therapy

TABLE 52G-6 Indications for Transfer for Angiography after Fibrinolytic Therapy

| | COR | LEVEL OF EVIDENCE |
|--|------------|--------------------------|
| Cardiogenic shock or acute severe heart failure that develops after initial evaluation | I | B |
| Intermediate- or high-risk findings on pre-discharge 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 |

Rescue PCI & Elective PCI

TABLE 52G-7 Indications for Percutaneous Coronary Intervention on an Infarct Artery in Patients Who Were Managed with Fibrinolytic Therapy or Who Did Not Receive Reperfusion Therapy

| | COR | LEVEL OF EVIDENCE |
|---|-----------------|-------------------|
| 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 h after STEMI in stable patients | III: No benefit | B |

فرآیند مدیریت درمان سکته حاد قلبی

کد: WP-38-01

صفحه 1 از 3

مراجعه بیمار به اورژانس از طریق آمبولانس یا خود بیمار*

*در صورت مشاهده علائم در بیمار بستری و تأیید STEMI، تمامی وظایف دستیار اورژانس (ذکر شده در فرآیند پیر عهده رزیدنت مسئول بیمار بستری در بخش و کلیه وظایف کادر پرستاری اورژانس نیز بر عهده کادر پرستاری بخش بستری می باشد .

تریاژ اولیه و تکمیل فرم تریاژ -تیت Door Time
پرستار تریاژ

گرفتن ECG در کمتر از 10 دقیقه بعد از ورود به بیمارستان - کمک بیمار اورژانس

دویت ECG و اولویت بندی ویزیت بیمار**
پرستار تریاژ

ویزیت بیمار و تفسیر ECG
دستیار قلب / متخصص قلب اورژانس

**پرستار تریاژ در صورت وجود Pain Chest و یا مشاهده افزایش ST در توار قلب، بلافاصله بیمار را جهت ویزیت به اتاقی معاینه هدایت می کند .

ادامه درمان طبق فرآیند بخش اورژانس

آیا STEMI تأیید می شود؟

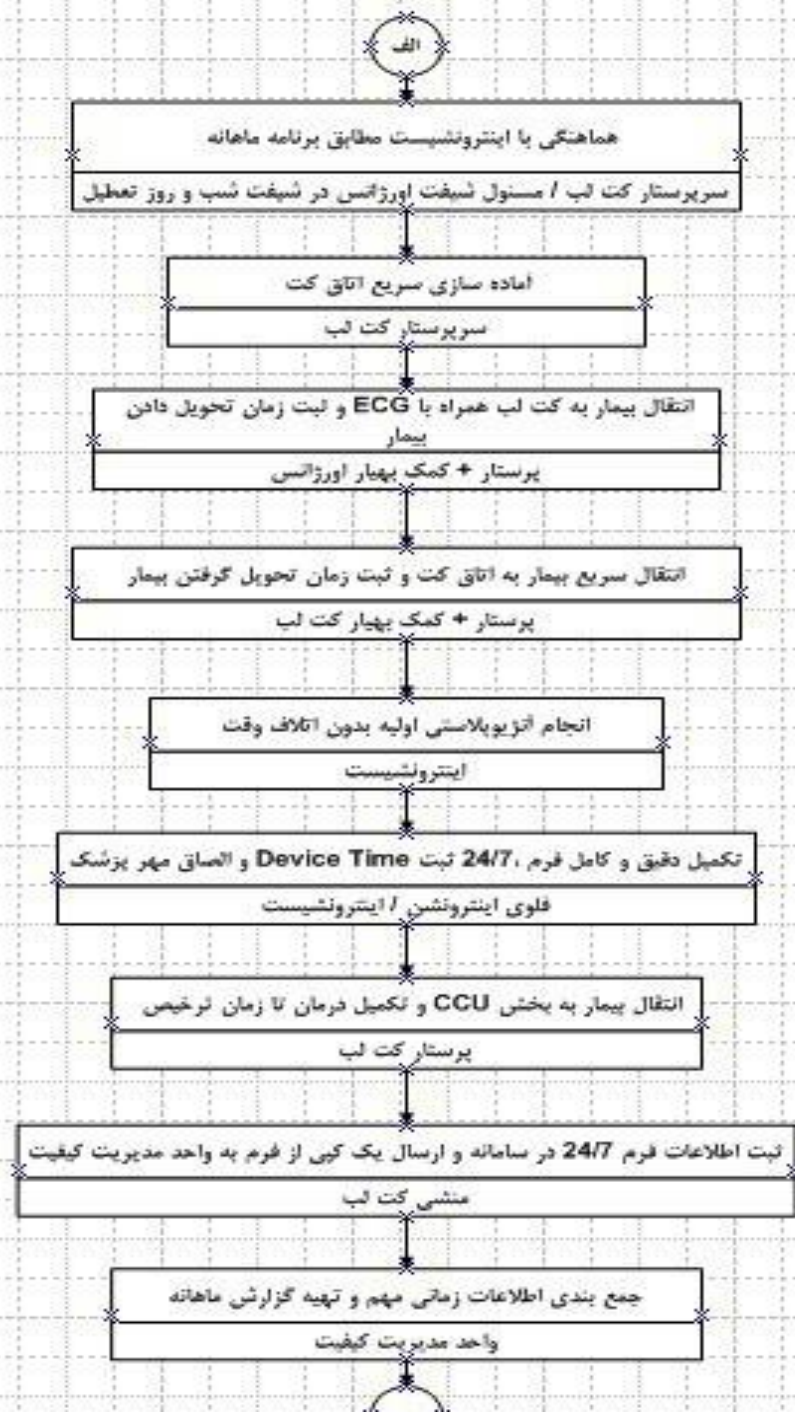
اعلام کد 24/7 بدون اتلاف وقت
دستیار ارشد قلب / متخصص قلب اورژانس

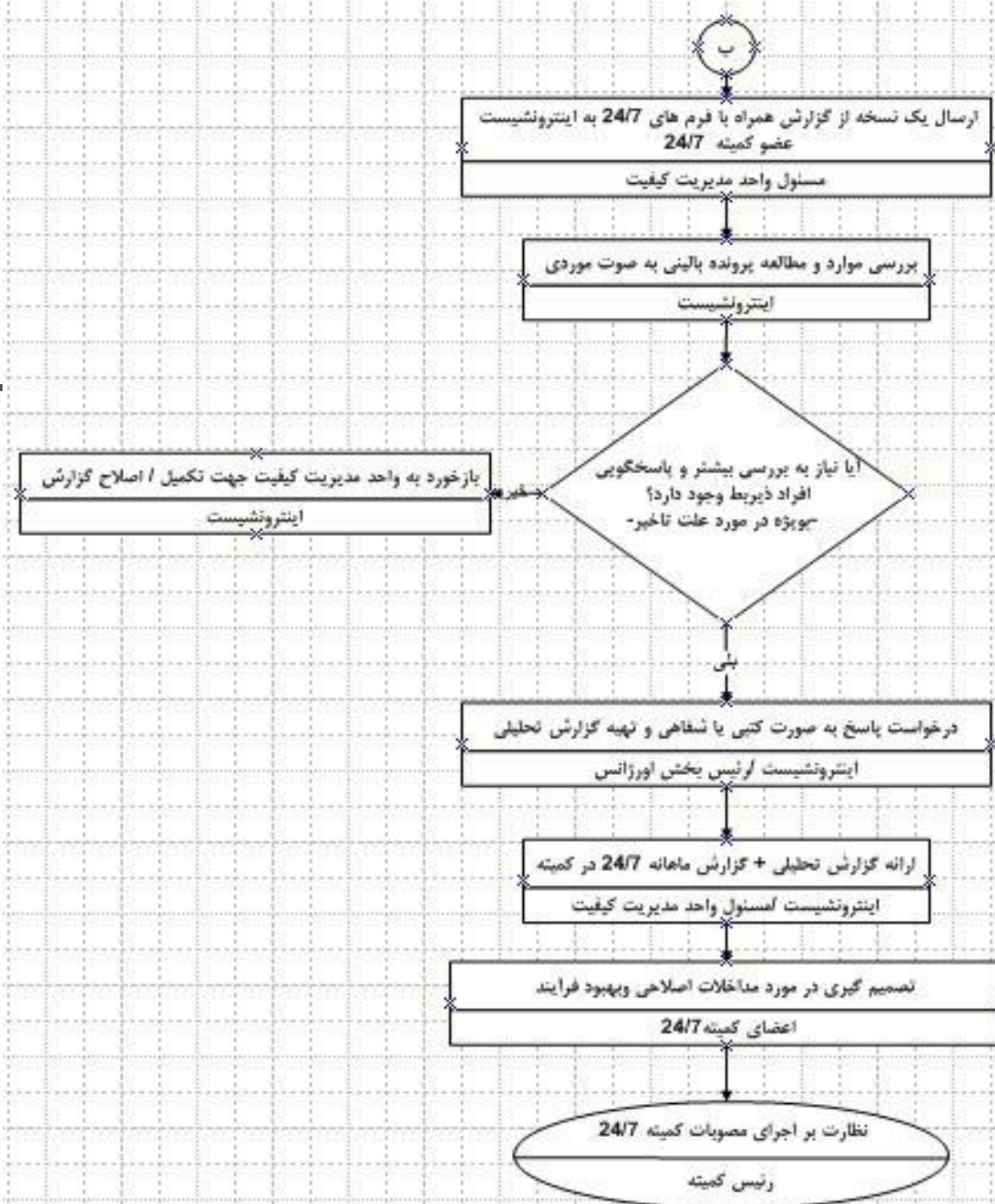
***در شیفت شب و روزهای تعطیل

اطلاع به سوپروایزر جهت حضور سریع در کت لب***
مسئول شیفت اورژانس

ثبت زمان تأیید STEMI و زمان اعلام کد و الصاق مهر در فرم 24/7
دستیار قلب بخش اورژانس

الف





STEMI Management Registry

کد: 18-19-
 ویرایش: 02
 تاریخ ویرایش: دی ۹۸

نام: (شماره گذرنامه در خصوص اتباع خارجی): سال تولد:
 Iranian Not Iranian
 نام خانوادگی: جنس: مرد زن تاریخ تولد:/...../۱۳.....

Demographic Data

شماره پرونده: نحوه مراجعه بیمار:
 انتقال به اورژانس بیمارستان توسط EMS مراجعه مستقیم بیمار
 ارجاع از بیمارستان بدون قابلیت PPCI بیمار بستری در بیمارستان

Time Intervals

Onset of Symptoms (yyyy/mm/dd) (hh/mm)
 First Medical Contact (yyyy/mm/dd) (hh/mm)
 Admission Time (Door Time) (yyyy/mm/dd) (hh/mm)
 Device Time (yyyy/mm/dd) (hh/mm)

STEMI ECG : (yyyy/mm/dd) (hh/mm)
 247 Code Time : (yyyy/mm/dd) (hh/mm)
 Arrival at Cath lab : (yyyy/mm/dd) (hh/mm)

Cath Lab Data

- Initial Reperfusion Therapy :

- Primary PCI
- Rescue PCI
- Urgent CABG
- Medical Treatment
- Unsuccessful P.PCI

Infarct related artery (IRA) : LM LAD LCX RCA
 Graft Diagonal Ramus OM PDA PLB

Initial TIMI flow grade in IRA 0 1 2 3
 Final TIMI flow grade in IRA 0 1 2 3

Condition at Discharge from Hospital

Condition at discharge: Alive Dead
 Ejection fraction at discharge: Not performed Good (> %55) Mild (%45- %55)
 Moderate (%30-%45) Severe (< %30)

مهر و امضاء پزشک اورژانس

مهر و امضاء فلوشیپ اینترونشن

مهر و امضاء پزشک معالج

| | COR | LEVEL OF EVIDENCE |
|--|-----|-------------------|
| Antiplatelet Therapy | | |
| <i>Aspirin</i> | | |
| • 162- to 325-mg loading dose before the procedure | I | B |
| • 81- to 325-mg daily maintenance dose (indefinite)* | I | A |
| • 81 mg daily is the preferred maintenance dose* | Ia | B |

P2Y₁₂ Inhibitors

Loading Doses

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

Maintenance Doses and Duration of Therapy

DES placed: Continue therapy for 1 year with

- Clopidogrel: 75 mg daily I B
- Prasugrel: 10 mg daily I B
- Ticagrelor: 90 mg twice a day* I B

BMS[†] placed: Continue therapy for 1 year with

- Clopidogrel: 75 mg daily I B
- Prasugrel: 10 mg daily I B
- Ticagrelor: 90 mg twice a day* I B

DES placed:

- Clopidogrel, prasugrel, or ticagrelor* continued beyond 1 year IIb C
- Patients with STEMI and previous stroke or TIA: prasugrel III: Harm B

| | COR | LEVEL OF EVIDENCE |
|--|-----------|-------------------|
| Anticoagulant Therapy | | |
| • UFH | | |
| • With a GP IIb/IIIa receptor antagonist planned: 50- to 70-unit/kg IV bolus to achieve therapeutic ACT ⁴ | I | C |
| • With no GP IIb/IIIa receptor antagonist planned: 70- to 100-unit/kg bolus to achieve a therapeutic ACT ⁵ | I | C |
| • 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 | I | B |
| • Reduce the infusion to 1 mg/kg/hr with estimated an CrCl <30 mL/min | | |
| • Preferred over UFH with a GP IIb/IIIa receptor antagonist in patients at high risk for bleeding | IIa | B |
| • Fondaparinux: not recommended as the sole anticoagulant for primary PCI | III: Harm | B |

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

| | | |
|--|-----|---|
| • Abciximab: 0.25-mg/kg IV bolus, then 0.125 μ g/kg/min (maximum, 10 μ g/min) | IIa | A |
| • Tirofiban (high bolus dose): 25- μ g/kg IV bolus, then 0.15 μ g/kg/min | IIa | B |
| • In patients with CrCl <30 mL/min, reduce the infusion by 50% | | |
| • Eptifibatid (double bolus): 180- μ g/kg IV bolus, then 2 μ g/kg/min; a second 180- μ g/kg bolus is administered 10 min after the first bolus | IIa | B |
| • In patients with CrCl < 50 mL/min, reduce the infusion by 50% | | |
| • Avoid in patients on hemodialysis | | |
| • Pre-catheterization laboratory administration of IV GP IIb/IIIa receptor antagonist | IIb | B |
| • Intracoronary abciximab: 0.25-mg/kg bolus | IIb | B |

Vascular Access

- Femoral & radial
- In femoral access → INR < 1.8
- In Radial Access → INR < 2.2

Rival Trial

| | FEMORAL (N = 3514) | RADIAL (N = 3507) | P VALUE |
|--|-----------------------|----------------------|--------------|
| Composite of death, MI, stroke, or non-CABG-related major bleeding at 30 days* | 4.0% | 3.7% | 0.50 |
| Death at 30 days | 1.5% | 1.3% | 0.47 |
| MI at 30 days | 1.9% | 1.7% | 0.65 |
| Stroke at 30 days | 0.4% | 0.6% | 0.30 |
| PCI success | 95.2% | 95.4% | 0.83 |
| Access site crossover | 2.0% | 7.6% | <0.0001 |
| Major vascular complications | 3.7% | 1.4% | <0.0001 |
| Access site major bleeding | 0.3% | 0.2% | Not provided |
| Symptomatic radial occlusion | NA | 0.2% | NA |
| Procedure time (min) | 35 | 34 | 0.62 |
| Fluoroscopy time (min) | 8.0 | 9.3 | <0.0001 |
| Contrast volume (mL) | 180 | 181 | 0.87 |
| Patient prefers radial access for next procedure | 50.7% | 90.2% | <0.0001 |


Vascular Access Complications

- Vascular access site complications occur after 3% to 7% of femoral PCIs and lead to significantly increased length of hospital stay, total cost, and morbidity and mortality. Complications range from relatively minor access site hematomas, to life-threatening retroperitoneal bleeding requiring emergency blood transfusion, to damage to the vasculature necessitating prompt surgical intervention



– Predisposing factor :

older age, female sex, larger vascular sheath size, low body mass index, renal insufficiency, and degree of anticoagulation during the procedure



| COMPLICATION | SCAI REGISTRY RISK (%) |
|------------------------------|------------------------|
| Mortality | 0.11 |
| Myocardial infarction | 0.05 |
| Cerebrovascular accident | 0.07 |
| Arrhythmias | 0.38 |
| Vascular complications | 0.43 |
| Contrast agent reaction | 0.37 |
| Hemodynamic complications | 0.26 |
| Perforation of heart chamber | 0.03 |
| Other complications | 0.28 |
| Total of major complications | 1.70 |

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- In femoral access , sheath removal is done when ACT < 180
 - In radial access , sheath removal is done after procedure and TR band is used
 - Cardiac monitoring
 - ECG
 - Evaluation for complication after PCI like hematoma , bleeding from access site , retroperitoneal hemorrhage , stroke , chest pain , arrhythmia
 - After Primary PCI → CBR until 12 h except in unstable hemodynamic
 - NPO or clear liquid diet until 4 – 12 h then Po low salt low fat

Post PCI Care

- Chest pain after PCI is relatively common.
- Chest pain after PCI , may occur as a result of acute or subacute stent thrombosis, residual dissections, plaque prolapse, side branch occlusion, or thrombus at the treatment site

TABLE 55G-5 ACC/AHA Postprocedure Recommendations for Antiplatelet Therapy in Patients Undergoing Percutaneous Coronary Intervention³

| INDICATION | CLASS | RECOMMENDATION | LOE |
|--|--|---|-----|
| Aspirin | Class I | After PCI, use of aspirin should be continued indefinitely | A |
| | Class IIa | After PCI, it is reasonable to use ASA, 81 mg/day, in preference to higher maintenance doses | B |
| P2Y ₁₂ inhibitors: STEMI and unstable angina/NSTEMI | Class I | In patients receiving a stent during PCI for ACS, P2Y ₁₂ inhibitor therapy should be given for at least 12 months. Options include clopidogrel, 75 mg/day; prasugrel, 10 mg/day; or ticagrelor, 90 mg twice daily | B |
| | Class I | In patients receiving a DES during PCI for a non-ACS indication, clopidogrel, 75 mg/day, should be given for at least 12 months if not at high risk for bleeding | B |
| | | In patients receiving a BMS during PCI for a non-ACS indication, clopidogrel, 75 mg/day, should be given for a minimum of 1 month and ideally up to 12 months (unless the patient is at increased risk for bleeding, in which case it should be given for a minimum of 2 weeks) | C |
| | Class IIa | PPIs should be used in patients with a history of previous GI bleeding who require DAPT | C |
| | | If the risk for morbidity from bleeding outweighs the anticipated benefit afforded by a recommended duration of P2Y ₁₂ inhibitor therapy after stent implantation, earlier discontinuation (e.g., <12 months) of P2Y ₁₂ inhibitor therapy is reasonable | C |
| | Class IIb | Use of PPIs is reasonable in patients with an increased risk for GI bleeding who require DAPT | C |
| Class III no benefit | Continuation of clopidogrel, prasugrel, or ticagrelor beyond 12 months may be considered in patients undergoing placement of a DES | C | |
| | Routine use of a PPI is not recommended for patients at low risk for GI bleeding | C | |



– Primary PCI without on site surgery → class IIa

– Elective PCI without on site surgery → Class IIb

Thanks for your attention

