

Management of Acute Myocardial Infarction with ST Segment Elevation. Narrative Review

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SUMMARY

Background: ST-segment elevation acute myocardial infarction (STEMI) is a cardiovascular emergency with high potential for morbidity and mortality, and reperfusion therapy is a priority.

Objective: To describe the clinical, electrocardiographic, and biochemical diagnostic criteria for ST-segment elevation myocardial infarction and to specify and delimit the initial management of STEMI with an updated scientific basis.

Inclusion Criteria: scientific articles written in the adult population with ST-segment elevation myocardial infarction, in Spanish, English, and Portuguese, between 2018 and 2023.

Methodology: A systematic search was conducted in PUDMED, LILACS, and COCHRANE, identifying 1,071 articles, of which 113 met the inclusion criteria.

Results: Primary percutaneous coronary intervention is the ideal method and reperfusion treatment of ST-segment elevation myocardial infarction of choice. It is recommended to perform it within a period of no more than 120 minutes, since this is the time when its greatest utility is achieved compared to thrombolysis.

Conclusion: Coronary reperfusion is the main objective of the treatment of the patient with STEMI and pharmacoinvasive therapy is ideal in an environment without rapid access to the haemodynamics or interventional cardiology centre in order to achieve prompt coronary reperfusion of the patient with STEMI and thus also improve their prognosis and quality of life.

Keywords: Acute Myocardial Infarction; Fibrinolysis; Pharmacoinvasive Strategy

Abbreviations: AMI: Acute Myocardial Infarction; PRISMA: Preferred Reporting Items for Systematic Reviews and Meta-Analyses; STEMI: ST-segment Elevation Myocardial Infarction; CVRF: Cardiovascular Risk Factors; CVD: Cardiovascular Disease; SAH: Systemic Arterial Hypertension; SBP: Systolic Blood Pressure; CVE: Cerebrovascular Events; HF: Heart Failure; CKD: Chronic Kidney Disease; LGI: leukoglycemic Index; RV: Right Ventricular; GOT: Glutamic Oxaloacetic Transaminase; CPK: Creatine Phosphokinase; LDH: Lactate Dehydrogenase

Introduction

Acute myocardial infarction (AMI) is the most common cause of death worldwide [1] affecting more than 7 million people worldwide annually [2]. The initial management of STEMI should be known and established by all health personnel, taking into account the premise that time is myocardium, time is tissue, time is life; early reperfusion, that is, the restoration of blood flow to the occluded artery, is the most effective way to preserve the viability of the ischemic myocar-

dium and limit the size of the infarct [3]. The European guidelines for STEMI establish reperfusion treatment <12 hours. PCI is preferred over fibrinolytic therapy and, since there is no haemodynamics room available, pharmacoinvasive therapy or strategy is recommended (immediate PCI after fibrinolytic therapy) [4]. The effectiveness of AMI management depends on the speed and accuracy of diagnosis and pharmacology [5]. The ultimate goal of STEMI treatment is to save a life, maintain a good quality of life and give the patient back a life as productive as before the AMI [6]. The objectives of this nar-

rative review are: To describe the clinical, electrocardiographic and biochemical diagnostic criteria for STEMI and to specify and delimit the initial management of STEMI with updated scientific foundations. The questions formulated to be answered in this review are: What are the clinical, electrocardiographic and biochemical diagnostic criteria for STEMI? What are the coronary reperfusion therapies that are carried out for the treatment of patients with STEMI?

Methodology

The structure of the narrative review was assessed based on the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) checklist modified for narrative analysis, as well as the SANRA (Scale for the quality assessment of narrative review articles) scale [7,8]. The inclusion criteria were: scientific articles, randomized clinical trials, clinical practice guidelines, narrative reviews, system-

atic reviews with meta-analysis, all of them carried out in the adult population (over 18 years of age) with a diagnosis of STEMI between 2018 and 2023, in Spanish, English and Portuguese. The exclusion criteria were: articles with incomplete clinical trials, in the recruitment phase or suspended, articles with only a summary or that included the pediatric population. To obtain information and select articles to carry out this narrative review, the following databases were chosen: PUBMED, LILACS and COCHRANE. A systematic search was performed and of the 1071 articles identified, 322 were eliminated due to duplications and 352 due to irrelevance, leaving 397 articles for eligibility. After analysis, 347 articles were excluded, leaving a total sample of 50. The search is presented in the following flowchart (Figure 1). The MeSH search terms used were: "ST-segment elevation myocardial infarction" (STEMI), "Treatment of ST-segment elevation acute myocardial infarction" and fibrinolysis in acute myocardial infarction.

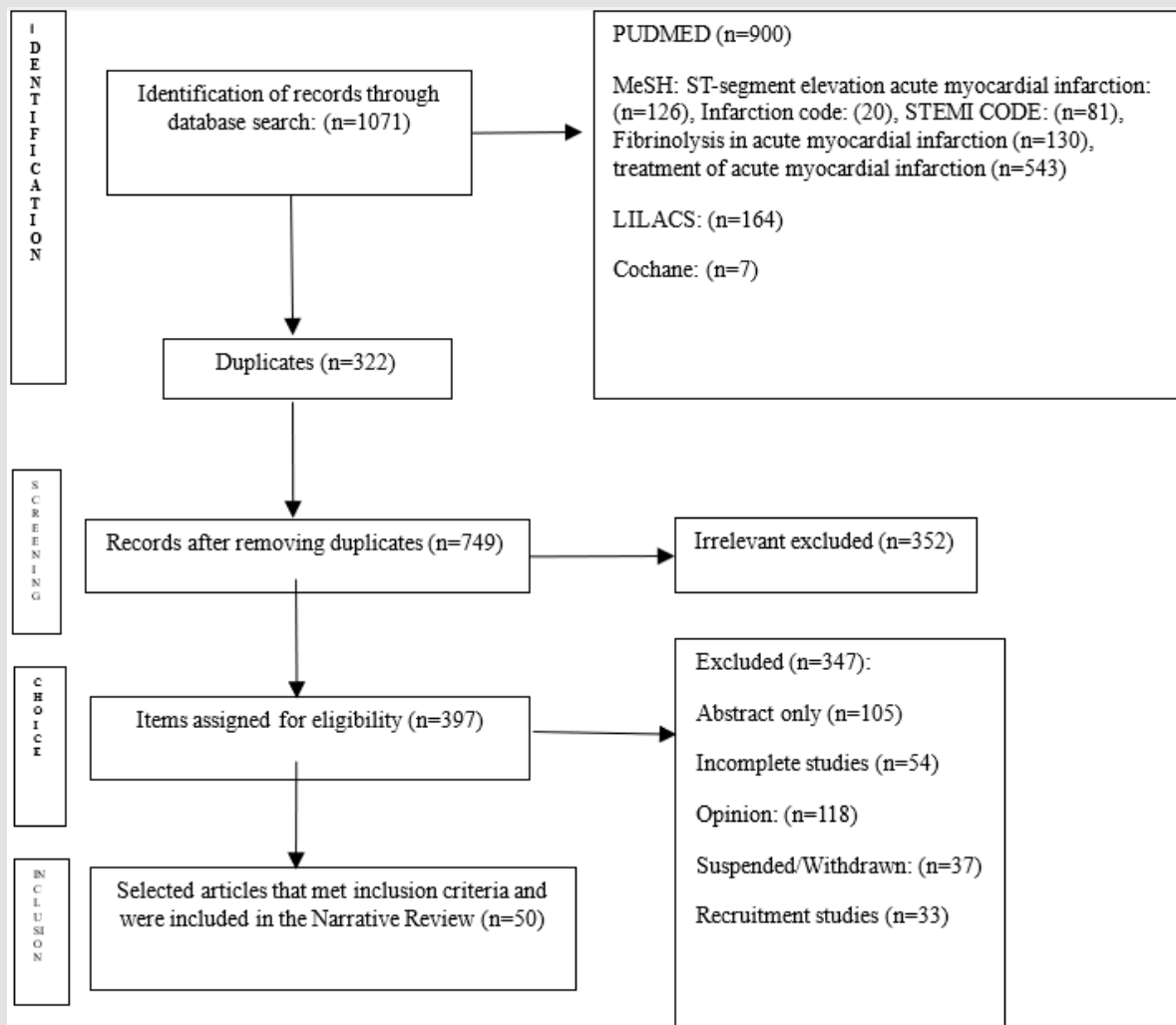


Figure 1: Flowchart of systematic search and selection of articles.

Epidemiology

It is estimated that by 2030, approximately 23.6 million people will die from AMI, and it is predicted to remain the leading cause of death globally. In North America, it has been reported that nearly 50% of all men and 33% of women over 60 years of age will develop some manifestation of cardiovascular disease (CVD) [9,10] patients ≥ 75 years of age experience 1/3 of AMI, more than 50% with fatal outcomes, [11] Among the cardiovascular risk factors (CVRF) according to the case-control study INTERHEART 90% are: smoking, dyslipidemia, systemic arterial hypertension (SAH), diabetes, morbid obesity, sedentary lifestyle, low daily consumption of fruits and vegetables, alcoholism and psychosocial index [12,13] in addition to the 2021 National Health and Nutrition Survey on COVID19 (ENSA-NUT CONTINUA 2021) It reports that a 2 mmHg increase in systolic blood pressure (SBP) is associated with a 7% increase in the risk of mortality from ischemic heart disease and a 10% increase in the risk of mortality from cerebrovascular events (CVE), which is why SAH is considered an important risk factor for AMI, ischemic and hemorrhagic stroke, heart failure (HF), chronic kidney disease (CKD), cognitive impairment and premature death [14].

Forecast

Cardiogenic shock, blood glucose values above 15 mmol/L, biventricular infarction, late arrival at the primary care site, age over 70 years, blood glucose values between 7.3 and 15 mmol/L, and creatinine values greater than 200 $\mu\text{mol/L}$ were risk factors for in-hospital mortality in NSTEMI. Cardiogenic shock, classified as Killip Class IV, is the most common cause of in-hospital mortality, with a mortality rate as high as 40% to 70% [15]. Hyperglycemia on admission is common in patients with AMI and is a strong predictor of mortality and in-hospital complications. Hyperglycemia is the best predictor of short-term prognosis of large infarcts, while elevated glycated hemoglobin (HbA1c) is associated with long-term clinical effects due to an increased baseline risk [16]. Pedraza Rodriguez et al. established the cut-off point at 7.8%, above which glycated hemoglobin was a predictor of mortality and major cardiac complications [17]. The leukoglycemic index (LGI) in the course of AMI may be associated with higher in-hospital mortality, prognostic stratification of SICA, determination of the risk of complications and mortality in STEMI. To calculate the LGI, the following formula was used: $\text{LGI} = (\text{glycemia [mg/dl]} \times \text{leukocytes [106 /l]}) / 1,000$ [18]. Cuesta Mero et al., in Ecuador, conducted a cohort, observational and cross-sectional study with 205 patients with AMI where it was concluded with an OR of 7.89 that an $\text{ILG} > 656.8$ represents a 7.89 times risk of developing complications in the population studied [5,19].

Pathophysiology

The pathophysiology of SICA involves myocardial ischemia/necrosis caused by rapid narrowing/obstruction of the coronary artery as a consequence of the disruption of the atheromatous plaque,

thrombogenesis or by the presence of coronary vasospasm [15], resulting in an imbalance between oxygen supply and demand. After the onset of ischemia, the first structural changes within the first 10-15 minutes are the decrease in cellular glycogen and relaxed myofibrils and the alteration of the sarcolemma, with myocyte necrosis occurring hours later [20] The usual initiation mechanism for AMI is rupture or erosion of a vulnerable coronary plaque [21] (if its fibrous cap is thinned, it is prone to rupture). The vulnerable plaque is described as an atherosclerotic lesion characterized by a large lipid-rich or necrotic core separated from the vessel lumen by a thin fibrous cap (coronary fibroatheroma). Atherosclerotic progression subsequently occurs through expansion of the lipid core and accumulation of macrophages at the plaque edges, leading to plaque rupture. After plaque rupture, thrombogenic material residing within the plaque is exposed to blood and circulating coagulation factors, resulting in the formation of a thrombus in the ruptured plaque. If the thrombus completely occludes the coronary vessel, aerobic metabolism in the affected myocardium stops, resulting in rapid ATP depletion as well as accumulation of metabolites such as lactate.

These metabolic effects, in turn, lead to electrolyte changes including a shift of K^+ into the extracellular space and a reduction in action potential duration and amplitude. Within seconds, these processes lead to a reduction in myocardial contractility. Although these effects are completely reversible if blood flow is restored rapidly, animal models have shown that a time interval of 20 to 30 minutes of sustained ischemia is sufficient to cause irreversible damage to cardiomyocytes. Necrosis occurs first in the endocardium, which is more distal to the blood supply, before it progresses to the subepicardial layers [3].

Diagnosis

Clinical Picture

The symptoms of AMI are: chest pain radiating to the upper limb, lower jaw or epigastrium during exercise or at rest, diffuse; not localized or positional, not affected by movement of the region [20,21] of sudden and progressive onset, lasting more than 20 minutes, (may be absent in patients with diabetes, advanced age and in women) [22] or an ischemic equivalent at rest or with minimal effort such as sudden onset dyspnea [23], fatigue [20] or syncope [23] or atypical symptoms such as pain in the neck, posterior chest, lower jaw, as well as nausea, asthenia and adynamia, which are more frequent in women than in men [24], in addition to dyspnea, chest discomfort, stabbing pain in the chest, pleuritic pain, syncope, feeling of anxiety or imminent death palpitations or even asymptomatic [20].

Electrocardiogram (ECG)

The ECG, which is inexpensive and widely available, is essential in the evaluation of AMI patients for diagnostic confirmation and for prognostic stratification [24]. A 12-lead ECG is essential and must be

performed within the first 10 minutes of the first contact (PCM) between the patient and the physician [15]. It is important to remember that ST segment elevation has 45-60% of S to diagnose AMI and about 50% of patients have normal or non-diagnostic ECG.¹¹ In addition, it is recommended that in patients with suspected posterior AMI, posterior chest wall leads (V7, V8, V9) [15] should be used and if right ventricular (RV) AMI is suspected, the right precordial leads (V3R and V4R) should be used (ST segment elevation in the precordial leads is short-lived, disappearing within 10 hours after the onset of symptoms in 50% of inferior AMI and RV infarction) [15], calling this recording "thoracic circle" [24].

Cardiac Biomarkers

Biochemical markers of myocardial injury should be measured in all patients with suspected AFS [24]. However, they should not delay reperfusion therapy [3,25] They should be measured on admission and repeated 6-9 hours later (9-12 hours if there is strong clinical suspicion; 3-6 hours with ultrasensitive troponin) [11] These include creatine phosphokinase (CPK), creatine phosphokinase fraction MB (CPK-MB), myoglobin, glutamic oxaloacetic transaminase (GOT), lactate dehydrogenase (LDH), cardiac troponin T, cardiac troponin I, and myosin light chains [15]. Troponins are the biochemical markers of choice [11].

1. Creatine phosphokinase (CPK): The most common traditional marker of myocardial necrosis, widely used for diagnosis and prognostic prediction of AMI. Myocardial cell death leads to the release of CPK into the plasma, existing in different isoenzymes, CPK-MB has its highest activity in cardiac muscle³ with 97% sensitivity (S) and 90% specificity (E) for the diagnosis of AMI, both are evaluated in STEMI ¹¹ When skeletal muscle is damaged by blows, direct currents, etc., CPK and CK-MB increase; however, skeletal muscle damage can be differentiated from AMI since the proportion of CK-MB does not exceed 5% [15].

2. Cardiac troponins I (cTnI) and T (cTnT): cTnI and cTnT are the preferred biomarkers for the assessment of myocardial injury [4], they are specific for myocardial injury, early diagnosis of AMI and assessment of prognosis, infarct size, reperfusion injury and remodeling; [3] detectable in 2-3 hours with a peak at 24-28 hours [2], compared to CPK-MB which is less sensitive and less specific [20]. The most widely used biomarker in the diagnosis and prognosis of SICA is high-sensitivity troponin⁴, that is, assays available for the rapid detection (qualitative and quantitative) of troponins T in very low quantities as they allow earlier detection of myocardial damage, presenting positive results in the first 60-120 minutes from the onset of pain [26]. Increases in ultrasensitive troponins are very sensitive to the evolution of the infarction, but not very specific [11].

Code Heart Attack

The Infarction Code is the international name of an efficient and effective medical care strategy for patients with AMI [27] and the

first protocol developed in Mexican institutional medicine; focused on emergency services, which complements the IMSS regulations, its objective is to guarantee the diagnosis and treatment of the patient who requires emergency care for AMI, so that they receive reperfusion treatment with primary angioplasty in the first 90 minutes, or fibrinolytic therapy in the first 30 minutes after admission to the IMSS Emergency Services [21]. Every 30-minute delay in early coronary intervention from the onset of AMI symptoms increases the potential for mortality by 8% in the following year [28,29].

Treatment

Initial medical care measures for the patient with STEMI include: [1]

1. The health personnel assigned to triage assigns the red code or Infarction Code to the patient with chest pain, dyspnea or loss of consciousness for immediate attention [21] If a patient is suspected of having AMI, while the anamnesis is being carried out, the 12-lead ECG should be performed and interpreted optimally (a chest circle) within the first 10 minutes from the PCM and the time of its performance should be noted.¹ Activate Infarction Code whenever a patient with symptoms and ECG compatible with STEMI or in a patient who presented cardiorespiratory arrest (CPA) with return of spontaneous circulation (ROSC) and suspicion in the ECG [1].
2. ABCDE (Airway, Breathing, Circulation, Disability, and Exposure) and continuous electrocardiographic monitoring with a monitor-defibrillator or a semi-automated external defibrillator (AED) [1].
3. General measures: It is important to monitor and control vital signs¹ Vital signs should be taken every 1 hour (first 6 hours); every 2 hours (rest of the first 24 hours) and then every 3-4 hours, in uncomplicated cases. Perform continuous electrocardiographic monitoring, collect medical history, record 12-lead ECG and perform laboratory tests within 10 minutes of arrival at the hospital [15]. Semi-Fowler position, bed rest [27] Place a peripheral intravenous line (18-20 G) in the elbow flexure of the left upper limb, remove dentures and metal objects. Take blood samples which include cardiac biomarkers (cTn, cTi, CPK, CPKMB), Complete blood count, Coagulation times, Blood chemistry, Serum electrolytes, Lipid profile and General urinalysis [21] At the same time, the following procedures should be avoided: Arterial punctures, Probes, Administering food or liquids orally (except if necessary for taking medication) [1].
4. Oxygen: Only if saturation is $\leq 90\%$, since hyperoxia can be harmful due to its vasoconstrictor effect¹, increasing systemic vascular resistance (SVR) and blood pressure (BP) and reducing cardiac output (CO) [11,30,31].
5. Analgesic treatment and statins: Persistent chest pain can

increase myocardial oxygen consumption, expand the infarct area and induce arrhythmia, so immediate analgesia or sedation should be provided [32] Regarding statins: Atorvastatin 80 mg PO DU loading dose⁶ Statins have pleiotropic effects, reduce low-density lipoproteins (LDL) and anti-inflammatory effects for plaque stabilization [3].

6. Antiplatelet therapy: Aspirin is the only anti-inflammatory drug routinely indicated for all patients with suspected AMI, except in cases of contraindication (allergy or intolerance to medications, active bleeding, hemophilia and active peptic ulcer)¹¹ Aspirin is recommended at an initial dose of 150-300 mg orally (PO) followed by a maintenance dose of 75-100 mg/day PO [6,24] and clopidogrel is recommended for AMI within the first 12 to 24 hours; this contributes to reducing mortality, reinfarction or stroke.¹⁴ Clopidogrel is recommended with a loading dose of 300 mg PO, followed by a maintenance dose of 75 mg/day PO², [6,24].

7. Anticoagulant Therapy: Its objective is to inhibit the formation or activity of thrombin, which plays an important role in the pathophysiology of STEMI and during primary PCI, therapy includes enoxaparin administered at doses < 75 years: Bolus 30 mg IV followed by 1 mg/kg subcutaneously (SC) 15 minutes later every 12 hours until revascularization or hospital discharge (maximum 8 days) and in ≥ 75 years: do not administer the bolus and start at 0.75 mg/kg SC every 12 hours [2,11] or unfractionated heparin (UFH) with an initial bolus.³² 60 IU/kg, IV bolus with a maximum of 4,000 IU, followed by an infusion of 12 IU/kg/h with a maximum of 1,000 IU for 48 hrs. Taking coagulation time the aPTT: 50-70 seconds or 1.5 to 2.0 times, control monitoring should be at 3, 6, 12 and 24 hours [24] Unless there is a clear indication to continue anticoagulant therapy (such as atrial fibrillation (AF), mechanical prosthetic valve or intraventricular thrombus, among others) it is not indicated after PCI [3].

8. Sublingual nitrates: isosorbide dinitrate 5 mg, one dose every 5 minutes in case of persistent pain (maximum 3 doses) [6].

9. Beta blockers: They are contraindicated in hypersensitivity to the drug, HR less than 60 bpm, arterial hypotension, second and third degree atrioventricular block (AVB). In hemodynamically stable patients, they should be started with metoprolol tartrate 25 to 50 mg IV every 6-12 hours, then in the following 2-3 days dose every 12 hours day or every 24 hours in the case of metoprolol succinate or Carvedilol 6.25 mg PO every 12 hours up to 25 mg [33].

Every patient with STEMI with or without reperfusion therapy should be admitted to a Cardiovascular Intensive Care Unit (ICU) for surveillance, monitoring and establishment of a therapeutic plan, in order to prevent, identify and treat acute complications and, therefore, increase survival [24].

Pharmacoinvasive, Pharmacological, Mechanical and Adjunct Therapy for STEMI

Reperfusion is the most important measure in the treatment of STEMI. The goal of reperfusion therapy is to restore coronary flow to the artery responsible for the infarction and ensure vessel patency. There are two types of therapy that can be used in STEMI reperfusion: pharmacological reperfusion with a fibrinolytic (a drug that lyses the coronary thrombus) and PCI with primary percutaneous transluminal coronary angioplasty (PPTA), which is the default method to define coronary anatomy and characterize the severity of coronary artery stenosis [34] or stent placement, which have been shown to decrease target vessel revascularization and subsequent AMI compared to balloon angioplasty alone (POBA) [2]. Angioplasty allows the coronary anatomy to be known and directly reach and ensure vessel flow [6]. Korean guidelines do not recommend primary PCI in asymptomatic STEMI patients who present >48 hours after ischemic symptoms and recommend fibrinolytic therapy in patients with STEMI and ischemic symptoms of <12 hours duration when primary PCI cannot be performed within 120 minutes [31]. Pharmacoinvasive strategy (PIS) refers to urgent fibrinolysis followed by early angioplasty in the first 2-24 hours to ensure vessel patency [6,31,35] Reperfusion therapy is indicated (PPACT or pharmacoinvasive strategy) within the first 12 hours from the onset of STEMI symptoms, with better outcomes the sooner it is started (especially within the first 3 hours) [6] However, the magnitude of the benefit in mortality and heart muscle salvage is greater the earlier treatment is started. This is why reducing delays until the start of therapy is vital to improve the prognosis of patients with STEMI [6].

EFI might be feasible in countries and regions where access to PCI is limited for geographic, cultural, or social reasons, such as Mexico [36]. Or in India, where almost 60% of the population lives in rural areas lacking hospitals with PCI, fibrinolytic therapy has been shown to prevent 30 premature deaths per 1000 patients treated within 6 h of symptom onset [30]. Indian guidelines also recommend fibrinolytic therapy within the first 12 h of symptom onset, if primary PCI cannot be performed within 90 min of being able to deliver fibrinolysis and within 120 min of PCM without contraindications [37]. Brazilian guidelines recommend administration of Tenecteplase (TNK-tPA) within 3 h of onset in patients diagnosed with STEMI followed by PCI within 6 to 24 h, if primary PCI cannot be performed within 60 min of onset. In the Saudi Arabian guidelines, the term facilitated PCI is preferred over EFI to address the use of fibrinolytic agents or glycoprotein 2b/3a inhibitors administered before arrival for cardiac catheterization in patients undergoing PPCI [38] The different pharmacological treatment regimens for specific fibrinolytic therapy are: [29]

a. to. - Alteplase: [24]

- Maximum dose (max) 100 mg. 15 mg IV bolus, followed by 0.75 mg/kg in 30 minutes (max 50 mg) followed by 0.5 mg/kg in

60 minutes (max 35 mg) [24].

b. Tenecteplase: [24]

- Bolus in 5 or 10 seconds calculated based on weight: [29]
- a. <60 Kg: 30 mg - 6 ml
- b. 61 to 70 Kg: 35 mg - 7 ml
- c. 71 to 80 Kg: 40 mg - 8 ml
- d. 81 to 90 Kg: 45 mg - 9 ml
- e. > 91 Kg: 50 mg -10 ml.29
- In patients > 75 years reduce dose to half the dose [11,24].

Primary PCI is the preferred method and treatment of choice for reperfusion of STEMI [24,27] it is recommended to perform it in a period of no more than 120 minutes since it is in this time when its greatest utility is achieved compared to thrombolysis [24]. Otherwise it is possible to start fibrinolysis and EFI [27] Although percutaneous coronary angioplasty is recommended in patients with STEMI 12-24 hours after the onset of symptoms to reduce mortality at 12 months [2,24,39]. The advantages of primary PCI over fibrinolysis include a lower rate of early death, reinfarction and intracranial hemorrhage [27]. For asymptomatic patients, primary PCI is not indicated after 48 hours of the onset of symptoms [24]. Complications in PCI are: hematoma at the puncture site, transient bradycardia compared to contrast injection, cardiac perforation, sudden closure of a coronary artery during coronary angioplasty, ARI, mortality (<1%) and stroke (0.07%) and transient ischemic attack (1%), the latter two increase their risk 17 times within the first 48 hours of the procedure [40].

Conclusion

STEMI requires prompt identification and timely management, preferably PCI, however, pharmacoinvasive therapy offers an opportunity in areas without rapid access to a catheterization laboratory.

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Conflicts of Interest

The authors declare that they have no conflicts of interest.

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