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Percutaneous Coronary Intervention in Acute Myocardial Infarction

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Abstract

Coronary artery disease (CAD) is a cardiovascular disease which has been found to be the leading cause of death in both developed and developing countries. CAD is an atherosclerotic disease which is inflammatory in nature, manifested by stable angina, unstable angina, myocardial infarction (MI), or sudden cardiac death. Preventive and therapeutic measures have substantially improved the prognosis of patients suffering from CAD or other cardiovascular diseases over the past few decades.Primary percutaneous coronary intervention (PCI) has replaced thrombolysis as the predominant revascularization method throughout the last ten years. Nevertheless, the provision of primary PCI within evidence-based timeframes is a difficult task, and levels of healthcare provision vary significantly across the globe. Consequently, even in the most favorable circumstances of a swift initial diagnosis, there is a possibility of lengthy transfer delays to the catheter laboratory. A delay in reperfusion may lead to a worse prognosis. In-hospital mortality increases after primary PCI ranged from 3.0% to 4.8% for 30- and 180-minute door-to-balloon durations, respectively Moreover, the 12-month mortality rate increases by 7.5% for every 30-minute delay.

Keywords: Percutaneous Coronary Intervention, Acute Myocardial Infarction, Acute ST elevation myocardial infarction.

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1. Introduction

The term myocardial injury should be used when there is evidence of elevated cardiac troponin values (cTn) with at least one value above the 99th percentile upper reference limit (URL). The myocardial injury is considered acute if there is a rise and/or fall of cTn values [1].

The term acute myocardial infarction 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 one value above the 99th percentile URL and at least one 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 (not for types 2 or 3 MIs) [1].

Post-mortem demonstration of acute atherothrombosis in the artery supplying the infarcted myocardium meets criteria for type 1 MI.

Evidence of an imbalance between myocardial oxygen supply and demand unrelated to acute atherothrombosis meets criteria for type 2 MI.

Cardiac death in patients with symptoms suggestive of myocardial ischemia and presumed new ischemic ECG changes before cTn values become available or abnormal meets criteria for type 3 MI [1].

Criteria for coronary procedure-related myocardial infarction (types 4 and 5 MI) [2]

- Percutaneous coronary intervention (PCI) related MI is termed type 4a MI.
- Coronary artery bypass grafting (CABG) related MI is termed type 5 MI.
- Coronary procedure-related MI ≤ 48 hours after the index procedure is arbitrarily defined by an elevation of cTn values > 5 times for type 4a MI and > 10 times for type 5 MI of the 99th percentile URL in patients with normal baseline values.
- Patients with elevated pre-procedural cTn values, in whom the pre-procedural cTn level are stable (≤ 20% variation) or falling, must meet the criteria for a > 5 or > 10-fold increase and manifest a change from the

baseline value of > 20%. In addition, with at least one of the following:

- New ischemic ECG changes (this criterion is related to type 4a MI only).
- Development of new pathological Q waves.
- Imagine evidence of loss of viable myocardium that is presumed to be new and, in a pattern, consistent with an ischemic etiology.
- Angiographic findings consistent with a procedural flow-limiting complication such as coronary dissection, occlusion of a major epicardial artery or graft, side-branch occlusion-thrombus, disruption of collateral flow or distal embolization.

Isolated development of new pathological Q waves meets the type 4a MI or type 5 MI criteria with either revascularization procedure if cTn values are elevated and rising but less than the pre-specified thresholds for PCI and CABG.

Other types of type 4 MI include type 4b MI stent thrombosis and type 4c MI restenosis that both meet type 1 MI criteria. Post-mortem demonstration of a procedure-related thrombus meets the type 4a MI criteria or type 4b MI criteria if associated with a stent [1].

Criteria for prior or silent/unrecognized myocardial infarction

Any one of the following criteria meets the diagnosis for prior or silent/unrecognized MI:

- Abnormal Q waves with or without symptoms in the absence of non-ischemic causes.
- Imaging evidence of loss of viable myocardium in a pattern consistent with ischemic etiology
- Patho-anatomical findings of a prior MI [1].

Diagnostic ECG criteria of acute myocardial infarction

Diagnostic ST elevation (STE) in the absence of left ventricular hypertrophy (LVH) or left bundle-branch block (LBBB) is defined as:

Measured at the J point, should be found in two contiguous leads and be:

- ≥0.25 mV in men below the age of 40 years,
- ≥0.2 mV in men over the age of 40 years, and/or
- ≥0.15 mV in women in leads V2–V3 and/or
- ≥0.1 mV in other leads [3].
- Electrocardiographic changes associated with previous myocardial infarction (in the absence of left ventricular hypertrophy and LBBB)
- Any Q wave in leads V2-V3 ≥0.02 sec or a QS complex in leads V2 and V3
- Q wave ≥0.03 sec and ≥0.1-mV deep or QS complex in leads I, II, aVL, aVF, or V4-V6 in any 2 leads of a contiguous lead grouping (I, aVL;V1-V6; II, III, aVF)
- R wave ≥0.04 sec in V1-V2 and R/S ≥1 with a concordant positive T wave in absence of a conductions defect [4].
- New or presumably new LBBB is considered a STEMI equivalent [3].
- ST depression in ≥2 precordial leads (V1–V4) may indicate transmural posterior injury [5].
- In patients with inferior myocardial infarction, it is advisable to record right precordial leads (V3R and V4R) & posterior precordial leads (V7 – V9) seeking ST elevation [5].
- Patients with new Q waves and ST-segment elevation diagnostic of STEMI in one territory often have ST-segment depression in other territories. These additional ST-segment changes, which imply a poor prognosis, result either from ischemia in a territory other than the area of infarction, termed ischemia at a distance, or from reciprocal electrical phenomena [6].
- Multi-lead ST depression with coexistent ST elevation in lead aVR has been described in patients with left main or proximal left anterior descending artery occlusion [7].
- Rarely, hyperacute T-wave changes may be observed in the very early phase of STEMI, before the development of ST elevation [6].
- In the presence of undetermined onset LBBB, the ECG diagnosis of acute myocardial infarction is difficult, but often possible if marked ST abnormalities are present. Algorithms offered to assist the diagnosis, but they do not provide diagnostic certainty (Sgarbossa's criteria) [8].
- The original three criteria used to diagnose infarction in patients with LBBB are:

• Concordant ST elevation > 1mm in leads with a positive QRS complex (score 5)
• Concordant ST depression > 1 mm in V1-V3 (score 3)
• Excessively discordant ST elevation > 5 mm in leads with a -ve QRS complex (score 2).

A total score of ≥ 3 is reported to have a specificity of 90% for diagnosing myocardial infarction [8].

Smith's Modification of Sgarbossa's Criteria

• ≥ 1 lead with ≥1 mm of concordant ST elevation
• ≥ 1 lead of V1-V3 with ≥ 1 mm of concordant ST depression
• ≥ 1 lead anywhere with ≥ 1 mm STE and proportionally excessive discordant STE, as defined by ≥ 25% of the depth of the preceding S-wave [9]. (Figure 1)

New ECG algorithm BARCELONA criteria:

based on the presence of concordant ST deviation ≥1 mm (0.1 mV) in any ECG lead and/or discordant ST deviation ≥1 mm (0.1 mV) in leads with max (R|S) voltage ≤6 mm (0.6 mV) was identified.

This algorithm significantly improved the diagnosis of AMI as compared with previous ECG rules, achieving a diagnostic performance for AMI similar to that of ECG in patients without LBBB [10].

Pathology of STEMI:

Atherosclerosis (as discussed in 1st chapter)

More than 80% of acute myocardial infarcts are the result of coronary atherosclerosis with superimposed luminal thrombus [4].

Non-atherosclerotic Causes of Acute Myocardial Infarction

Other pathological processes other than atherosclerosis can involve coronaries and result in STEMI as [4]:

- Arteritis as polyarteritis nodosa, systemic lupus erythematosus
- Trauma to coronary arteries could be caused iatrogenic or by radiation.
- Metabolic diseases casing coronary mural thickening.
- Spasm of coronary arteries as prinzemetal angina
- Aortic dissection

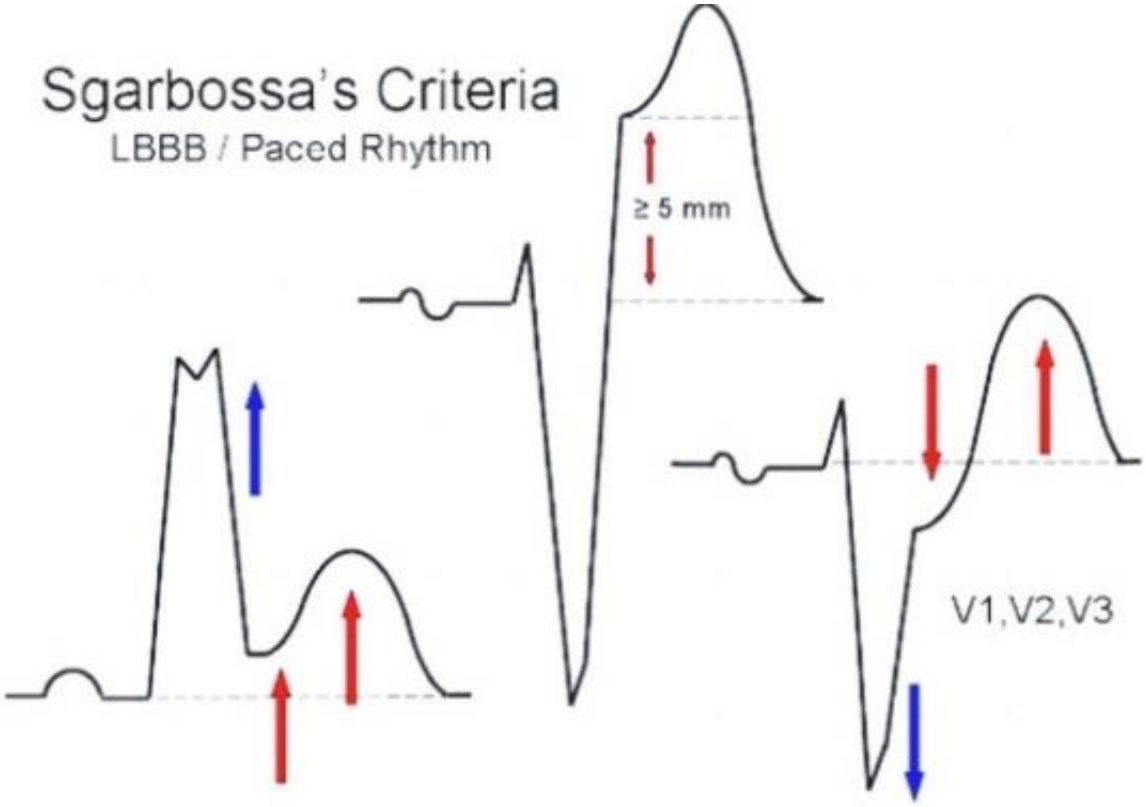

Figure 1: Sgarbossa criteria for diagnosis of acute myocardial infarction in the setting of left bundle branch block [8].

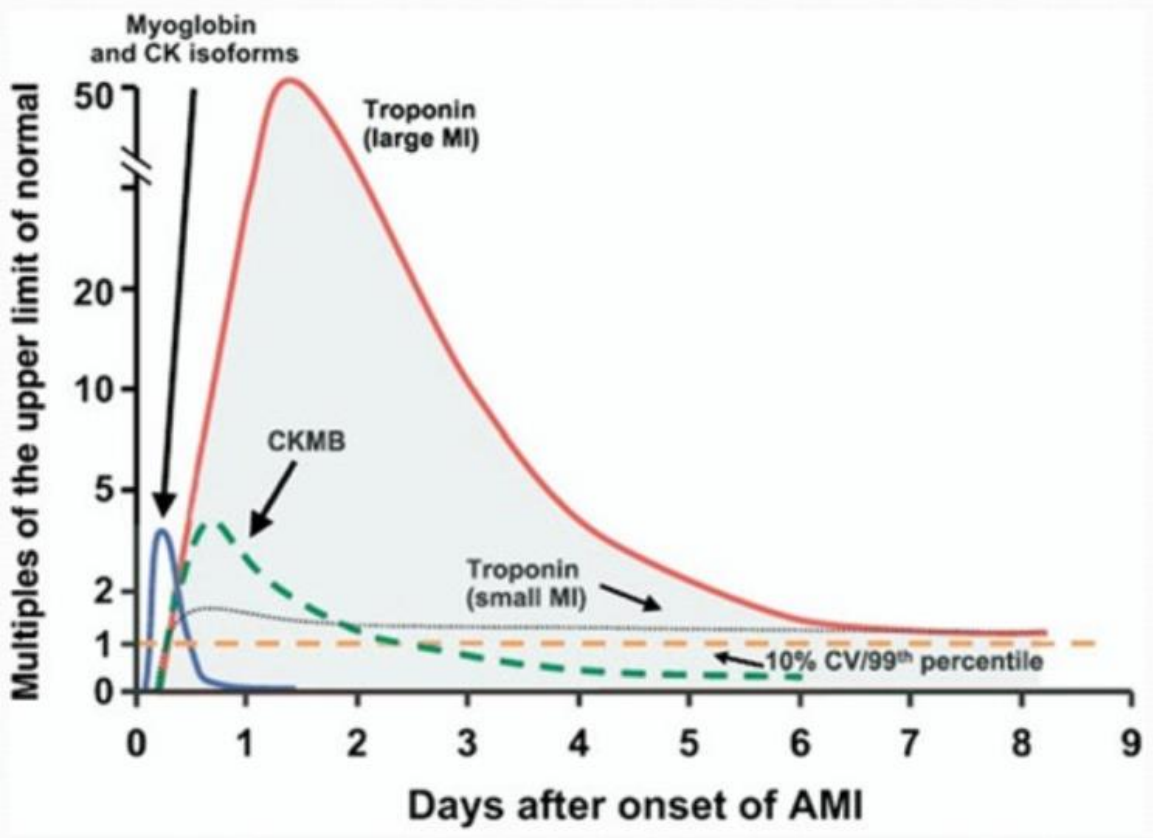

Figure 2: Timing of Release of Various Biomarkers After Acute Myocardial Infarction [12]

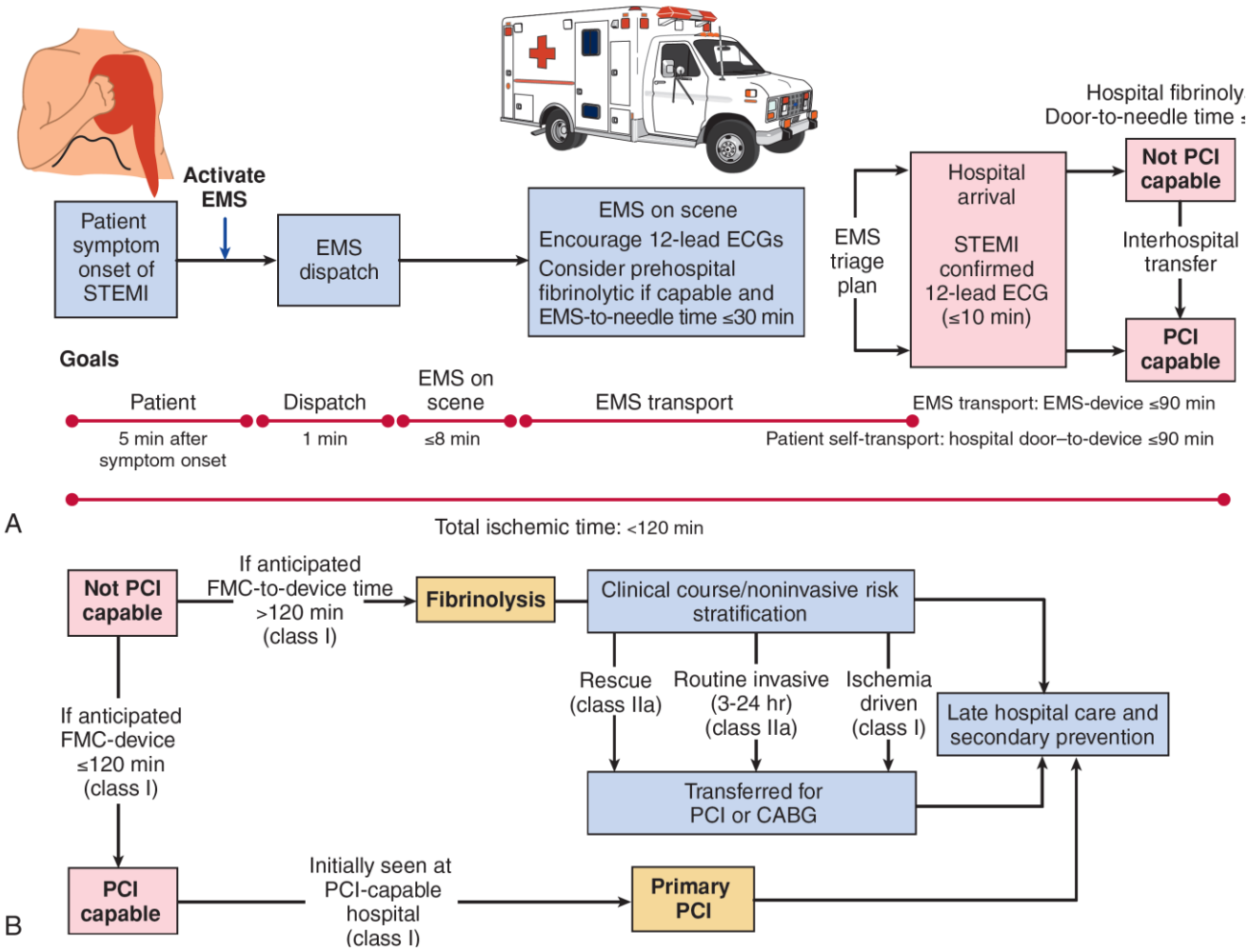

Figure 3: System goals and initial reperfusion treatment of patients with STEMI [3].

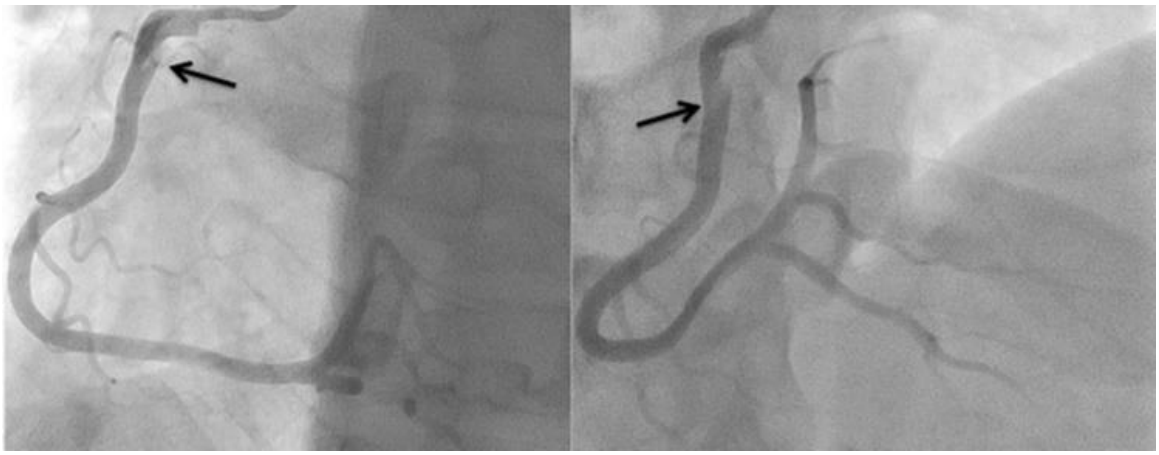

Figure 4: Right coronary artery dissection by angiography [34]

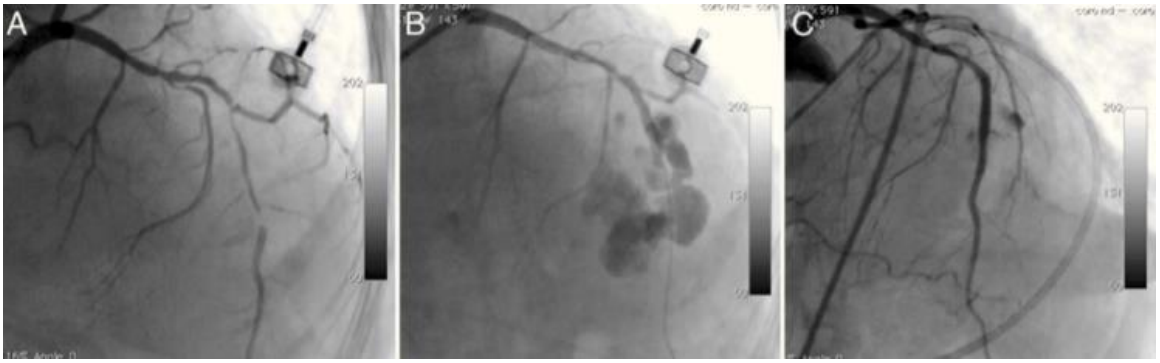

Figure 5: Coronary perforation [35]

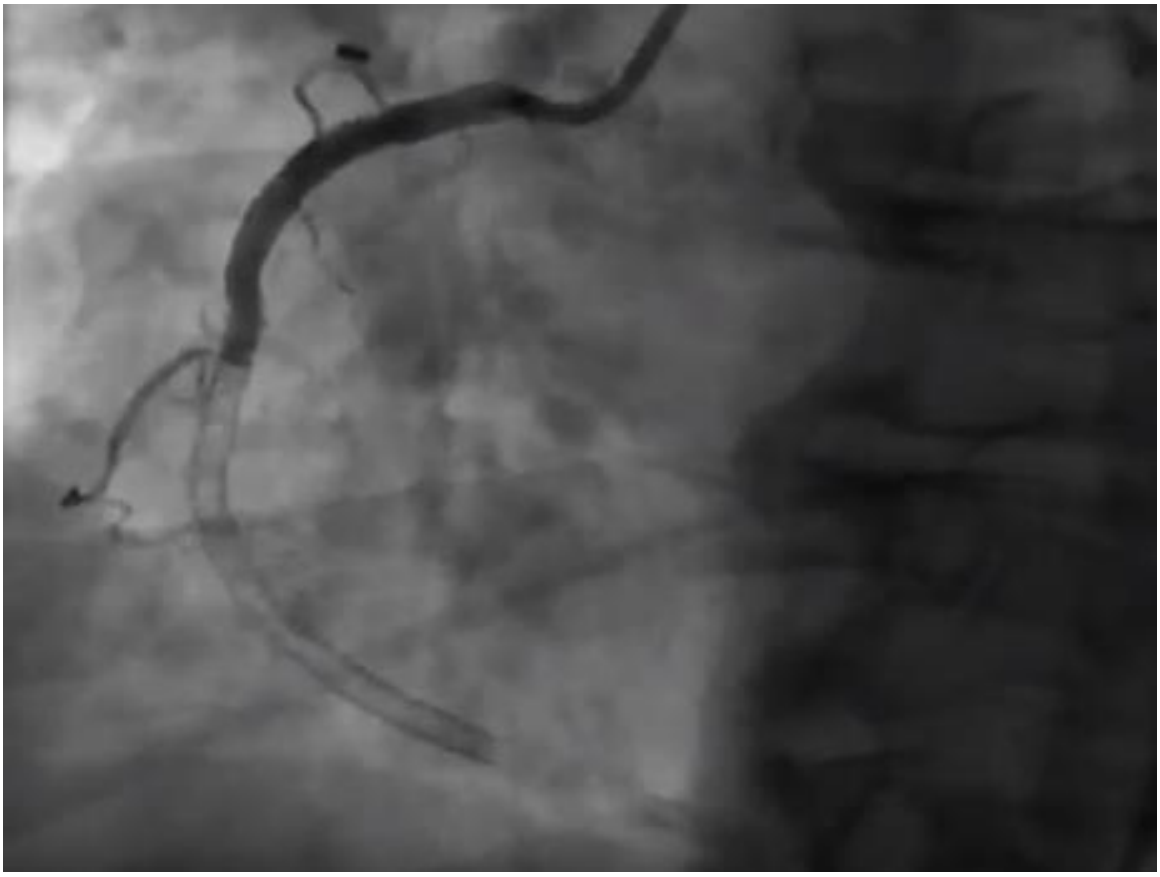

Figure 6: Acute stent thrombosis in right coronary artery [37]

Table 1: Predisposing factors for stent thrombosis [38]

Clinical variables	Anatomic variables	Procedural factors
▪ **Acute MI** ▪ **Clopidogrel non-compliance** ▪ **Clopidogrel bioavailability** ▪ **Diabetes mellitus** ▪ **Renal Failure** ▪ **Congestive heart failure**	▪ Long lesions ▪ Smaller vessels ▪ Multi-vessel disease ▪ Bifurcational lesion	▪ Stent underexpansion ▪ Incomplete wall opposition ▪ Residual inflow and ouflow disease ▪ Margin dissection ▪ Crush technique ▪ Overlapping stent ▪ Polymer materials

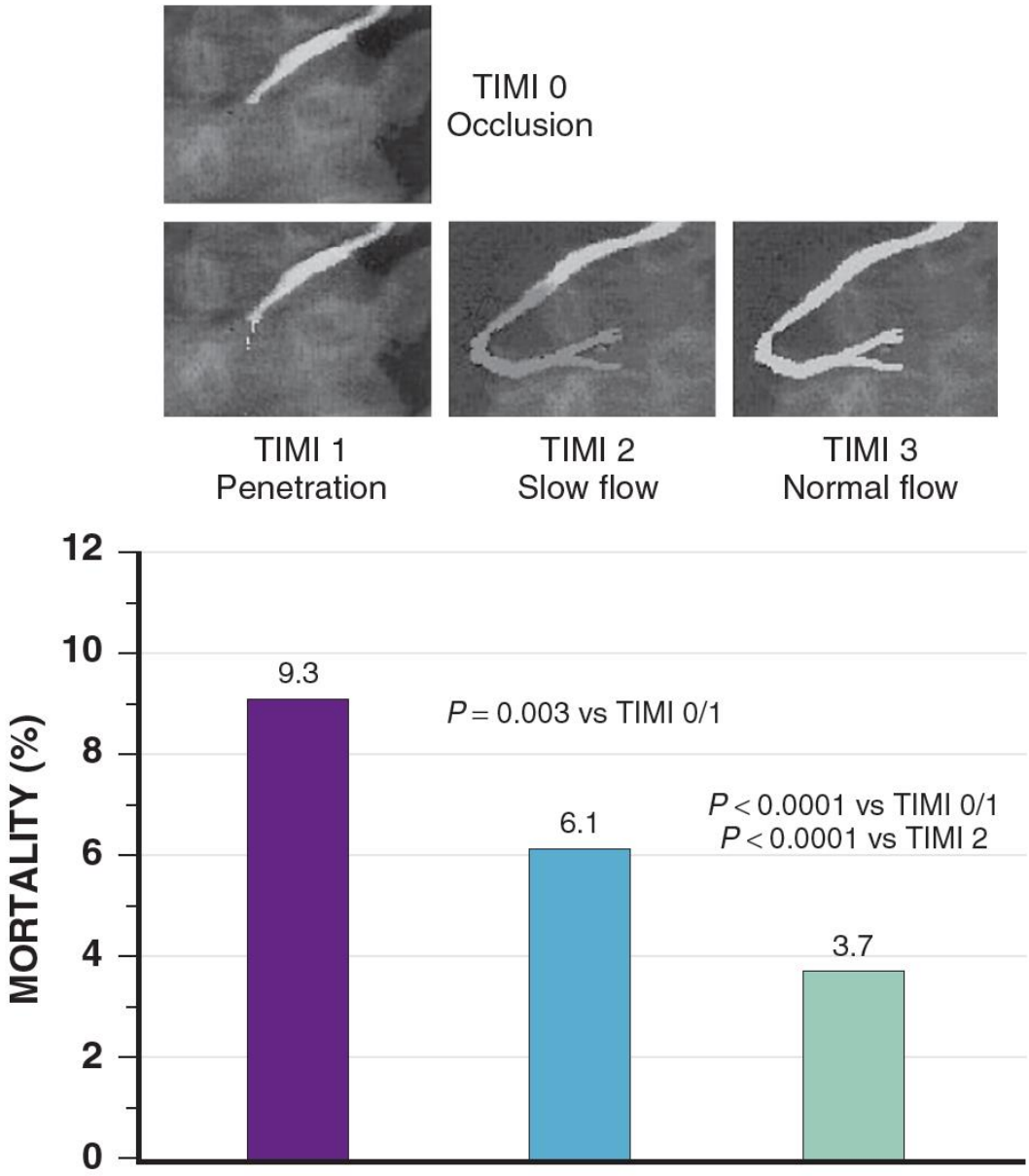

Figure 7: TIMI (Thrombolysis in Myocardial Infarction) trial grading system and correlation of TIMI flow grade and mortality [44]

- Emboli to coronary arteries as caused by infective endocarditis, prosthetic valve embolism and mural thrombus from left atrium or left ventricle.
- Congenital coronary artery anomaly as anomalous origin of left coronary artery from pulmonary artery.
- Myocardial oxygen demand – supply mismatch as caused by aortic stenosis, thyrotoxicosis and anemia.
- Takotsubo cardiomyopathy.

Laboratory findings

Myocardial injury can be detected by the presence of circulating proteins released from damaged myocardial cells. Even though the availability of cardiac markers has enabled clinicians to identify much lower levels of injury, they do not provide information about the cause of the damage [11]. (Figure 2)

- **Cardiac-Specific Troponins**

The preferred biomarker to detect myocardial injury is cardiac troponin, which consists of three subunits that regulate the calcium-mediated contractile process of striated muscle. These subunits include troponin C, troponin I (TnI), and troponin T (TnT) [13].

Following myocyte injury, the initial release of cardiac specific TnT and TnI is from the cytosolic pool, followed subsequently by release from the structural (myofilament-bound) pool [14].

Detection of a rise and fall in cTnT or cTnI in the appropriate clinical setting is now at the center of the new diagnostic criteria for MI [13].

In patients with MI, cTnT and cTnI first begin to rise by approximately 3 hours after the onset of chest pain. Because of continuous release from a degenerating contractile apparatus in necrotic myocytes, elevations in cTnI may

persist for 7 to 10 days after MI; elevations in cTnT may persist for up to 10 to 14 days. The prolonged time course of the elevation in cTnT and cTnI is advantageous for the late diagnosis of MI. Patients with STEMI who undergo successful recanalization of the infarct-related artery have a rapid release of cardiac troponins, which can indicate reperfusion [1].

High-sensitivity cardiac tro¬ponin (hsTn) enables precise measure¬ment of very low concentrations of cardiac-specific troponin. They are more sensitive than previous-generation assays but also have diminished clinical specificity for MI because they detect true myocardial injury in a variety of other clinical settings [15].

• **Creatine Kinase-MB**

Cardiac muscle contains both the MM and MB iso-enzymes of CK. Other tissues can contain small quantities of the MB iso-enzyme of CK. Strenuous exercise, particularly in trained long-distance runners or professional athletes, can cause an elevation in both total CK and CK-MB [1].

Like cardiac-specific troponin, the diagnosis of MI requires a maximal concentration of CK-MB exceeding the 99th percentile of values for sex-specific reference levels on two successive samples in a rise and fall pattern. CK-MB is inaccurate in circumstances involving skeletal muscle injury[13].

• **Other biomarkers**

Other biomarkers may be used to assess the potential causes and complications of MI. C-reactive protein (CRP) rises substantially in the setting of STEMI because of the inflammatory response to myocyte necrosis [16]. B-Natruritic Peptide (BNP) and related peptides reflect the hemodynamic impact of the MI and are associated with prognosis [16]. Other markers as myoglobin, hemoglobin, leucocytic count, erythrocyte sedimentation rate (ESR), and serum lipids exhibit changes in their value after symptoms onset, no clear guidance is available on how to structure specific therapeutic maneuvers in the setting of STEMI in response to these biomarkers [16]. Platelets play a crucial role in the thrombus formation after the rupture of an atherosclerotic plaque. There will be increased release of larger platelets with more dense granules that are highly active metabolically and enzymatically. There is accumulating evidence showing that the MPV may predict CVD, as well as outcomes in patients with CAD. There is also evidence linking MPV and comorbidities (e.g. diabetes mellitus and impaired glycaemic control) that are expected in patients with CAD. The effect on MPV of drugs commonly used to treat CAD has not been clarified, but there is some evidence that they may exert a beneficial effect on the MPV. More specifically, the MPV may predict the effect of antiplatelet drugs (e.g. clopidogrel) [17].

Imaging in STEMI:

If in doubt regarding the possibility of acute evolving myocardial infarction, If locally available, emergency coronary angiography is the modality of choice, as it can be followed immediately by primary PCI if the diagnosis is confirmed [18].

In hospitals or settings in which coronary angiography is not immediately available, provided it does not delay transfer, rapid confirmation of segmental wall-motion abnormalities by two-dimensional echocardiography may assist in making a decision for emergency transfer to a PCI center, since regional wall-motion abnormalities occur within minutes following coronary occlusion, well before necrosis. However, wall-motion abnormalities are not specific to acute myocardial infarction and may be due to other causes such as ischemia, an old infarction or ventricular conduction defects. Two-dimensional echocardiography is of particular value for the diagnosis of other causes of chest pain, such as pericardial effusion, massive pulmonary embolism or dissection of the ascending aorta. The absence of wall-motion abnormalities excludes major myocardial infarction. In the emergency setting, the role of computed tomography (CT) scan should be confined to differential diagnosis of acute aortic dissection or pulmonary embolism [19].

Management of STEMI

•Prehospital management

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. Also providing automated external defibrillators to first responders, placing automated external defibrillators in critical public locations, and greater coordination of the Emergency Medical Service [3]. Major components of the time from the onset of ischemic symptoms to reperfusion include:

- The time for the patient to recognize the problem and seek medical attention.
- Prehospital evaluation, treatment, and transportation.
- 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).
- The time from initiation of treatment to restoration of flow [3].

•Prehospital fibrinolysis

Multiple observational studies and several randomized trials have evaluated the potential benefits of prehospital versus in-hospital fibrinolysis. Earlier treatment generally provides greater benefit, and a meta-analysis of all the available trials demonstrated a 17% reduction in mortality. The CAPTIM (Comparison of Primary Angioplasty and Pre-hospital Fibrinolysis in Acute Myocardial Infarction) trial, for example, reported a trend toward a lower mortality rate in patients with STEMI who received prehospital fibrinolysis compared with patients who received primary PCI, especially if they were treated within 2 hours of the onset of symptoms [4].

General treatment measures

-Acetyl salicylic acid (aspirin)

Aspirin is effective across the entire ACS spectrum and is part of the initial management strategy for patients with suspected STEMI. Because low doses take several days to achieve a full antiplatelet effect, 150 to 300 mg chewable

(non-enteric coated) should be administered at the first opportunity after initial medical contact. To achieve therapeutic blood levels rapidly, the patient should chew the tablet to promote buccal absorption rather than absorption through the gastric mucosa [20].

-P2Y12 inhibitor

Ticagrelor (180 mg loading and 90 mg maintenance bid) is superior to Clopidogrel (600mg loading and 150 mg maintenance dose in the first week in the setting of PCI, the use of Clopidogrel should be restricted to circumstances when Ticagrelor is not available or contraindicated. Contraindication of Ticagrelor includes previous hemorrhagic stroke, patients on oral anticoagulant and patients with moderate to severe liver disease. Also, Ticagrelor cannot be used when fibrinolytic is the reperfusion strategy [3]. In the setting of thrombolytic therapy Clopidogrel 300 mg loading and 75 mg maintenance and in patients above 75 years old loading is 75mg only [21].

-Relief of pain, breathlessness, and anxiety:

Pain is associated with sympathetic activation that causes vasoconstriction and increases the workload of the heart. Titrated intravenous opioids (e.g. morphine) are the analgesics most commonly used in this context [22]. Oxygen (by mask or nasal prongs) should be administered to those who are breathless, hypoxic, or who have heart failure. Non-invasive monitoring of blood oxygen saturation greatly helps when deciding on the need to administer oxygen or ventilatory support [23].

-Nitrates:

Nitrates are mainly venodilators, which reduces cardiac preload and. However, sustained administration causes tolerance and is associated with pro-oxidant effects, endothelial dysfunction and increased sensitivity to vasoconstrictors [24].

Reperfusion

Reperfusion is the cornerstone in the management of STEMI. Late spontaneous reperfusion occurs in some patients, thrombotic occlusion persists in most patients with STEMI. Timely reperfusion of jeopardized myocardium is the most effective way of restoring the balance between myocardial oxygen supply and demand [25]. The efficacy of fibrinolytic agents decreases as coronary thrombi matures over time. Analyses adjusted for baseline risk also demonstrate a statistically significant increase in in-hospital and long-term mortality with progressive delays between the onset of symptoms and PCI. Each 30-minute delay from symptom onset to PCI increases the relative risk (RR) for 1-year mortality by 8% [26]. Prevention of cell death by restoration of blood flow depends on the severity and duration of the preexisting ischemia and on collateral coronary vessels. Even after successful reperfusion and despite the absence of irreversible myocardial damage, a period of post ischemic contractile dysfunction can occur a phenomenon called myocardial stunning [27].

Percutaneous coronary angioplasty (PCI)

Types of PCI: [3]

- Primary PCI: PCI is used as primary reperfusion therapy in patients with STEMI with no use of thrombolytics.
- Pharmacoinvasive strategy: fibrinolysis combined with rescue PCI (in case of failed fibrinolysis) or routine early PCI strategy (in case of successful fibrinolysis.)

For patients who arrive at an experienced primary PCI center, primary PCI should be performed in those with STEMI who present within 12 hours of symptom onset and those with later arrival who have ongoing ischemia, HF, or shock [28].

If the time from first medical contact to PCI is expected to be more than 120 minutes, fibrinolysis is recommended in the absence of

- Significant contraindications to fibrinolysis.
- Shock or acute severe heart failure.
- Late presentation 12 to 24 hours after symptom onset.
- Otherwise, transfer for primary PCI is generally favored if any of these conditions are present, even if the delay to revascularization will be greater than 120 minutes [4].

In primary PCI, drug-eluting stents (DES) reduce the risk of repeated target vessel revascularization, compared with bare-metal stents (BMS). There have been concerns about increased risks of very late stent thrombosis and re-infarction with DES, compared with BMS. However, use of DES has not been associated with an increased risk of death, myocardial infarction or stent thrombosis on long-term follow up [3].

An issue with the routine use of DES in this setting is that it is often difficult to determine reliably the ability of patients to comply with or tolerate the protracted use of dual anti-platelet therapy (DAPT) [29].

Approximately 50% of STEMI patients have significant multivessel disease. In the 2017 ESC guidelines on the management of STEMI routine revascularization of non-culprit vessels should be considered before discharge (Class IIa). Radial approach is now considered superior to femoral approach if done by experienced operator [3].

Successful PCI

Anatomic (or angiographic) success: attainment of a residual diameter stenosis of less than 50%, which is generally associated with at least a 20% improvement in diameter stenosis and relief of ischemia. With the widespread use of coronary stents, the angiographic criterion for success is 20% stenosis or less when stents are used [30].

Procedural success: angiographic success without the occurrence of major complications (death, MI, or CABG) within 30 days of the procedure [30].

Clinical success: procedural success without the need for urgent repeated PCI or surgical revascularization within the first 30 days of the procedure [30].

Complications of PCI

Early outcomes

Mortality

Although mortality after PCI is rare (<1%), it is higher in the setting of STEMI, in cardiogenic shock, and in patients with previously poor LV function in whom STEMI occurs [30].

Myocardial infarction:

In clinical practice, asymptomatic CK-MB elevation (<5 times upper limit of normal) occurs after 3% to 11% of technically successful PCIs and has little apparent clinical consequence. Larger degrees of myonecrosis (CK-MB 5 to 8 times upper limit of normal) are associated with higher 1-year mortality rates and should be considered a periprocedural MI [31].

A consensus definition of periprocedural MI now uses a troponin level elevated more than five times normal when it occurs in conjunction with clinical evidence of MI with symptoms, changes on the electrocardiogram (ECG), angiographic findings, or a new imaging abnormality [32].

Angiographic complications

Coronary dissection: maybe barotrauma induced dissection or caused by guiding catheter. If dissection extends deeper into the media or adventitia begin to compromise the true lumen of the vessel, clinical ischemia may develop. Most intra-procedural dissections can be treated promptly by stenting, significant residual dissections of the treated artery occur in 1.7% of patients. These residual dissections increase the risk for post-procedural MI, need for emergency CABG, and the incidence of stent thrombosis and increase mortality threefold [33]. (Figure 4)

Coronary perforation: develops in 0.2% to 0.5% of patients undergoing PCI and is more common with atheroablative devices and hydrophilic wires than with balloon angioplasty or conventional guidewires. Depending on the rate of flow through the vessel perforation, cardiac tamponade and hemodynamic collapse can occur within minutes, thus requiring immediate recognition and treatment of the perforation. Strategies for controlling coronary perforations include reversal of intraprocedural anticoagulation and prolonged inflation (at least 10 minutes) of an oversized balloon at low pressure at the site of the perforation to encourage sealing of the tear in the vessel. Management strategies for perforations include the use of perfusion balloons, which provide a small amount of distal perfusion, and the use of stents, which may control free perforations, in addition to decompression of pericardial pressure with prompt pericardiocentesis. Approximately one third of patients with PCI-associated coronary artery perforation require emergency cardiac surgery [33]. (Figure 5)

No-reflow is defined as reduced anterograde perfusion in the absence of a flow-limiting stenosis and occurs in up to 2% to 3% of PCI procedures, typically during interventions on degenerated Saphenous vein grafts, during rotational atherectomy, and during acute MI interventions. No-reflow is probably caused by distal embolization of atheromatous and thrombotic debris dislodged by balloon inflation, atherectomy, or stent implantation. Once it occurs, no-reflow can cause severe short- and long-term consequences, including a fivefold increased risk for periprocedural MI and a threefold increased risk for death [36]. Stent thrombosis: With the routine use of a high-pressure stent after dilation and DAPT after stent implantation, the rate of stent thrombosis has declined to approximately 1% within the first year after stenting, although it can be higher in patients with STEMI or after complex PCI. Certain clinical, angiographic, and procedural factors predispose to its development. The timing of stent thrombosis is defined as acute (<24 hours), subacute (24 hours to 30 days), late (30 days to 1 year), and very late (>1 year) [37] (Figure 6) (Table 1)

Vascular access site complications

Occur after 3% to 7% of femoral PCIs and lead to significantly increased length of hospital stay, total cost and mortality [39]. They range from minor access site hematomas to life-threatening retroperitoneal bleeding requiring emergency blood transfusion, to damage to the vasculature necessitating prompt surgical intervention [39]. Major vascular complications of the femoral approach include limb-threatening ischemia (0.1%) and retroperitoneal hemorrhage (0.4%), which are associated with a 2- to 10-fold increased risk for death in the first 30 days after PCI [40]. They are more common in older age, female sex, larger vascular sheath size, low body mass index, renal insufficiency, and degree of anticoagulation during the procedure. The location of the entry point for transfemoral access predicts the risk and type of vascular complication. If the access site is above the level of the inguinal ligament, the risk for retroperitoneal hemorrhage increases substantially. If the access site is distal to the femoral bifurcation, pseudoaneurysms (0.4%) and arteriovenous fistulas (0.2%) may occur [40]. Trans-radial access is associated with a generally lower rate (2%) of vascular complications. A meta-analysis suggested that radial access reduced major bleeding in comparison to femoral access. In the RIVAL (Radial vs Femoral Access for Coronary Intervention) trial, which randomly assigned patients to either femoral or radial access, no significant difference was found in the primary endpoint of major ischemic events or bleeding, but the rate of vascular complications was significantly reduced with the radial approach [39, 40].

Late outcomes

Ischemic events within the first year after PCI result from one of three processes:

- Lumen narrowing requiring repeated target-lesion revascularization occurs in 20% to 30% of patients undergoing balloon angioplasty because of reparative arterial constriction, also known as “negative remodeling.”
- Clinical restenosis after stent implantation is less common (10% to 20%) and attributable to intimal hyperplasia within the stent.
- Clinical recurrence caused by restenosis is least common (3% to 5%) after DES placement because of focal tissue growth within the stent or at its margins [39].

Reperfusion injury

Reperfusion, although beneficial in terms of myocardial salvage, may cause adverse sequelae described by the term reperfusion injury. Several types of reperfusion injury occur in experimental animals [27, 41].

- Lethal reperfusion injury, which refers to reperfusion-induced death of cells that were still viable at restoration of coronary blood flow.
- Vascular reperfusion injury, which is progressive damage to the microvasculature such that there is an expanding

area of no-reflow and loss of coronary vasodilatory reserve.
- Stunned myocardium, in which salvaged myocytes display a prolonged period of contractile dysfunction after restoration of blood flow because of abnormalities in intracellular metabolism, leading to reduced energy production.
- Reperfusion arrhythmias, which refers to bursts of VT (and occasionally VF) that occur within seconds of reperfusion.

Reperfusion arrhythmias

Transient sinus bradycardia occurs in many patients with inferior infarcts at the time of acute reperfusion, often accompanied by some degree of hypotension. This combination of hypotension and bradycardia with a sudden increase in coronary flow may involve activation of the Bezold-Jarisch reflex. Premature ventricular contractions (PVCs) accelerated idioventricular rhythm, and non-sustained VT also usually follow successful reperfusion [3, 4].

Assessment of reperfusion

Most clinicians utilize the TIMI score (Thrombolysis In Myocardial Infarction):
- TIMI 0 flow (no perfusion) refers to the absence of any antegrade flow beyond a coronary occlusion.
- TIMI 1 flow (penetration without perfusion) is faint antegrade coronary flow beyond the occlusion, with incomplete filling of the distal coronary bed.
- TIMI 2 flow (partial reperfusion) is delayed or sluggish antegrade flow with complete filling of the distal territory.
- TIMI 3 is normal flow which fills the distal coronary bed completely [42].

Patients with TIMI 0 or TIMI 1 flow had the highest rate of mortality, TIMI 2 flow associated with an intermediate rate of mortality, and the lowest rate of mortality was observed in patients with TIMI 3 flow, [43]. (Figure 7)

Myocardial blush grade (MBG) is defined as the amount of contrast opacification of the myocardium supplied by the infarct-related artery (IRA) in relation to its supplying epicardial density as seen by the operator.

a) MBG 0: there is an absence of contrast opacification of the affected myocardium.
b) MBG 1: there is a minimal opacification or persistent staining seen.
c) MBG 2: a reduced myocardial blush in the infarct area when compared to the unaffected territories
d) MBG3: normal contrast density, comparable to that obtained with angiography of a contralateral or ipsilateral artery [45].

Contemporary guidelines and controversies

Guidelines on Myocardial Revascularization gives a Class I recommendation for CABG in patients with ischemic cardiomyopathy and ejection fraction 35% or less, particularly those with disease of the left anterior descending (LAD) artery, left main, or multiple coronary artery territories. PCI may only be considered (class IIb recommendation) if anatomy is suitable, in the presence of viable myocardium, and where surgery is not indicated [46].

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