ACS –GOLDEN HOUR

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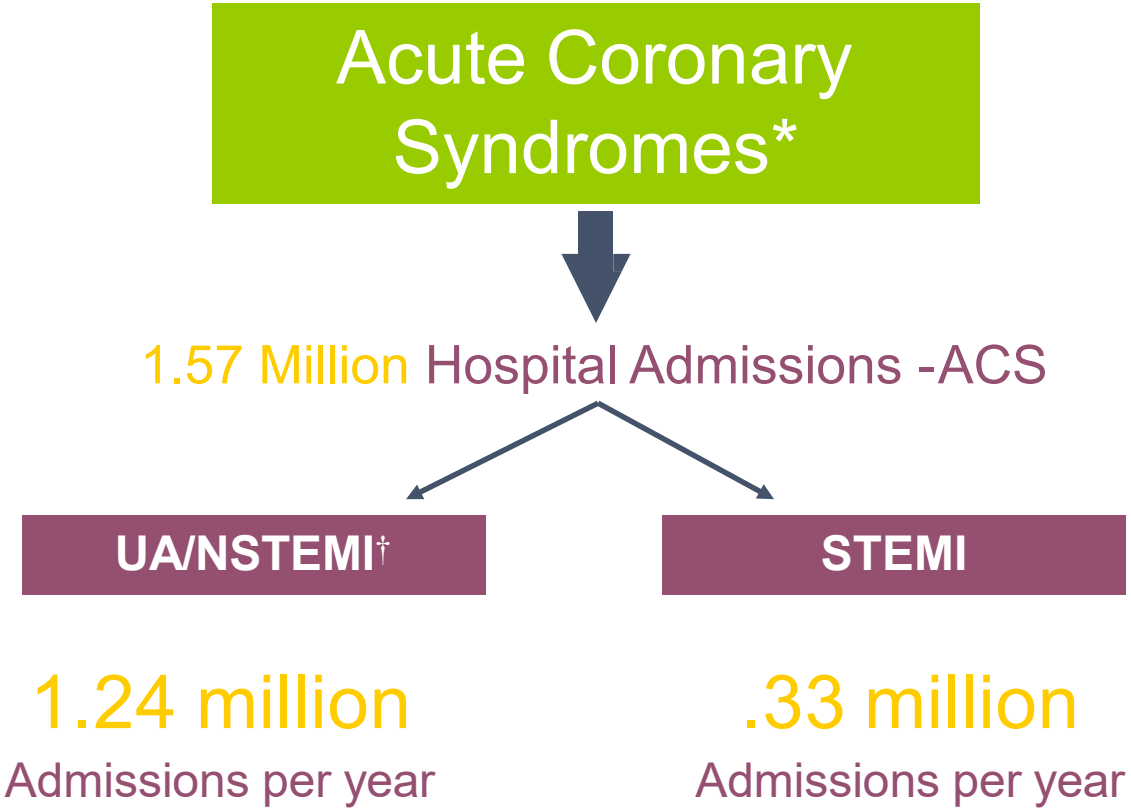
Acute Coronary Syndrome

Definition:

The spectrum of acute ischemia related syndromes ranging from UA to MI with or without ST elevation *that are secondary to acute plaque rupture or plaque erosion*.

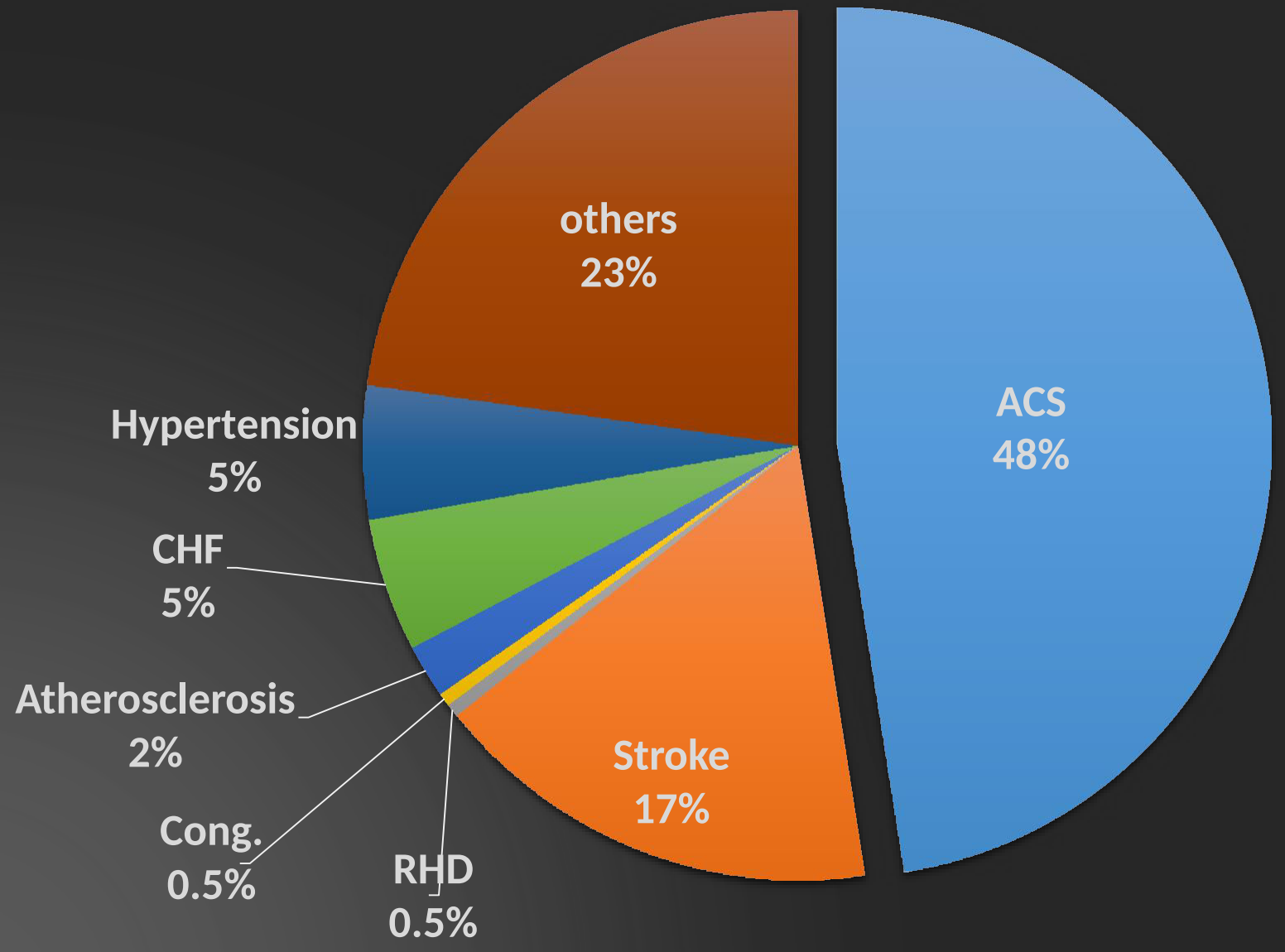
[___UA_____NSTEMI_____STEMI___]

A constellation of symptoms related to obstruction of coronary arteries with **chest pain** being the most common symptom in addition to nausea, vomiting, diaphoresis etc.



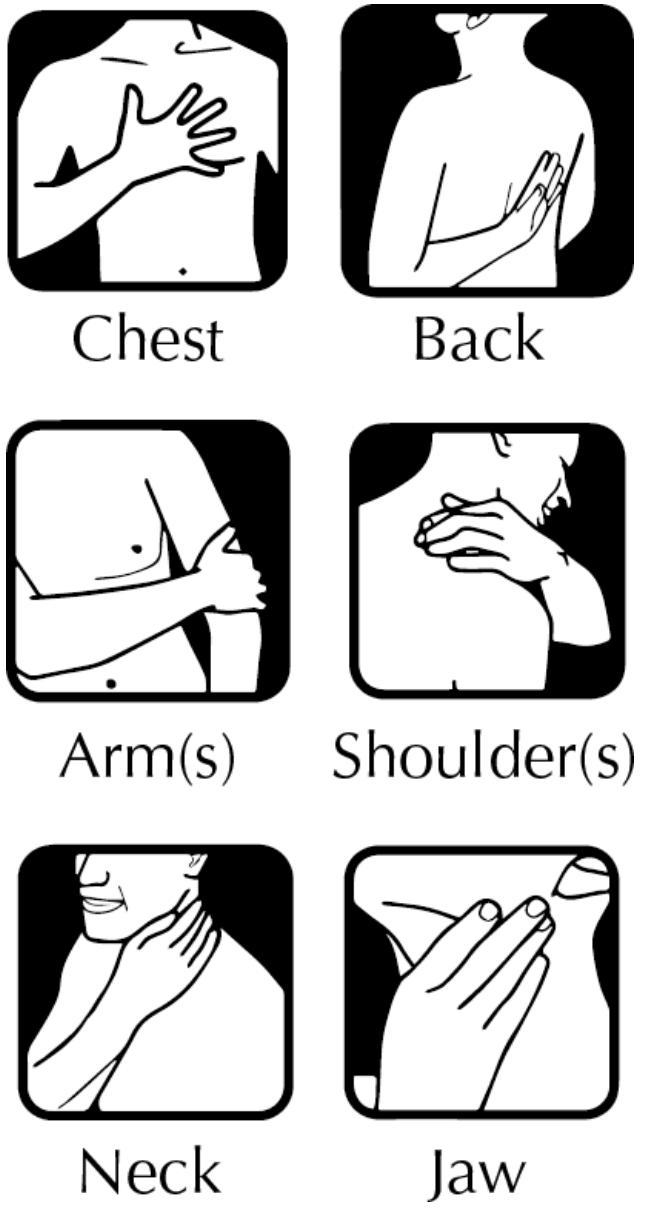
Heart Disease and Stroke Statistics – 2007 Update. Circulation 2007;115:69-171.
*Primary and secondary diagnoses. †About 0.57 million NSTEMI and 0.67 million UA.

Deaths from ACS



Signs and symptoms of ACS presentation

- Symptoms may include:
  - chest discomfort (tightness, pressure, heaviness) at rest or for a prolonged period (> 10 minutes, not relieved by sublingual nitrates)
  - recurrent chest discomfort
  - discomfort associated with syncope/acute heart failure.
- The pain may spread to other parts of the upper body, including:
  - Back, neck, jaw, arm(s), shoulder(s) or epigastric pain.
- The person may also experience:
  - Dyspnoea (shortness of breath), diaphoresis (profuse perspiration), dizziness, nausea or vomiting
  - Recent research shows that women, the elderly and people with diabetes are less likely to experience chest pain as a symptom.



Angina Equivalent

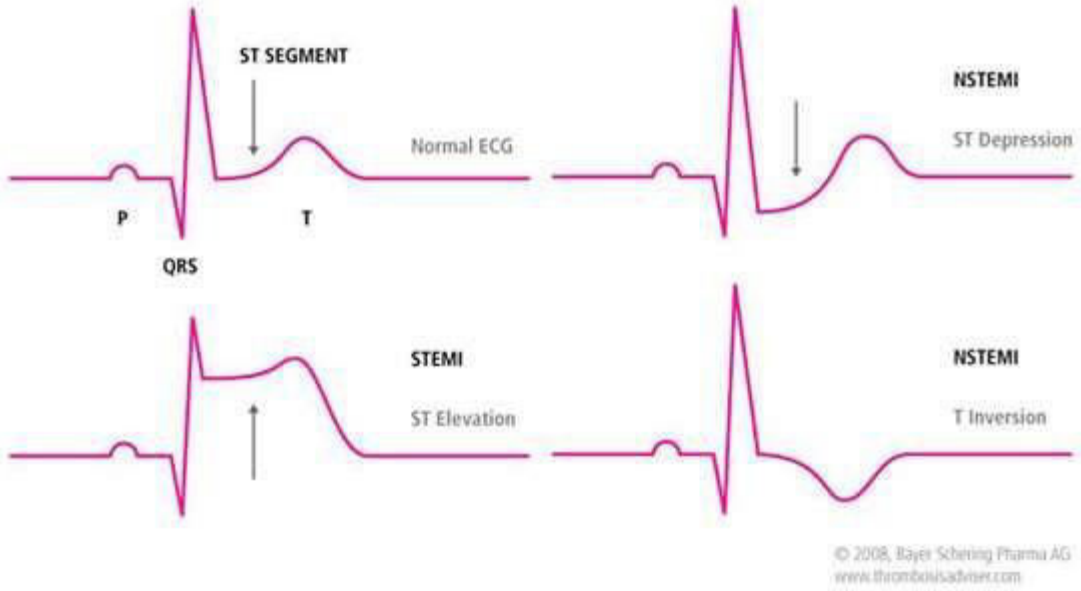
- Patients who are experiencing ACS, yet do not complain of typical chest pain; rather, these patients note atypical pain, dyspnea, weakness, diaphoresis, or emesis—these complaints, in fact, are the manifestation of the ACS event.
- The most frequently encountered anginal equivalent chief complaint is dyspnea, which is found in 10% to 30% of patients with AMI.

CHARACTERISTICS OF TYPICAL ANGINAL CHEST PAIN

CHARACTERISTIC	SUGGESTIVE OF ANGINA	LESS SUGGESTIVE OF ANGINA
TYPE OF PAIN	DULL PRESSURE/CRUSHING PAIN	SHARP/STABBING
DURATION	2-5 MIN, <20 MIN	SECONDSTO HOURS/CONTINUOUS
ONSET	GRADUAL	RAPID
LOCATION/CHEST WALL TENDERNESS	SUBSTERNAL, NOT TENDER TO PALP.	LATERAL CHEST WALL/TENDER TO PALP.
REPRODUCIBALITY	WITH EXERTION/ACTIVITY	WITH BREATHING/MOVING
AUTONOMIC SYMPTOMS	PRESENT USUALLY	ABSENT

Based on ECG and cardiac enzymes, ACS is classified into:

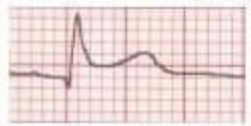
- **STEMI:** ST elevation, elevated cardiac enzymes
- **NSTEMI**: ST depression, T-wave inversion, **elevated** cardiac enzymes
- **Unstable Angina:** Non specific EKG changes, **normal** cardiac enzymes



Acute Coronary Syndrome

Electrocardiogram

ST-elevation



No ST-elevation

Cardiac markers

negative → Unstable angina

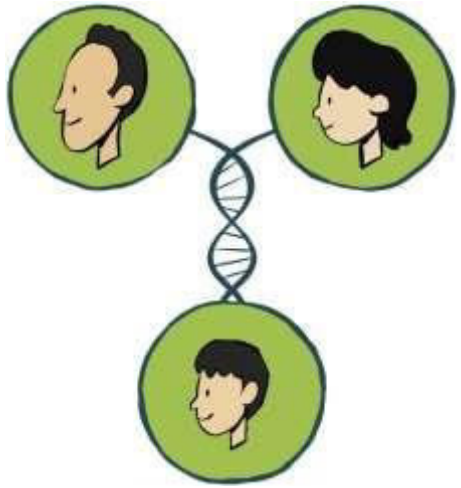
positive

Myocardial infarction

STEMI → Q-wave MI

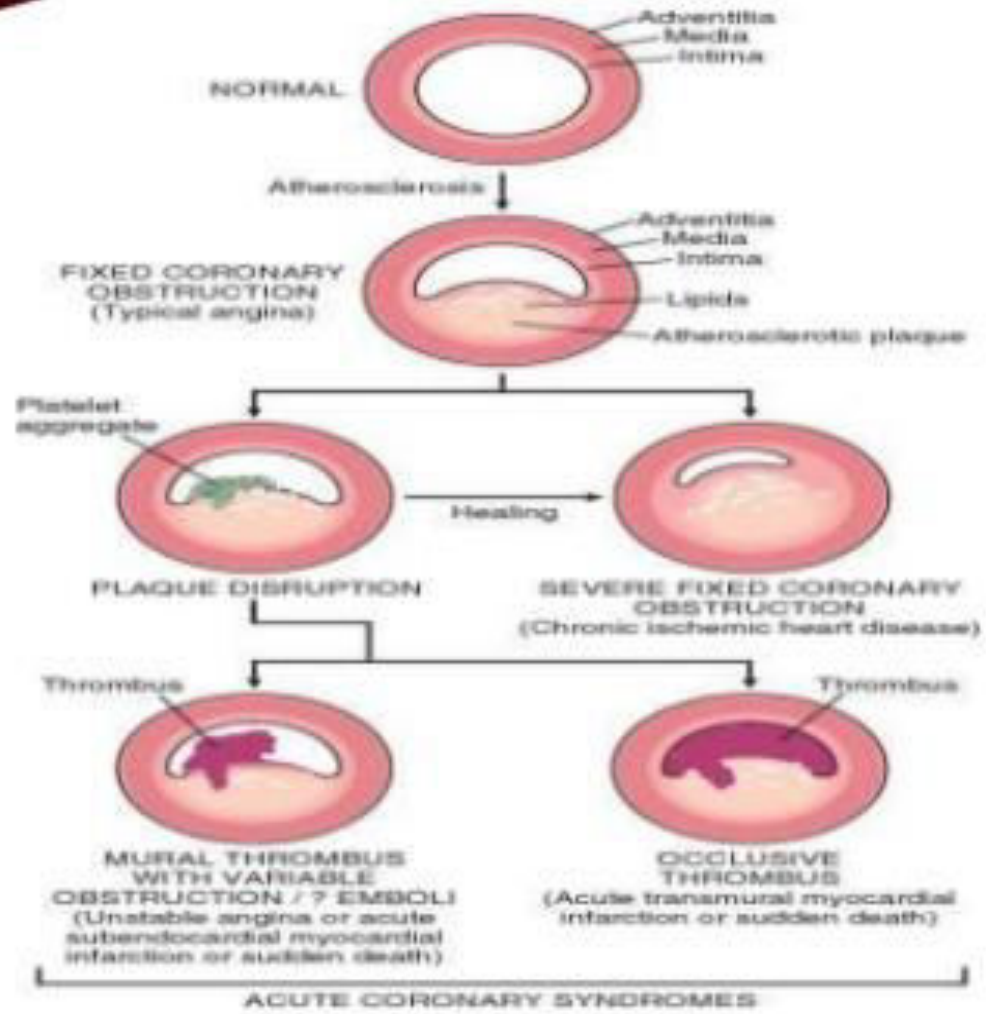
NSTEMI → non-Q-wave MI

RISK FACTORS



Risk Factors

PATHOGENESIS



Unstable Angina

Non occlusive thrombus

Non specific ECG

Normal cardiac enzymes

NSTEMI

Non-occlusive thrombus sufficient to cause tissue damage & mild myocardial necrosis

ST depression +/- T wave inversion on ECG

Elevated cardiac enzymes

STEMI

Complete thrombus **occlusion**

ST elevations on ECG or new LBBB

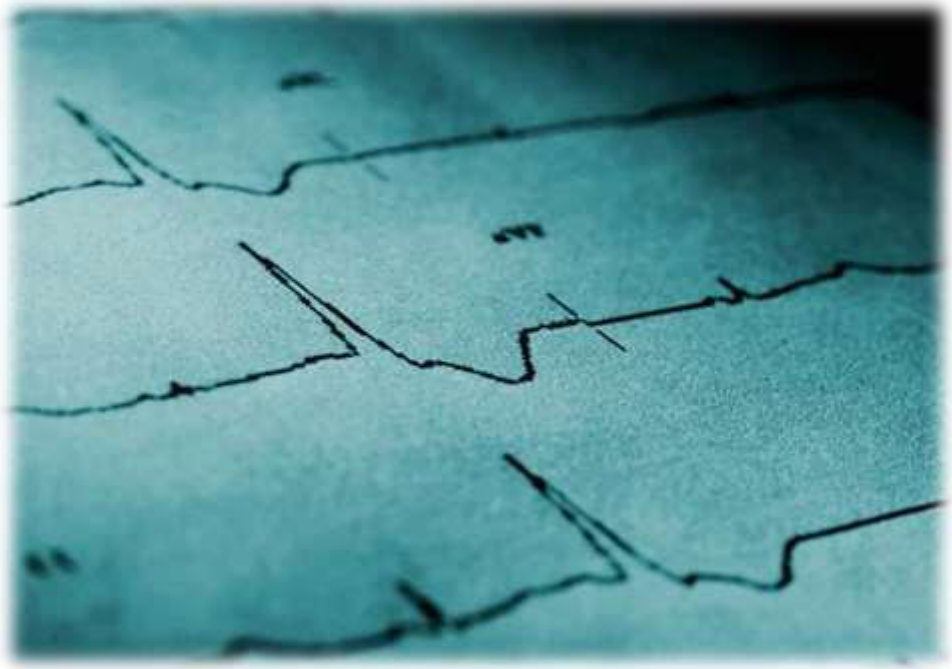
Elevated cardiac enzymes

More severe symptoms

Diagnosis of ACS

At least 2 of the following

- History (angina or angina equivalent)
- Acute ischemic ECG changes
- Typical rise and fall of cardiac markers
- Absence of another identifiable etiology



Diagnosis

Diagnosis requires a rise in serum levels of **cardiac markers** (preferably troponin) together with:

Defined clinically by patient history

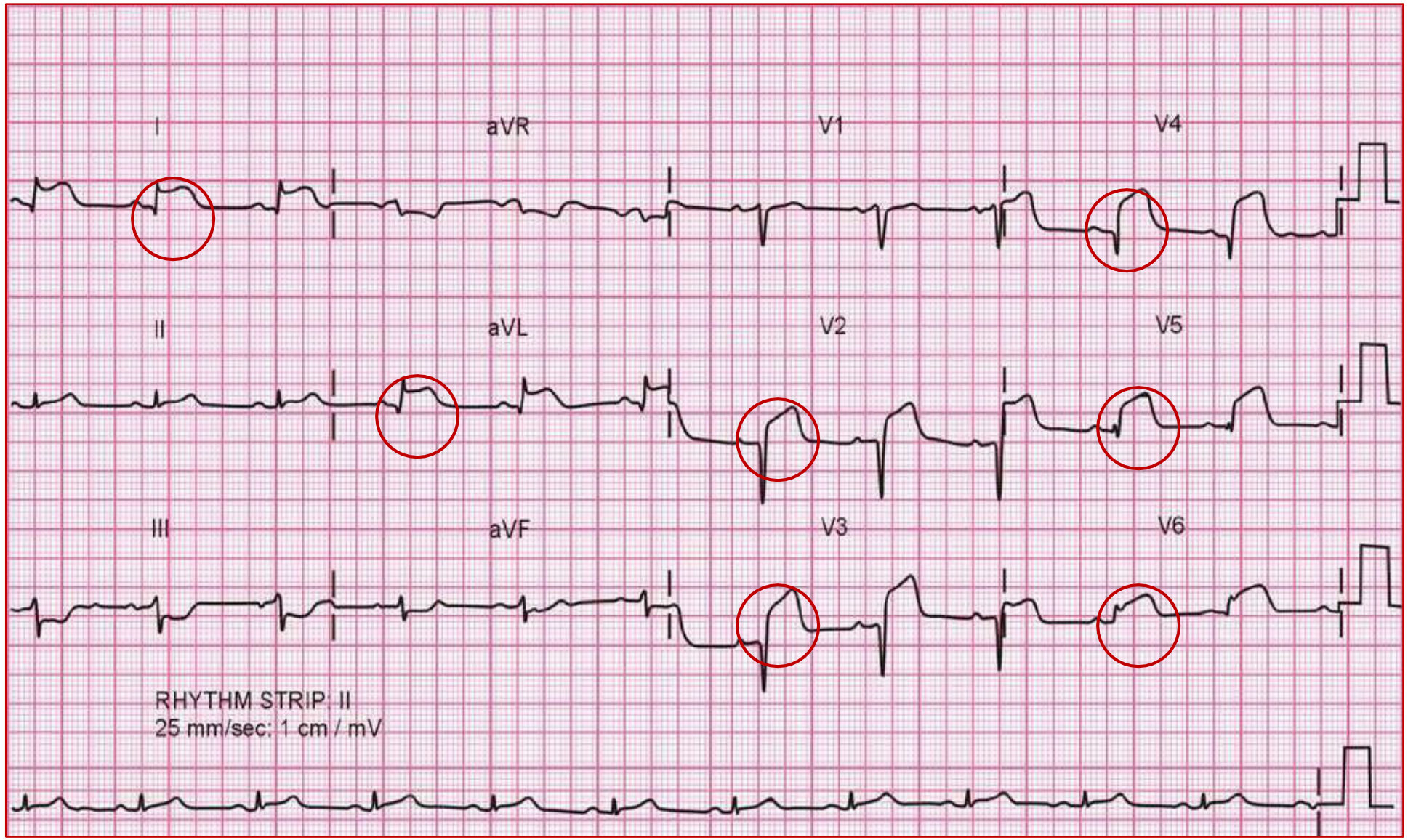
ECG (new ST-T wave changes, new left bundle branch block or evolving pathological Q waves)

Imaging evidence of new regional wall motion abnormality.

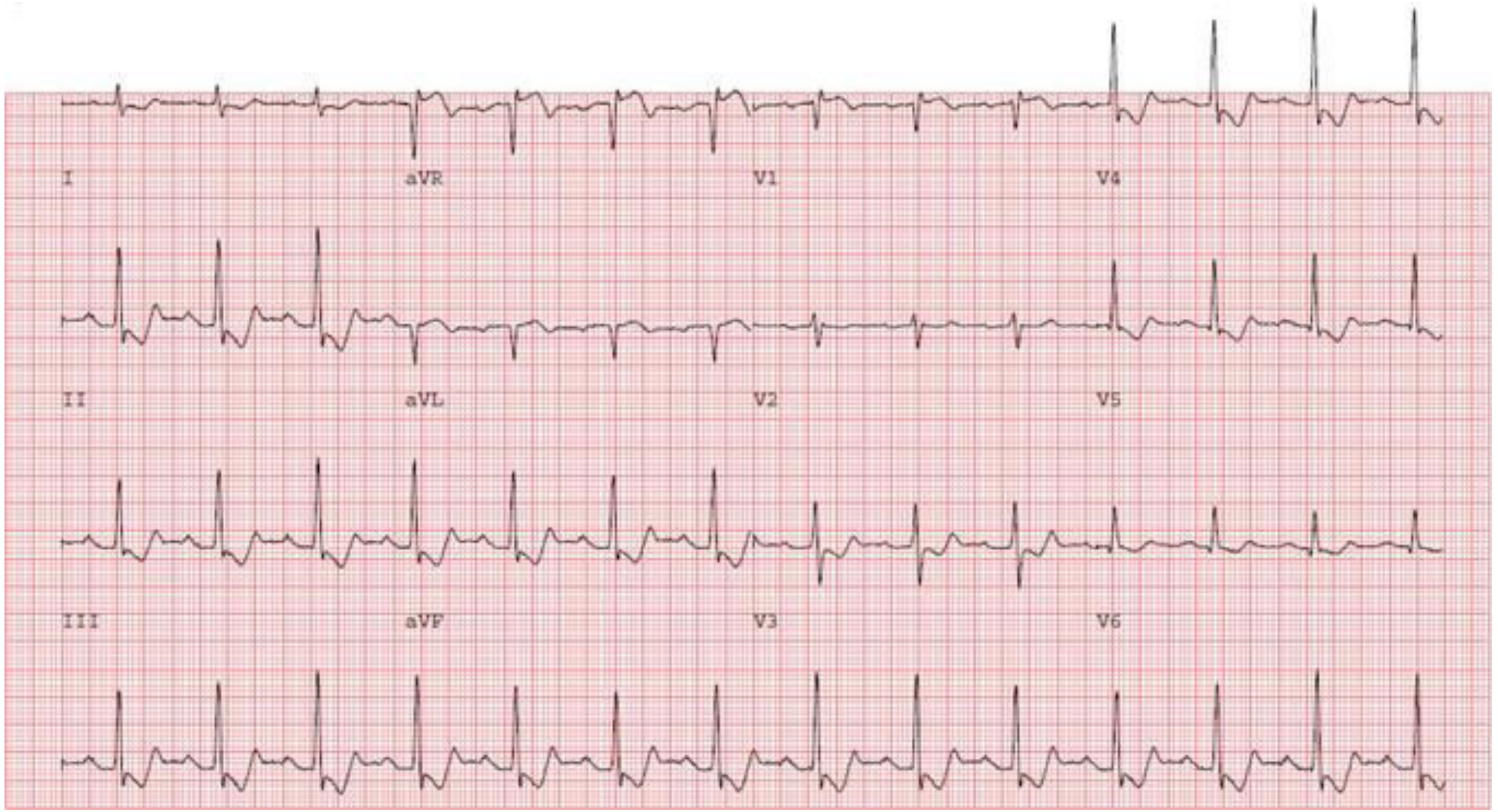
STEMI

- An ST-segment elevation myocardial infarction (STEMI) can be confirmed by an ECG.
- A 12-lead ECG should be performed and interpreted expeditiously.
- STEMI is **defined as** presentation with clinical symptoms consistent with an ACS with ECG features including any of:
  - ✓ Persistent ST-segment elevation ≥ 1 mm in two contiguous limb leads
  - ✓ ST-segment elevation ≥ 2 mm in two contiguous chest leads
  - ✓ New left bundle branch block (LBBB) pattern.

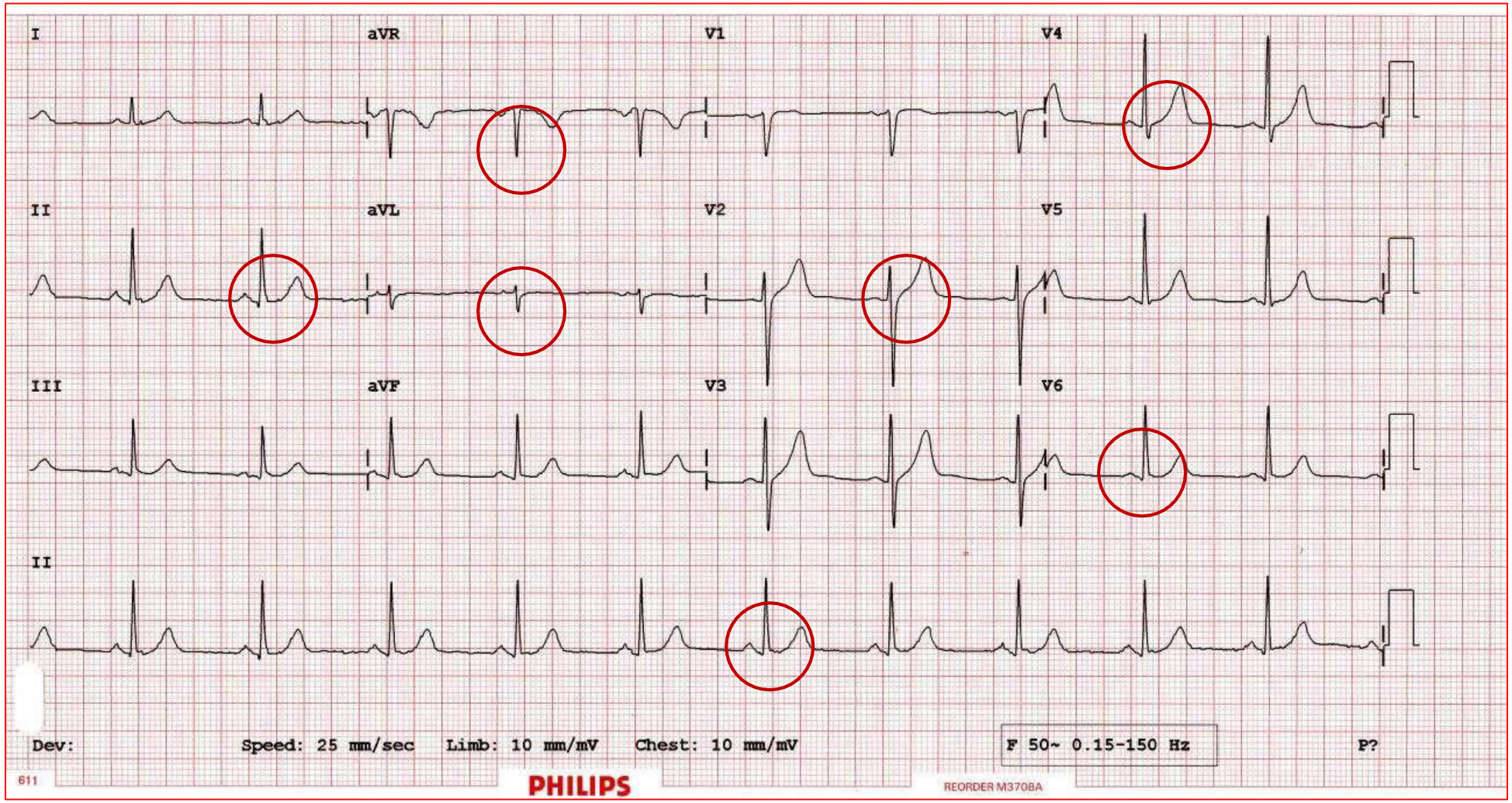
Acute ST Elevation MI



NSTEMI



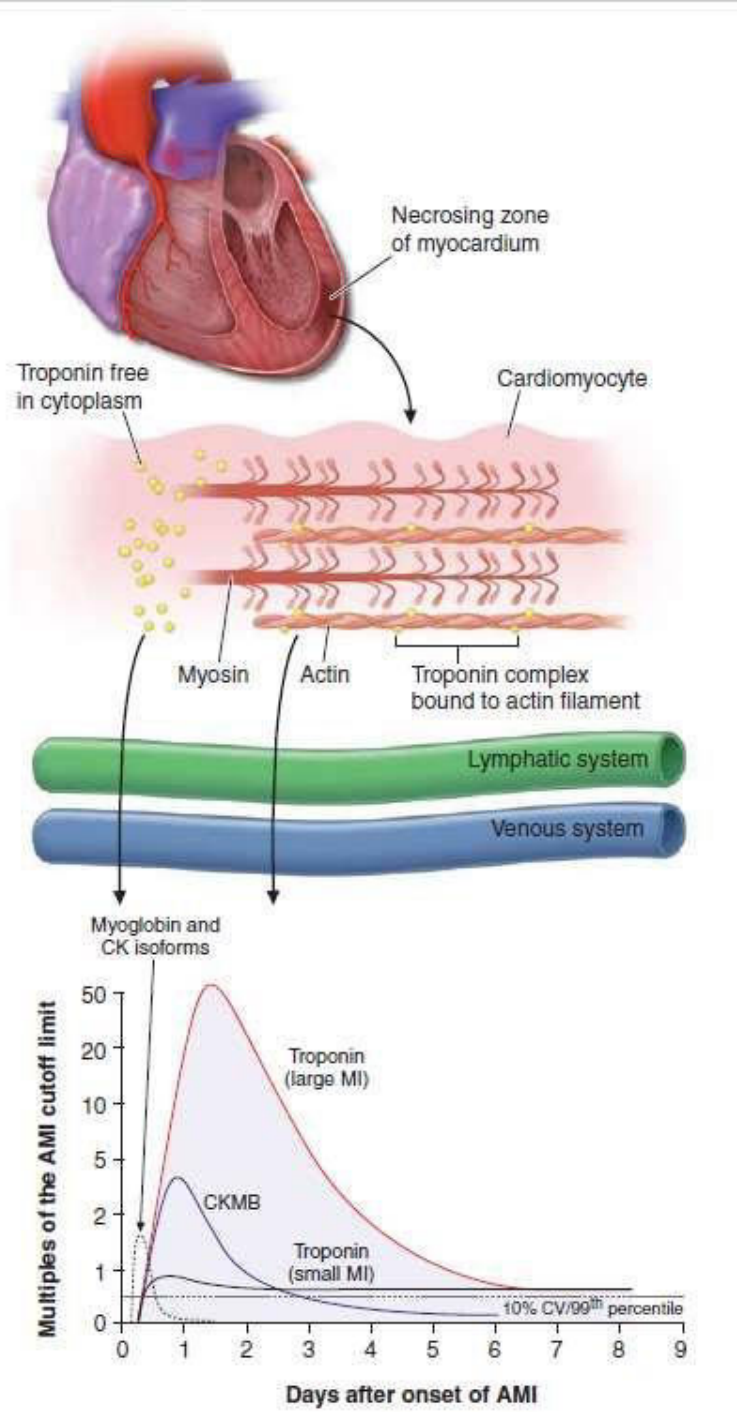
Normal ECG


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
Dev:
Speed: 25 mm/sec
Limb: 10 mm/mV
Chest: 10 mm/mV
F 50~ 0.15-150 Hz
P?
611
PHILIPS
REORDER M3708A

Serum cardiac markers

IDEAL MARKER:

- High concentration in myocardium
- Myocardium specific
- Released early in injury
- Proportionate to injury
- Non expensive testing



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

Diadvantages

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 re-infarction

CAUSES OF SERUM TROPONIN T AND I ELEVATIONS, INCLUDING BOTH ACUTE CORONARY SYNDROMES, NONCORONARY CARDIAC EVENTS, AND NONCARDIAC AILMENTS

Acute coronary syndrome/acute myocardial infarction
Shock of any form (cardiogenic, obstructive, distributive)
Myocarditis and myopericarditis
Cardiomyopathies
Acute congestive heart failure (pulmonary edema)
Sepsis
Pulmonary embolism
Renal failure
Sympathomimetic ingestions
Polytrauma
Burns
Acute CNS event
Rhabdomyolysis
Cardiac neoplasm, inflammatory syndromes, and infiltrative diseases
Congenital coronary anomalies
Extreme physical exertion

- Cardiac Imaging
  - ✓ 2D ECHO may reveal wall motion abnormality and LV dysfunction. It may also detect RV infarction, Ventricular aneurysms, pericardial effusion and LV Thrombus.
  - ✓ Myocardial perfusion imaging with Tl^{201} and Tc^{99m} - sestamibi reveals defect in most patient.

Time from symptom onset and likely outcome
< 1 hour Aborted heart attack or only little heart muscle damage
1–2 hours Minor heart muscle damage only(Golden hour for reperfusion)
2–4 hours Some heart muscle damage with moderate heart muscle salvage
4–6 hours Significant heart muscle damage with only minor heart muscle salvage
6–12 hours No heart muscle salvage (permanent loss) with potential infarct healing benefit
> 12 hours Reperfusion is not routinely recommended if the patient is asymptomatic and haemodynamically stable

Therapy STEMI

- Once STEMI is suspected or diagnosed, the immediate concerns are to ensure the patients stability and to intervene to limit infarct size by restoring the blood flow to the infarct artery as soon as possible..

Early Therapy

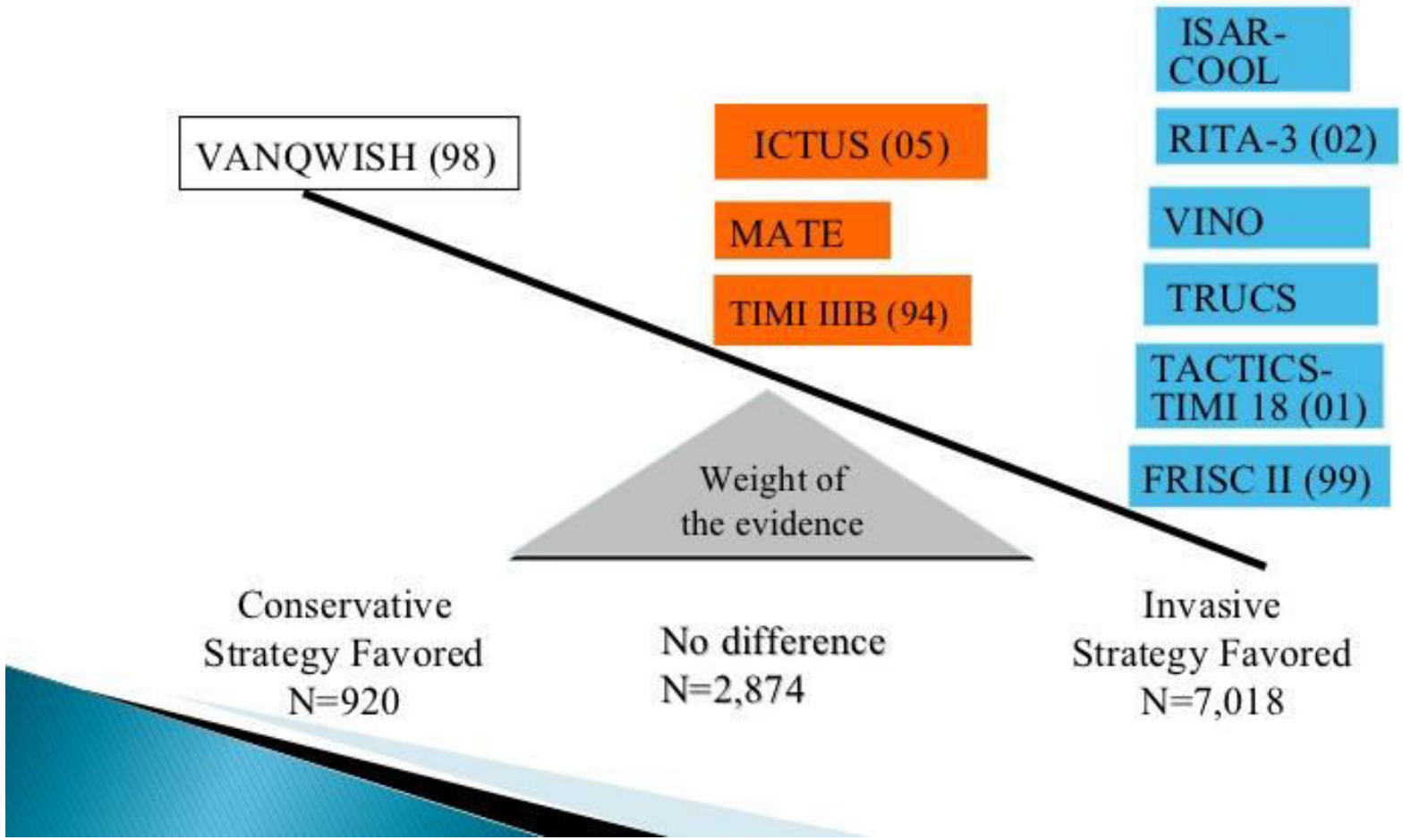
- Patients presenting with suspected myocardial ischemia should undergo a rapid evaluation, that is a 12 lead ECG, cardiac markers and related laboratory tests.
- Should be treated with oxygen
- Aspirin, 160 to 325 mg and P2Y2 inhibitors orally.
- Opiates relieve pain and also reduce anxiety.

REPERFUSION THERAPY

1. Pharmacological

2. Primary PCI

Invasive vs Conservative Strategy
Clinical Trials



Reperfusion Therapy

i. Percutaneous coronary intervention

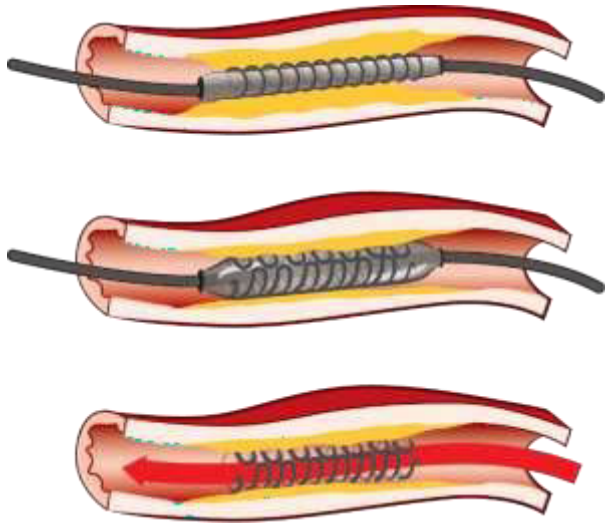
- It includes angioplasty, with or without deployment of an intracoronary Bare-metal or Drug-eluting stent, with support of pharmacological measures to prevent thrombosis.

- Recent meta-analyses comparing **direct PTCA with fibrinolytic therapy** have suggested lower rates of mortality and re-infarction among those receiving direct PTCA. Thus direct angioplasty, if performed in a **timely manner (ideally within 60 minutes)** by highly experienced personnel, may be the preferred method of revascularization, since it offers more complete revascularization with improved restoration of normal coronary blood flow and detailed information about coronary anatomy.

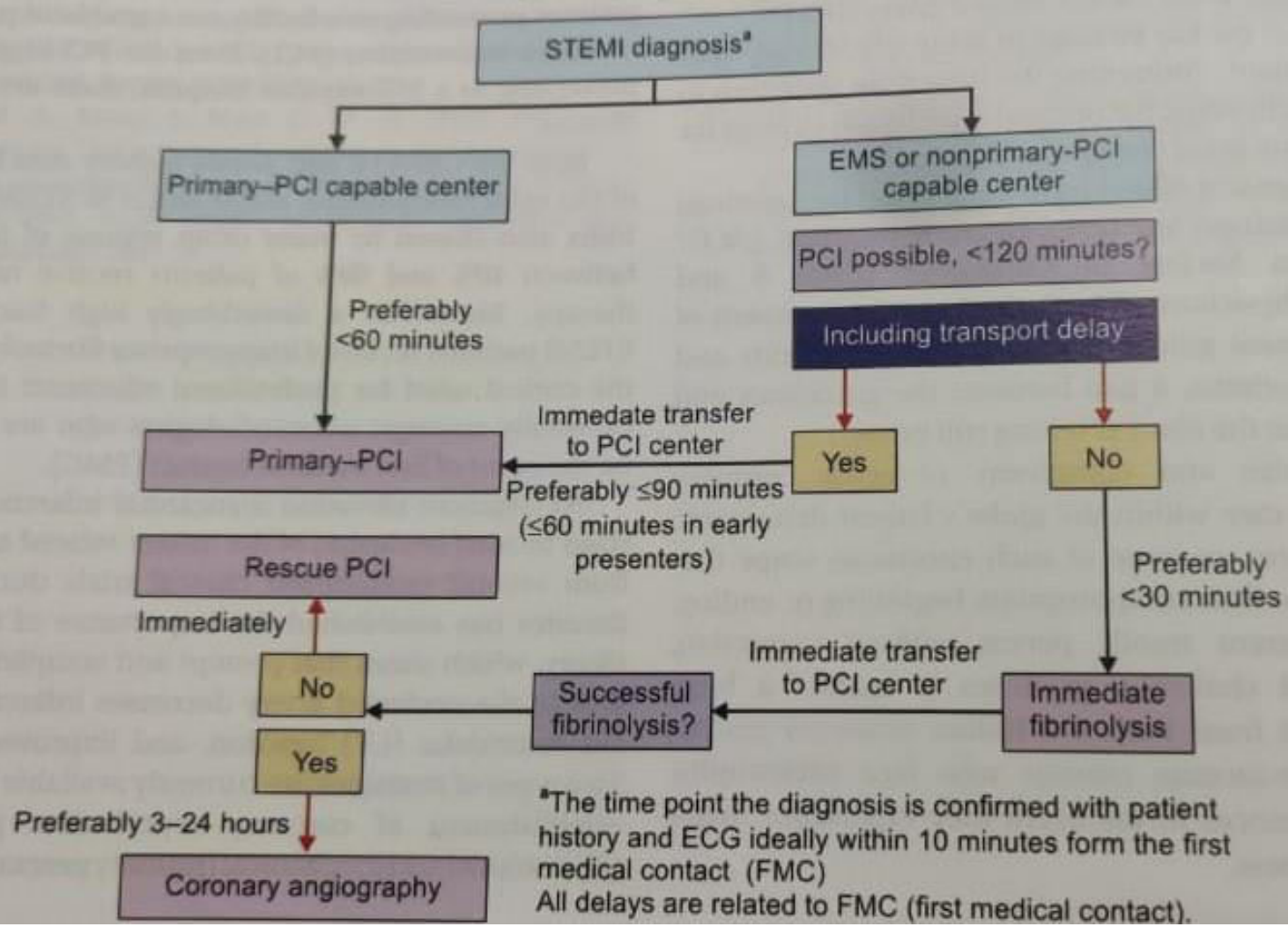
- Early recognition and diagnosis of STEMI are key to achieving the desired door-to-needle (or medical contact–to-needle) time for **initiation of fibrinolytic therapy** of **30 minutes** or door-to-balloon (or medical contact–to balloon) time for **PCI under 90 minutes**.
- Achieving reperfusion in timely matter correlates with improvement in ultimate infarct size, LV function, and survival.
- The ultimate goal is to restore adequate blood flow through the infarct-related artery to the infarct zone, as well as to limit microvascular damage and reperfusion injury.

Coronary Stents

- The use of coronary stents has been shown to reduce restenosis and adverse cardiac outcomes in both routine and high-risk PCI.
- A large, randomized, multicenter trial involving 900 patients did not show a difference in mortality at 6 months **but** did show improvement in ischemia driven target vessel revascularization and less angina in the stented patients compared to balloon angioplasty alone.
- Whether to use a bare metal stent or a drug-eluting stent in acute MI is a question that has not yet been addressed definitively by clinical trials; selection is currently based on both patient and angiographic characteristics.



FLOW CHART 1: ESC STEMI guidelines 2012



*The time point the diagnosis is confirmed with patient history and ECG ideally within 10 minutes form the first medical contact (FMC)
All delays are related to FMC (first medical contact).

Fibrinolytic therapy

- Early reperfusion of an occluded coronary artery is indicated for all eligible candidates.
- Fibrinolytic agents administered early in the course of an acute MI reduce infarct size, preserve LV function, and reduce short-term and long-term mortality.
- Multiple studies conclude that greatest mortality benefit is seen if fibrinolytics are administered within the first 12 hours of symptom onset, but it is reasonable to administer fibrinolytics to patients whose onset of symptoms exceeds 12 hours but who have continued clinical or ECG evidence of ischemia.

ACC/AHA Guidelines Fibrinolysis

- Class 1 recommendations
- ST elevation >0.1 in 2 or more contiguous leads
- Less than 12 hrs of onset symptoms
- NO need to have persistent symptoms

Fibrinolytic agents Used in ST elevation MI	
Streptokinase	1.5 million intravenous over 30-60 mins
Alteplase (tPA)	a 15-mg bolus, then 0.75 mg/kg (up to 50 mg) IV over the initial 30 minutes, and 0.5 mg/kg (up to 35 mg) over the next 60 minutes
Reteplase (rPA)	two 10-U boluses 30 minutes apart
Tenecteplase (TNK-tPA)	IV Bolus adjusted for weight (30mg if < 60 kg; 35mg if 60-70 kg; 40 mg if 70-80 kg, 45mg if 80-90 kg; 50mg if > 90 kg)

INDICATIONS FOR AND CONTRAINDICATIONS TO FIBRINOLYTIC THERAPY IN ACUTE MYOCARDIAL INFARCTION

Indications

- Symptoms consistent with acute myocardial infarction
- ECG showing 1-mm (0.1 mV) ST elevation in at least two contiguous leads, or new left bundle-branch block
- Presentation within 12 hours of symptom onset
- Absence of contraindications

Contraindications

Absolute

- Active internal bleeding
- Intracranial neoplasm, aneurysm, or AV malformation
- Stroke or neurosurgery within 6 weeks
- Trauma or major surgery within 2 weeks which could be a potential source of serious rebleeding
- Aortic dissection

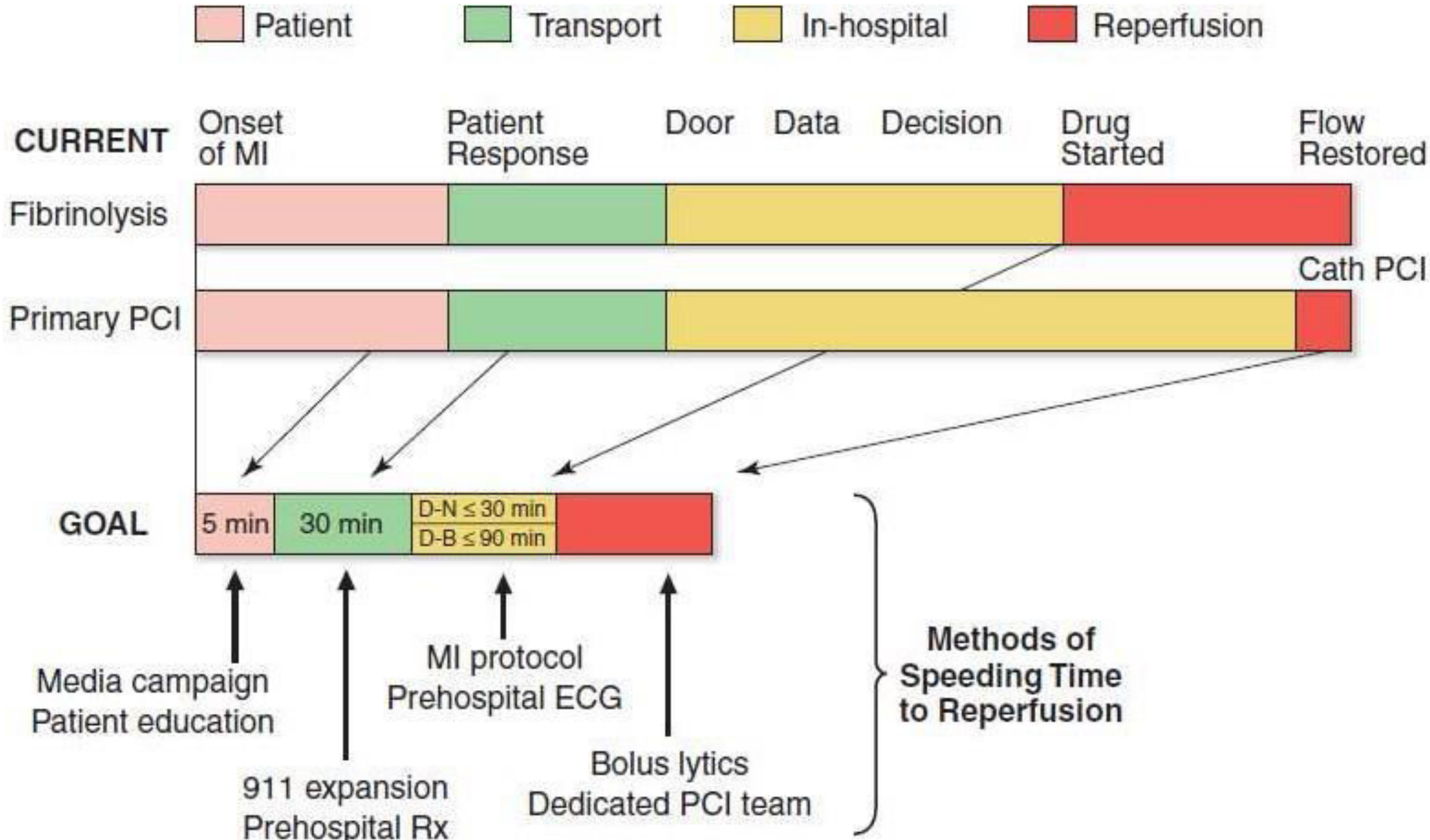
Relative

- Prolonged (>10 minutes) or clearly traumatic cardiopulmonary resuscitation*
- Noncompressible vascular punctures
- Severe uncontrolled hypertension (>200/110 mm Hg)*
- Trauma or major surgery within 6 weeks (but more than 2 weeks)
- Preexisting coagulopathy or current use of anticoagulants with INR > 2-3
- Active peptic ulcer
- Infective endocarditis
- Pregnancy
- Chronic severe hypertension

Indications	COR	LOE	References
Primary PCI*			
STEMI symptoms within 12 h	I	A	(379–382)
Severe heart failure or cardiogenic shock	I	B	(383,384)
Contraindications to fibrinolytic therapy with ischemic symptoms <12 h	I	B	(399,400)
Clinical and/or electrocardiographic evidence of ongoing ischemia between 12 and 24 h after symptom onset	IIa	B	(401–403)
Asymptomatic patients presenting between 12 and 24 h after symptom onset and higher risk	IIb	C	N/A
Noninfarct artery PCI at the time of primary PCI in patients without hemodynamic compromise	III: Harm	B	(404–408)
Delayed or elective PCI in patients with STEMI			
Clinical evidence for fibrinolytic failure or infarct artery reocclusion	IIa	B	(385,386)
Patent infarct artery 3 to 24 h after fibrinolytic therapy	IIa	B	(390,391)
Ischemia on noninvasive testing	IIa	B	(410,411)
Hemodynamically significant stenosis in a patent infarct artery >24 h after STEMI	IIb	B	(413–417)
Totally occluded infarct artery >24 h after STEMI in a hemodynamically stable asymptomatic patient without evidence of severe ischemia	III: No Benefit	B	(418–420)

***Systems goal of performing primary PCI within 90 min of first medical contact when the patient presents to a hospital with PCI capability (394,395) (*Class I; LOE: B*) and within 120 min when the patient presents to a hospital without PCI capability (396–398) (*Class I; LOE: B*).**

COR indicates class of recommendation; LOE, level of evidence; N/A, not applicable; PCI, percutaneous coronary intervention; and STEMI, ST-elevation myocardial infarction.


Patient
Transport
In-hospital
Reperfusion
CURRENT
Onset of MI
Patient Response
Door
Data
Decision
Drug Started
Flow Restored
Fibrinolysis
Cath PCI
Primary PCI
GOAL
5 min
30 min
D-N ≤ 30 min
D-B ≤ 90 min
Media campaign
Patient education
911 expansion
Prehospital Rx
MI protocol
Prehospital ECG
Bolus lytics
Dedicated PCI team
Methods of Speeding Time to Reperfusion

Adjunctive therapy

1. Aspirin:

- Reduce mortality in acute infarction to the same degree as fibrinolytic therapy, and its effects are additive to fibrinolytics. In addition, aspirin reduces the risk of reinfarction.
- Unless contraindicated, all patients with a suspected ACS (STEMI, NSTEMI, UA) should be given aspirin as soon as possible.

2. Thienopyridines:

Patients presenting with MI should be considered for a thienopyridine regardless of whether or not they underwent reperfusion therapy. The duration of thienopyridine use in this population has yet to be defined.

3. Anticoagulants:

Administration of full-dose heparin after fibrinolytic therapy with tPA is essential to diminish re-occlusion after successful reperfusion.

4. Glycoprotein IIb/IIIa Receptor antagonist:

Role in STEMI uncertain.

Not routinely recommended.

Oral Antiplatelet Therapy	
Aspirin	Initial dose of 162–325 mg nonenteric formulation followed by 75–162 mg/d of an enteric or a nonenteric formulation
Clopidogrel	Loading dose of 300–600 mg followed by 75 mg/d
Prasugrel	Pre-PCI: Loading dose 60 mg followed by 10 mg/d
Intravenous Antiplatelet Therapy	
Abciximab	0.25 mg/kg bolus followed by infusion of 0.125 µg/kg per min (maximum 10 µg/min) for 12 to 24 h
Eptifibatide	180 µg/kg bolus followed by infusion of 2.0 µg/kg per min for 72 to 96 h
Tirofiban	0.4 µg/kg per min for 30 min followed by infusion of 0.1 µg/kg per min for 48 to 96 h

Heparins[a]	
Unfractionated Heparin (UFH)	Bolus 60–70 U/kg (maximum 5000 U) IV followed by infusion of 12–15 U/kg per h (initial maximum 1000 U/h) titrated to a PTT 50–70 s
Enoxaparin	1 mg/kg SC every 12 h; the first dose may be preceded by a 30-mg IV bolus; renal adjustment to 1 mg/kg once daily if creatine Cl <30 cc/min
Fondaparinux	2.5 mg SC qd
Bivalirudin	Initial bolus intravenous bolus of 0.1 mg/kg and an infusion of 0.25 mg/kg per hour. Before PCI, an additional intravenous bolus of 0.5 mg/kg was administered, and the infusion was increased to 1.75 mg/kg per hour.

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"This is one of those new miracle drugs.
If you can afford it, it's a miracle."

5. Nitrates:

- They **reduce** myocardial oxygen demand by decreasing preload and afterload, and they may also **improve** myocardial oxygen supply by increasing sub-endocardial perfusion and collateral blood flow to the ischemic region.
- Also reduce platelet aggregation.
- Nitrates are first-line agents for the symptomatic relief of angina pectoris and when MI is complicated by congestive heart failure.

6. β Blockers:

- Routine use of *intravenous* beta-blockers in the absence of systemic hypertension is no longer recommended.
- **Relative contraindications** to oral beta-blockers include,
  - ✓ heart rate less than 60 bpm,
  - ✓ systolic arterial pressure less than 100 mm Hg,
  - ✓ moderate or severe LV failure,
  - ✓ signs of peripheral hypoperfusion,
  - ✓ shock,
  - ✓ PR interval greater than 0.24 second, second- or third-degree AV block,
  - ✓ active asthma, or
  - ✓ reactive airway disease.

7. ACE inhibitors

Improve hemodynamic, functional capacity and symptoms, and survival in patients with chronic congestive heart failure. Moreover, ACE inhibitors prevent the development of congestive heart failure in patients with asymptomatic LV dysfunction

8. Lipid Lowering agents

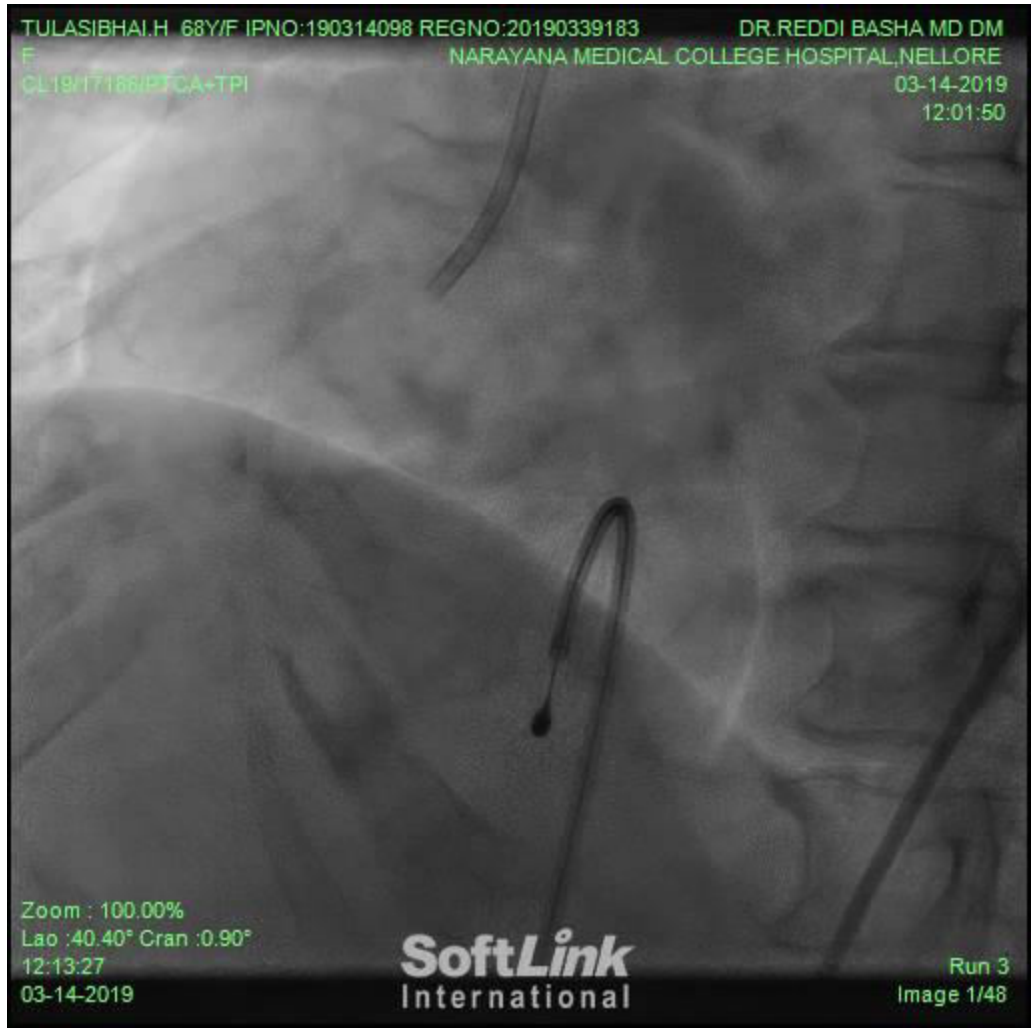
- All patients with ACS should be started on a statin prior to discharge unless there is a contraindication

9. Calcium channel blockers

- May be useful for patients whose postinfarction course is complicated by recurrent angina, because these agents not only reduce myocardial oxygen demand but also inhibit coronary vasoconstriction.

Complications

- Ventricular dysfunction
- Cardiogenic shock
- Right Ventricular Infarction
- Pericarditis,Ventricular septal rupture, Free wall rupture.
- Thromboembolism
- Left ventricular aneurysm
- Sinus bradycardia
- AV Block
- Ventricular tachycardia and fibrillation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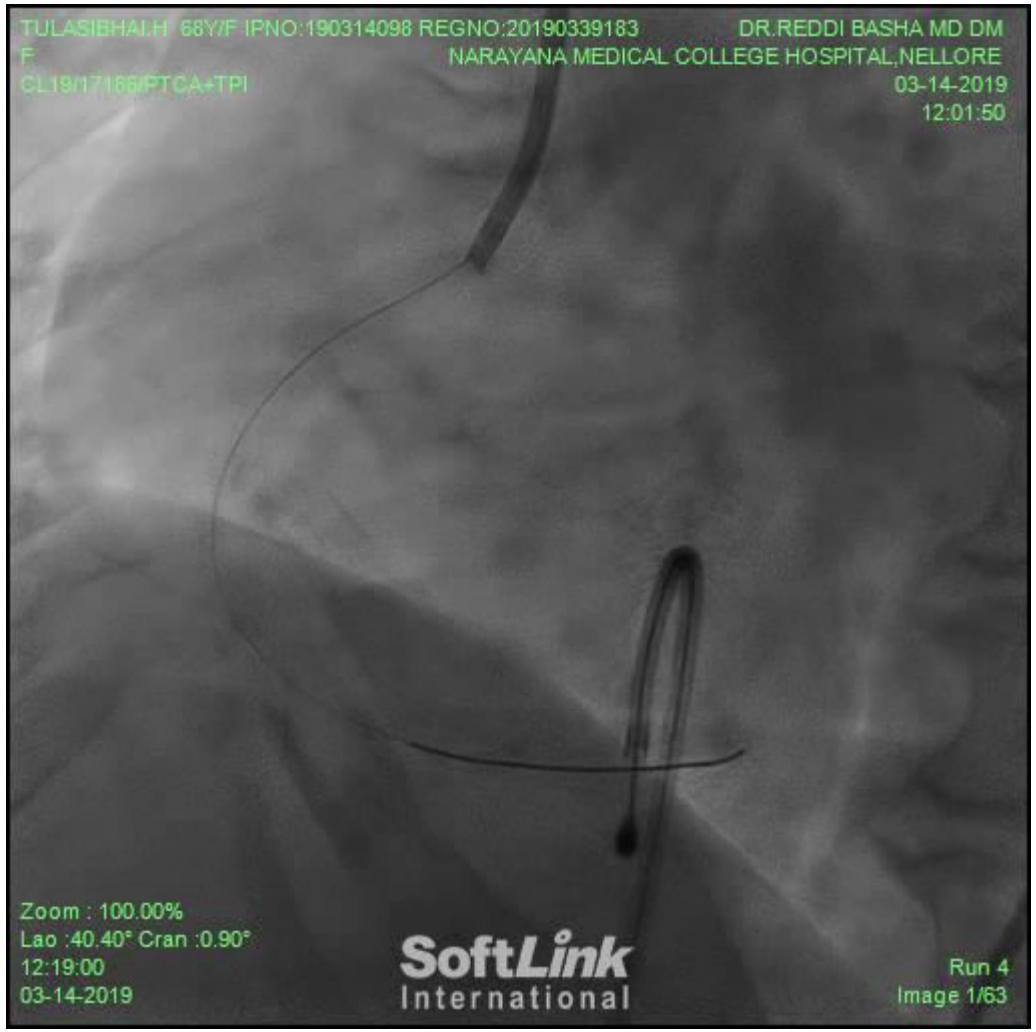
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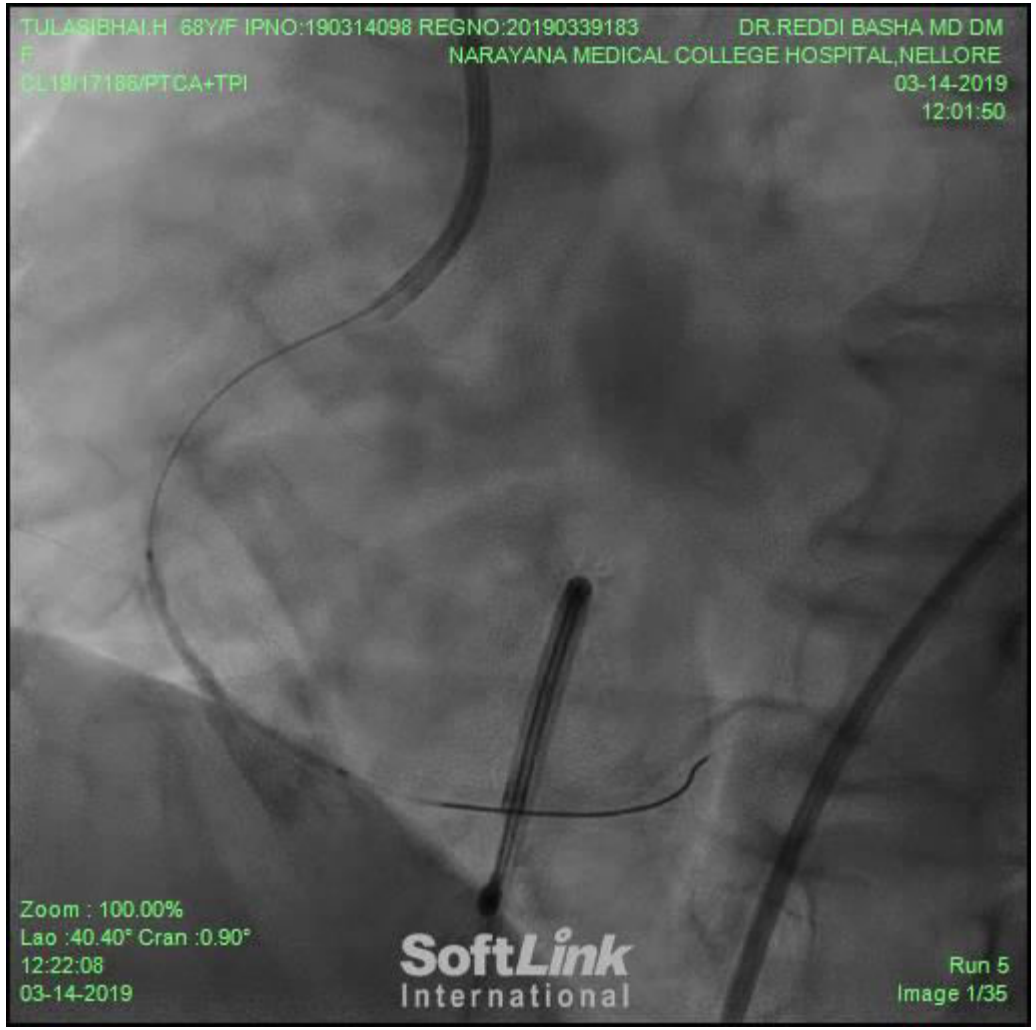
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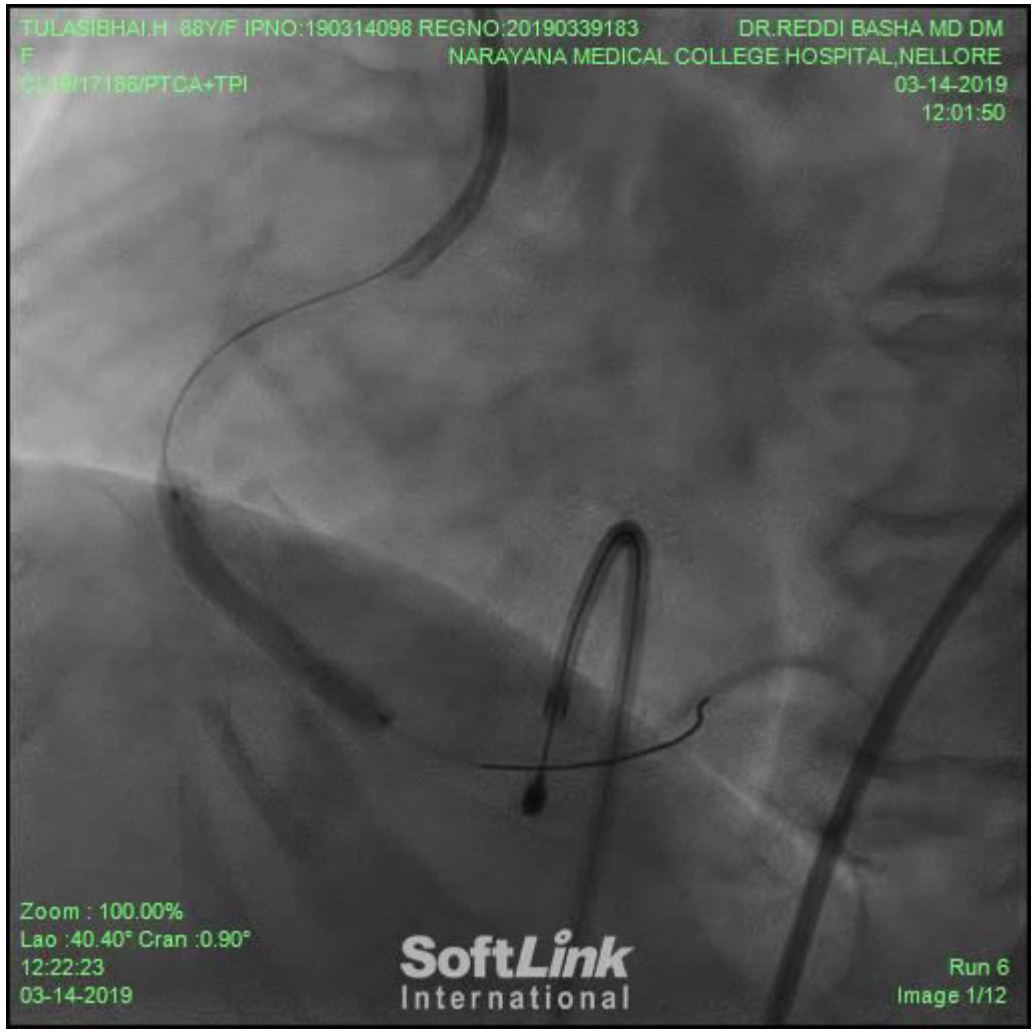
Image 1/35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CL19I17186/PTCA+TPI
DR.REDDI BASHA MD DM
NARAYANA MEDICAL COLLEGE HOSPITAL,NELLORE
03-14-2019
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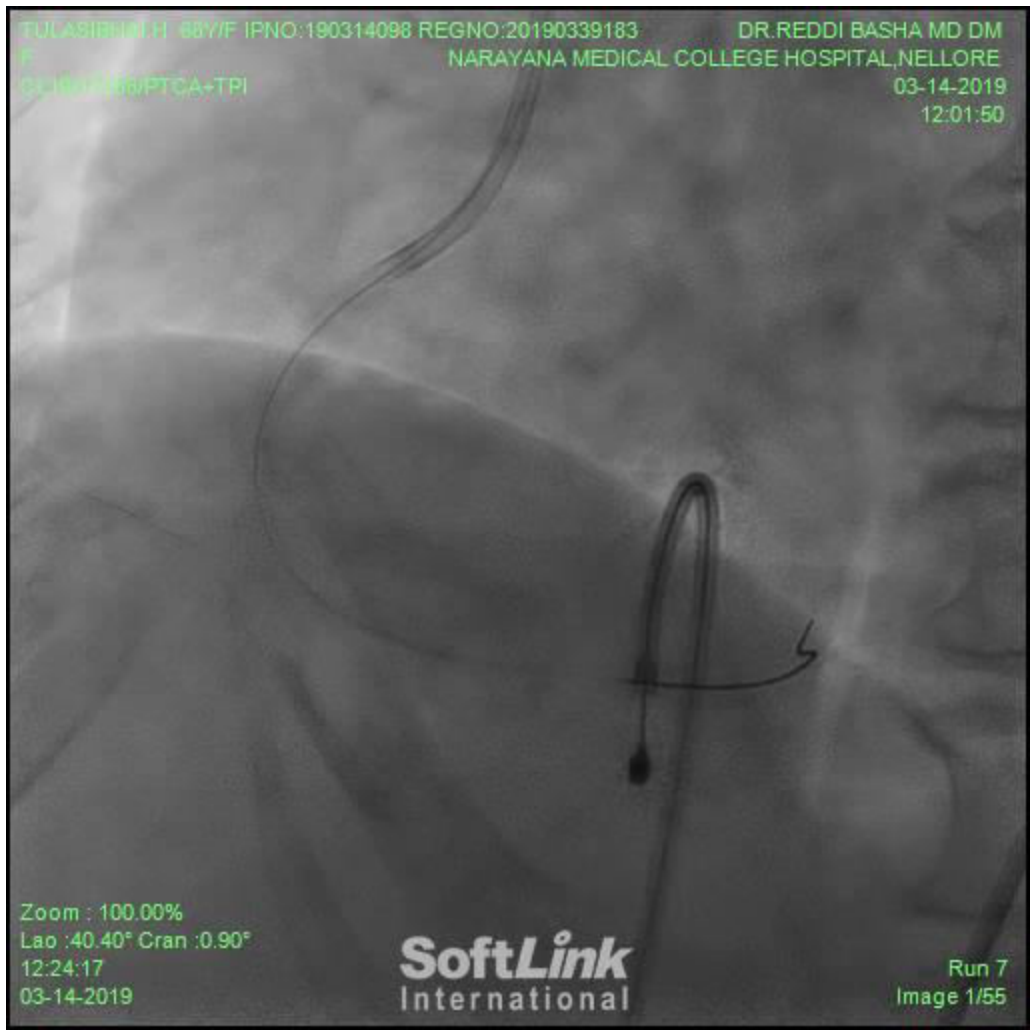
Lao :40.40° Cran :0.90°
12:22:23
03-14-2019
SoftLink
International
Run 6
Image 1/12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DR.REDDI BASHA MD DM
F
NARAYANA MEDICAL COLLEGE HOSPITAL,NELLORE
03-14-2019
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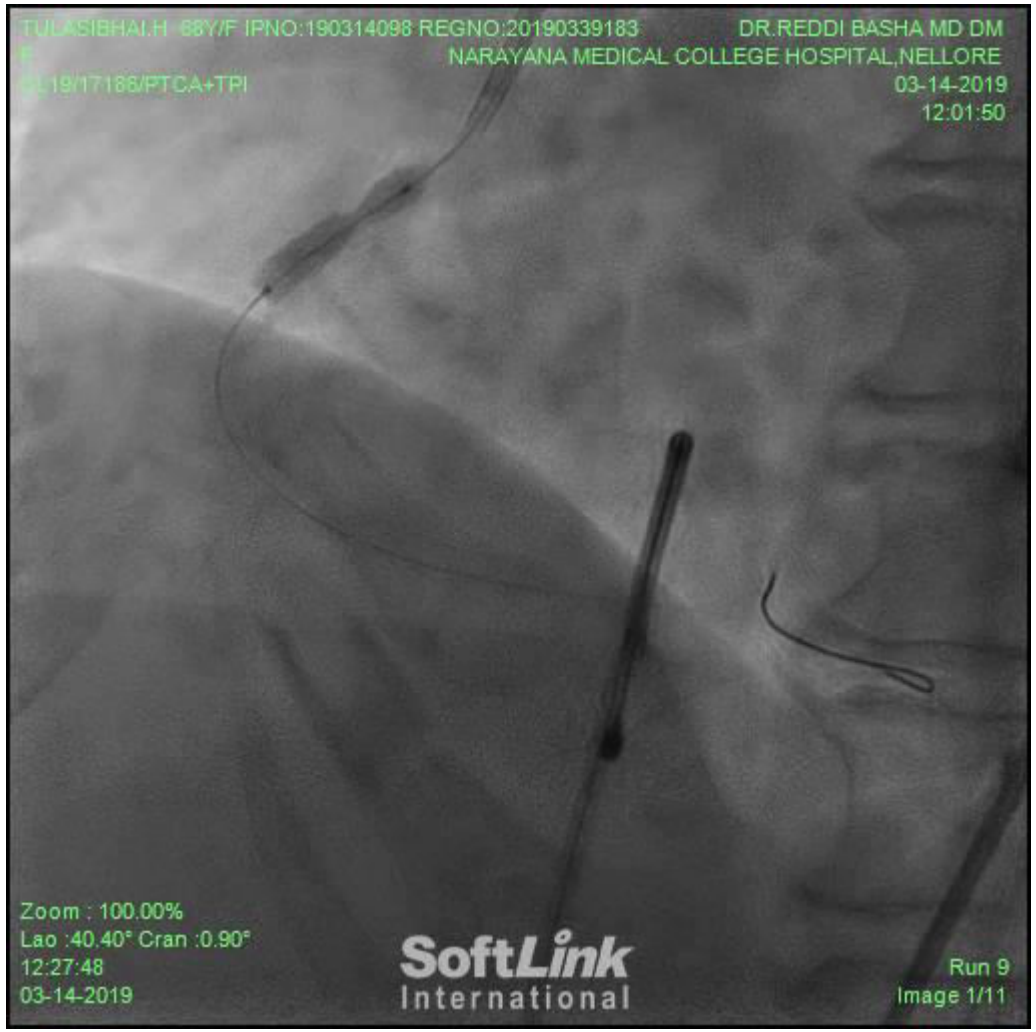
03-14-2019
SoftLink
International
Run 7
Image 1/55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DR.REDDI BASHA MD DM
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NARAYANA MEDICAL COLLEGE HOSPITAL,NELLORE
19/17186/PTCA+TPI
03-14-2019
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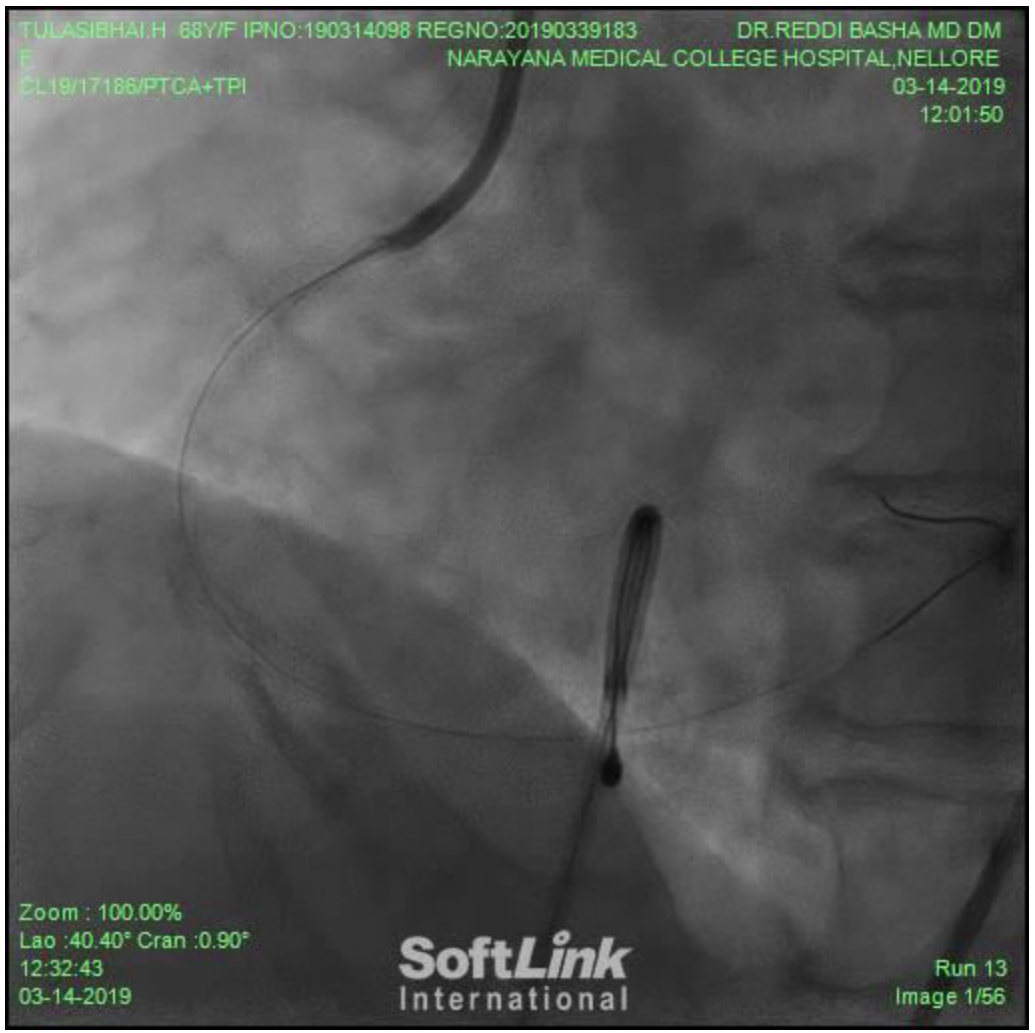
03-14-2019
SoftLink
International
Run 9
Image 1/11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DR.REDDI BASHA MD DM
NARAYANA MEDICAL COLLEGE HOSPITAL,NELLORE
03-14-2019
12:01:50
Zoom : 100.00%
Lao :40.40° Cran :0.90°
12:32:43
03-14-2019
SoftLink
International
Run 13
Image 1/56


TULASIBHAI.H 68Y/F IPNO:190314098 REGNO:20190339183
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CL19/17186/PTCA+TPI
DR.REDDI BASHA MD DM
NARAYANA MEDICAL COLLEGE HOSPITAL,NELLORE
03-14-2019
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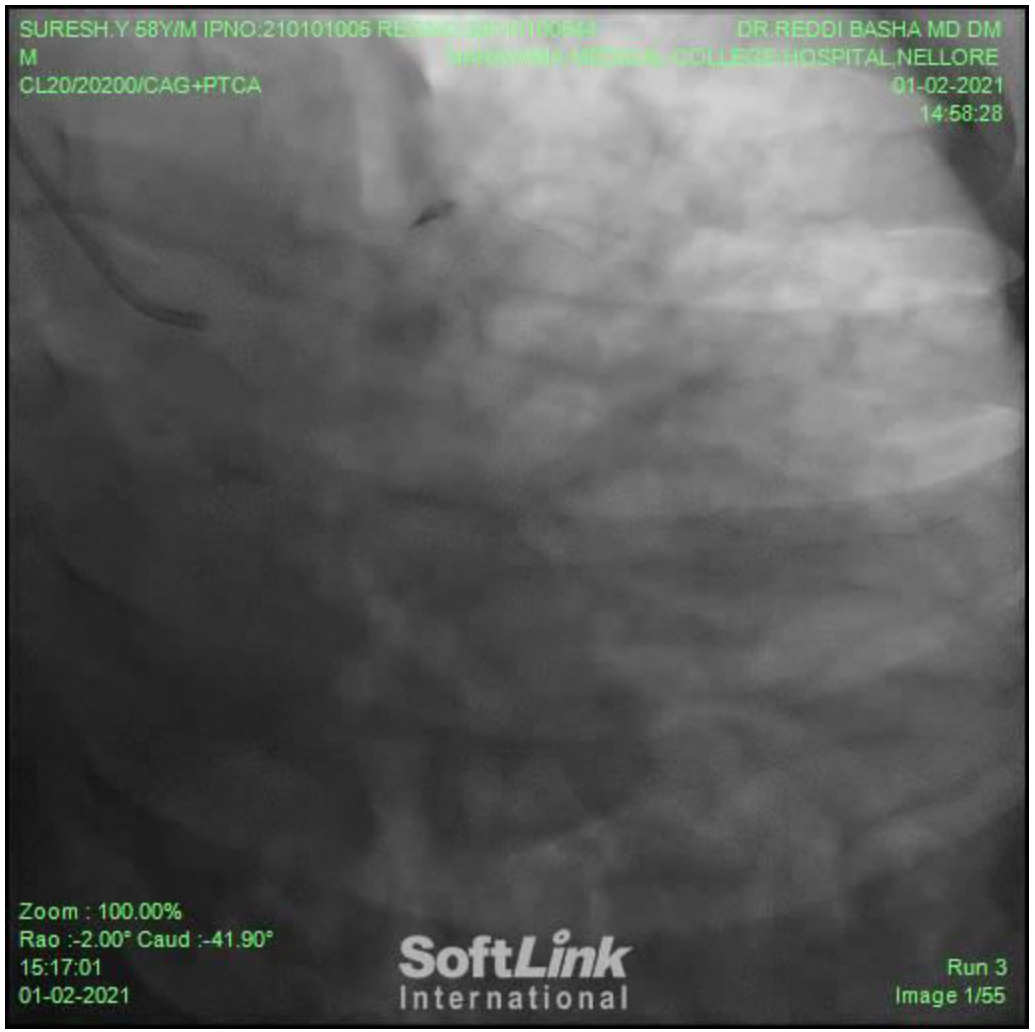
Lao :21.30° Cran :42.60°
12:34:41
03-14-2019
SoftLink
International
Run 15
Image 1/68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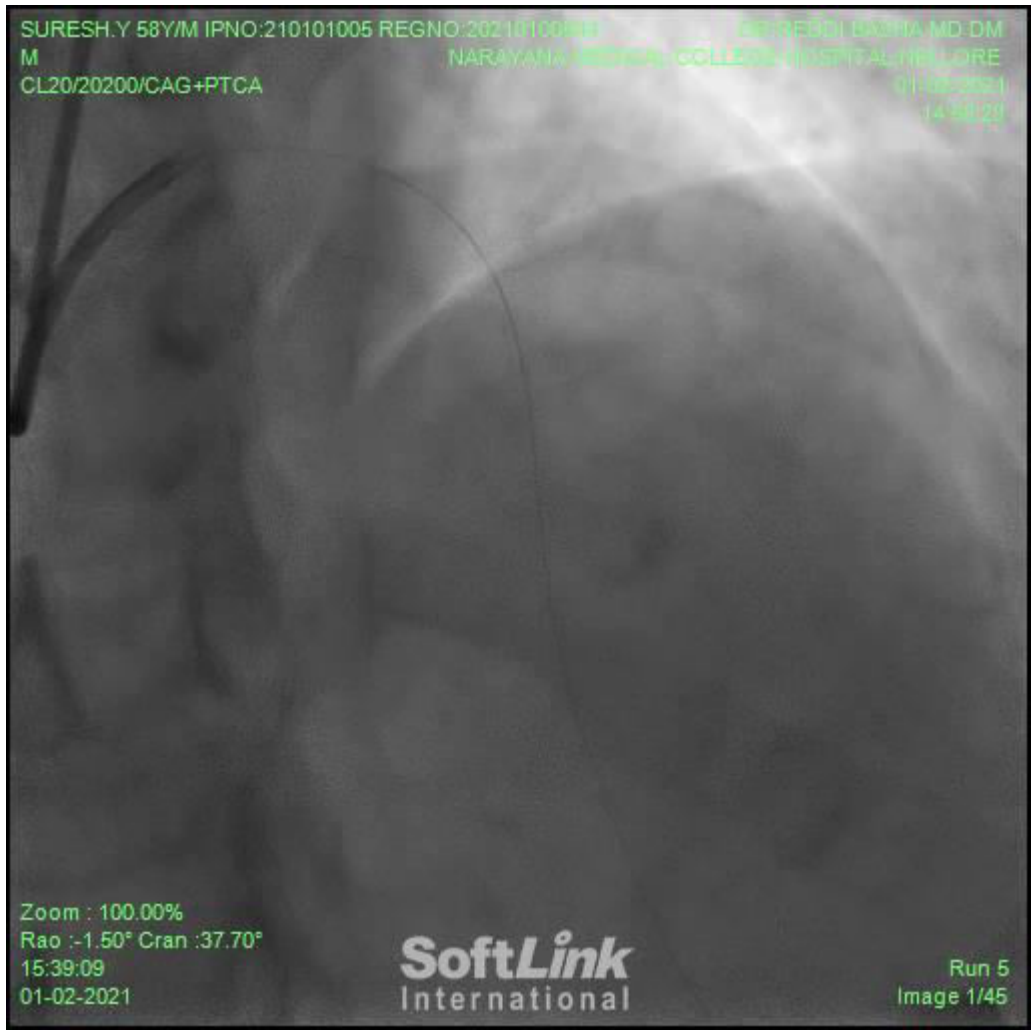
CL20/20200/CAG+PTCA
DR.REDDI BASHA MD DM
NARAYANA MEDICAL COLLEGE HOSPITAL,NELLORE
01-02-2021
14:58:28
Zoom : 100.00%
Rao :-2.00° Caud :-41.90°
15:17:01
01-02-2021
SoftLink
International
Run 3
Image 1/55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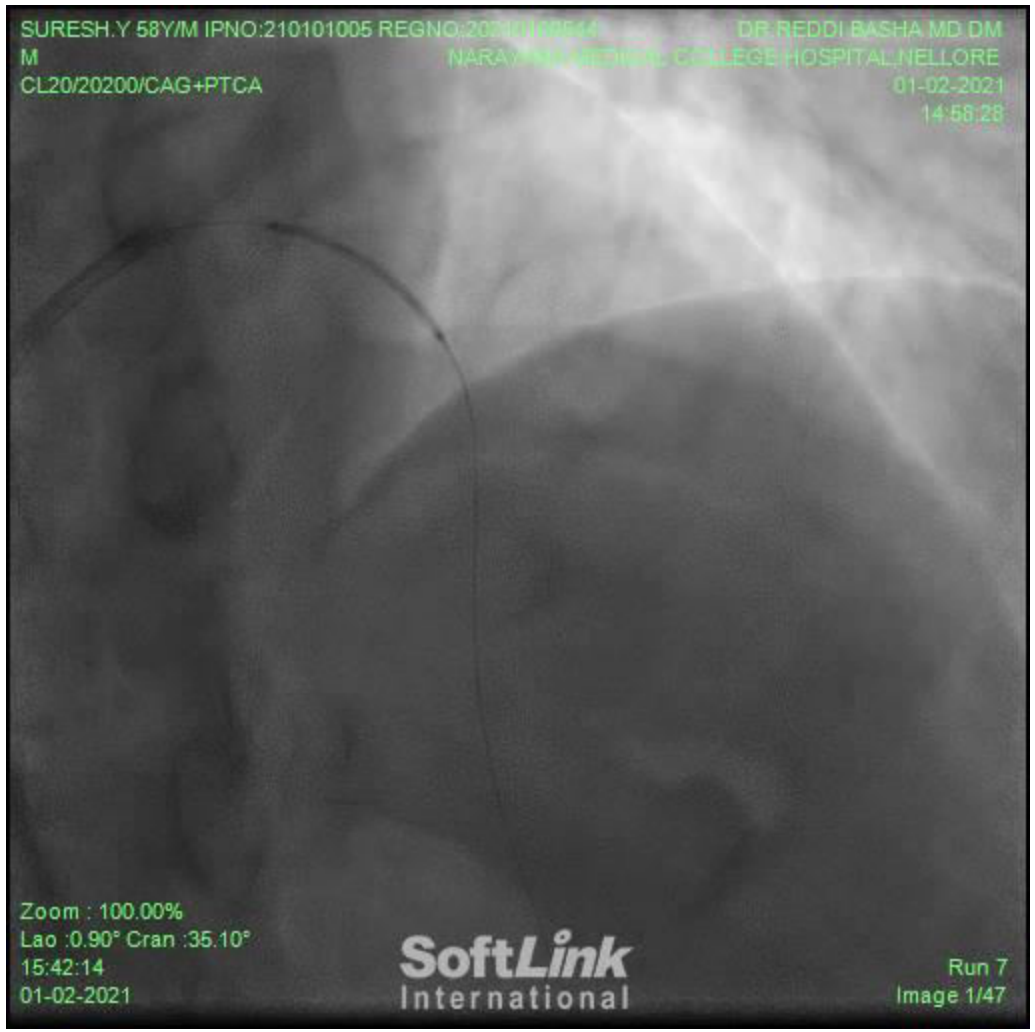
CL20/20200/CAG+PTCA
DR.REDDI BASHA MD DM
NARAYANA MEDICAL COLLEGE HOSPITAL,NELLORE
Zoom : 100.00%
Rao :-1.50° Cran :37.70°
15:39:09
01-02-2021
SoftLink
International
Run 5
Image 1/45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CL20/20200/CAG+PTCA
DR.REDDI BASHA MD DM
NARAYANA MEDICAL COLLEGE HOSPITAL,NELLORE
01-02-2021
14:58:28
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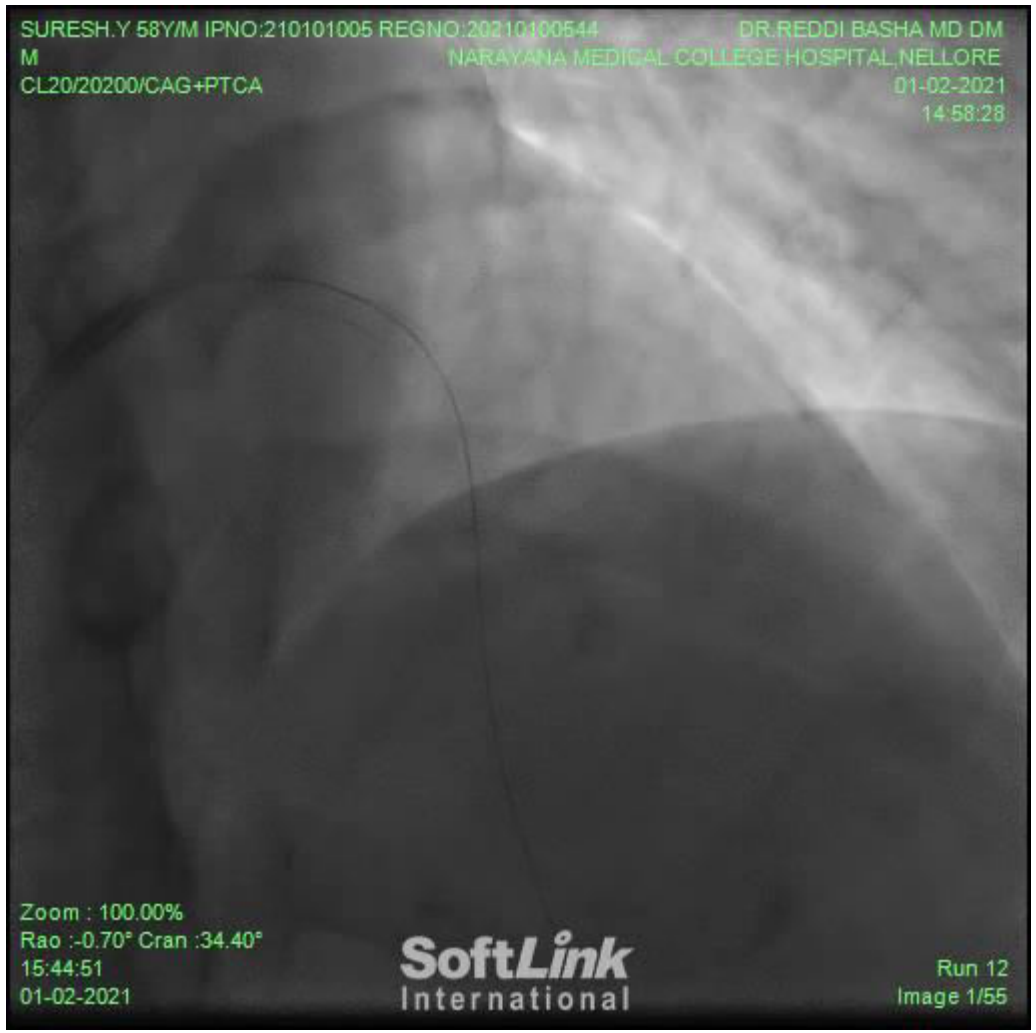
Lao :0.90° Cran :35.10°
15:42:14
01-02-2021
SoftLink International
Run 7
Image 1/47


SURESH.Y 58Y/M IPNO:210101005 REGNO:20210100544
M
CL20/20200/CAG+PTCA
DR.REDDI BASHA MD DM
NARAYANA MEDICAL COLLEGE HOSPITAL,NELLORE
01-02-2021
14:58:28
Zoom : 100.00%
Rao :-0.70° Cran :34.40°
15:44:51
01-02-2021
SoftLink International
Run 12
Image 1/55

Thank you