Overview of Acute Myocardial Infarction: Pathophysiology; Diagnosis and Management

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ABSTRACT

Myocardial infarction (MI) was considered a major cause of death and disability worldwide. MI may be the first manifestation of coronary artery disease (CAD) or it may occur, repeatedly, in patients with established disease.In the developed world, roughly 10% of persons who have had a STEMI will die, with the number of MI cases in the US hovering around a million per year.Acute myocardial infarction is often associated with dynamic changes in ECG waveform and serial ECG acquisition can provide critical information, particularly if the ECG at initial presentation is non-diagnostic. After initial evaluation, coronary angiography may be used in patients with evidence of ongoing ischemia (ECG findings or symptoms), hemodynamic instability, recurrent ventricular tachyarrhythmias, and other abnormalities that suggest recurrence of ischemic events.Initial therapy for acute MI is directed toward restoration of perfusion. This may be accomplished through medical or mechanical means, such as percutaneous coronary intervention (PCI), or coronary artery bypass graft (CABG) surgery. The aim of the present study was to review the pathophysiology; diagnosis and managementof acute myocardial infarction.

Keywords: Myocardial infarction; coronary artery disease; Diagnosis; Management

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INTRODUCTION

Myocardial infarction (MI) describes the tissue death (infarction) of the heart muscle (myocardium). It is a subtype of acute coronary syndrome, which is defined as a sudden or brief shift in symptoms associated with cardiac blood flow. A blood test for biomarkers can detect cell death, which distinguishes myocardial infarction from other acute coronary syndromes causes such unstable angina (the cardiac protein troponin or the cardiac enzyme CK-MB).When there is evidence of MI, it may be classified as an ST elevation myocardial infarction (STEMI) or Non-ST elevation myocardial infarction (NSTEMI) based on the results of an ECG**(1).**

Worldwide, about 15.9 million acute myocardial infarctions occurred in 2015. More than 3 million people had an ST elevation AMI and more than 4 million had an NSTEMI. STEMIs occur about twice as often in men as women **(2).**

AMI was one of the top five most expensive conditions during inpatient hospitalizations in the US **(3).** Rates of death from ischemic heart disease (IHD) have slowed or declined in most high-income countries, although cardiovascular disease still accounted for one in three of all deaths in the USA in 2008. For example, rates of death from cardiovascular disease have decreased almost a third between 2001 and 2011 in the United States **(4).**

Pathological Characteristics of acute myocardial Infarction:

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Acute myocardial infarction is defined in pathology as myocardial cell death due to prolonged ischemia. After the onset of myocardial ischemia, histological cell death is not immediate, but takes a definite period of time to develop, as little as 20 min, or less in some animal models **(5)**.

It takes several hours before myocardial necrosis can be identified by macroscopic or microscopic post-mortem examination. Complete necrosis of myocardial cells at risk requires at least 2-4 h, or longer, depending on the presence of collateral circulation to the ischemic zone, persistent or intermittent coronary arterial occlusion, the sensitivity of the myocytes to ischemia, pre-conditioning, and individual demand for oxygen and nutrients. The entire process leading to a healed infarction usually takes at least 5-6 weeks. Reperfusion may alter the macroscopic and microscopic appearance**(6)**.

Pathophysiology of acute myocardial infarction:

Myocardial infarction is defined as myocardial necrosis in a clinical setting consistent with myocardial ischemia These conditions can be satisfied by a rise of cardiac biomarkers (preferably cardiac troponin (cTn)) above the 99th percentile of the upper reference limit (URL) plus at least one of the following: symptoms of acute myocardial ischemia; ECG changes indicative of new ischemia (significant ST/T changes or left bundle branch block);development of pathological Q waves;imaging evidence of new loss of viable myocardium or new regional wall motion abnormality and angiography or autopsy evidence of intracoronary thrombus.Slightly different criteria are used to diagnose MI during and after percutaneous coronary intervention or coronary artery bypass grafting, and as the cause of sudden death **(1).**

MI can be classified into 5 types based on pathological, clinical, and prognostic differences, along with different treatment strategies: Type (1) Spontaneous MI caused by ischemia due to a primary coronary event (eg, plaque rupture, erosion); Type (2) Ischemia due to increased oxygen demand (eg, hypertension), or decreased supply (eg, coronary artery spasm or embolism, arrhythmia, hypotension); Type (3) Related to sudden unexpected cardiac death; Type (4a) Associated with percutaneous coronary intervention (signs and symptoms of myocardial infarction with cT values > 5 x 99th percentile URL); Type (4b) Associated with documented stent thrombosis; and Type (5) Associated with coronary artery bypass grafting (signs and symptoms of myocardial infarction with cT values >10 x 99th percentile URL)**(7)**.

Infarctions locations:

MI affects predominantly the left ventricle (LV), but damage may extend into the right Ventricle (RV) or the atria. RV infarction usually results from obstruction of the right coronary or a dominant left circumflex artery; it is characterized by high RV filling pressure, often with severe tricuspid regurgitation and reduced cardiac output. An infero-posterior infarction causes some degree of RV dysfunction in about half of patients and causes hemodynamic abnormality in 10 to 15%. RV dysfunction should be considered in any patient who has infero-posterior infarction and elevated jugular venous pressure with hypotension or shock. RV infarction complicating LV infarction significantly increases mortality risk. Anterior infarctions tend to be larger and result in a worse prognosis than infero-posterior infarction. They are

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usually due to left coronary artery obstruction, especially in the anterior descending artery; infero-posterior infarctions reflect right coronary or dominant left circumflex artery obstruction **(1).**

Transmural infarctions involve the whole thickness of myocardium from epicardium to endocardium and are usually characterized by abnormal Q waves on ECG.Nontransmural or subendocardial infarctions do not extend through the ventricular wall and cause only ST-segment and T-wave (ST-T) abnormalities. Subendocardial infarctions usually involve the inner one third of myocardium, where wall tension is highest and myocardial blood flow is most vulnerable to circulatory changes. These infarctions may follow prolonged hypotension. Because the transmural depth of necrosis cannot be precisely determined clinically, infarctions are usually classified as STEMI or NSTEMI by the presence or absence of ST-segment elevation or Q waves on the ECG **(1).**

Diagnosis of acute myocardial infarction:

- **Clinical Features of Myocardial Ischaemia and Infarction**

Myocardial ischemia is a clinical setting can usually be identified from the patient's history and from the ECG. Possible ischemic symptoms include various combinations of chest, upper extremity, mandibular or epigastric discomfort (with exertion or at rest) or an ischemic equivalent such as dyspnoea or fatigue. MI may occur with atypical symptoms such as palpitations or cardiac arrest or even without symptoms. For example in women, the elderly, diabetics, or post-operative and critically ill patients **(6).**

- **Laboratory Diagnosis of acute myocardial Infarction**

The following biomarkers have been described in association with acute myocardial infarction:

(a) Troponins: itremains elevated longer than CK up to 14 days. This makes troponins a superior marker for diagnosing myocardial infarction.Various causes have been suggested for the release of structural proteins from the myocardium, including normal turnover of myocardial cells, apoptosis, cellular release of cTn degradation products, increased cellular wall permeability, the formation and release of membranous blebs, and myocyte necrosis **(8).**

(b) Total CK: is a simple and inexpensive test However, an elevation in total CK is not specific for myocardial injury, because most CK is located in skeletal muscle, and elevations are possible from a variety of non-cardiac conditions **(9).**

(c) Creatine kinase-MB fraction (CK-MB):is part of total CK and more specific for cardiac muscle than other striated muscles. It tends to increase within 3 to 4 hours of myocardial necrosis, then peak in a day and return to normal within 36 hours. It is less sensitive than troponins The CK-MB is also useful for diagnosis of reinfarction because it begins to fall after a day so subsequent elevations are indicative of another event **(9,10).**

(d) Myoglobin: is a very sensitive indicator of muscle injury, it is not specific for cardiac muscle and can be elevated with any form of skeletal muscle injury. The rise in myoglobin can help to determine the size of an infarction. A negative myoglobin can help to rule out myocardial

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infarction. It is elevated even before CK-MB **(8).**

Electrocardiographic Detection of Myocardial Infarction:

ECG is the most important test and should be done within 10 min of the first medical contact.Recording several standard ECGs with fixed electrode positions at 15 – 30 min intervals for the initial 1 - 2 h, or the use of continuous computer-assisted 12-lead ECG recording to detect dynamic ECG changes, is reasonable for patients with persistent or recurrent symptoms or an initial non-diagnostic ECG**(11)**.

Serial or continuous ECG recordings may be helpful in determining reperfusion or reocclusion status. In general electrocardiography (ECG) has a unique value in the diagnosis and provides prognostic information for patients with AMI. Despite traditional ECG markers including ST elevation and pathological Q wave in ST elevation AMI, there is no specific ECG finding for diagnosing or predicting the poor prognosis of patients with non-ST elevation AMI. The lack of such ECG markers has prompted clinicians to explore new parameters **(12).**

The first such parameter was the QRS duration that is prolonged owing to the ischemia-related conduction delay in the Purkinje system and the myocytes. In patients with myocardial infarction, prolonged QRS duration was reported to be associated with cardiac remodeling, low left ventricular ejection fraction, imperfect reperfusion, and a high incidence of in-hospital cardiovascular events **(13).**Recently, P wave duration, P wave dispersion, and P wave peak time (PWPT) have been studied in patients with ST-segment elevation myocardial infarction and shown to be associated with reperfusion success and AF development **(14).**

Echocardiography

The strength of echocardiography is the assessment of cardiac structure and function, in particular myocardial thickness, thickening and motion. In addition to providing fundamental information on the chamber size, systolic function, and valvular integrity, 2D echocardiography can be used to analyse characteristics of diastolic filling**(15)**.

1-Transmitral flow velocity: the early diastolic peak filling velocity when the transmitral pressure gradient is greatest generates the E wave velocity on the echocardiogram. The late diastolic peak filling velocity associated with atrial contraction generates the A wave**(Figure 1)**. Because the normal atrial contribution to total diastolic filling is only 30%, a normal A wave is smaller than the mitral E wave, with an E/A ratio >1. DD initially produces a low E wave and a high A wave velocity, with reversal of the E:A ratio. As disease progresses and LV compliance is reduced further, LA pressure progressively increases to maintain a transmitral pressure gradient. The E wave increases until E/A ratios are >1.5. During the process of this transition, the E/A ratio will temporarily normalize, despite the presence of moderately severe disease. This is referred to as pseudonormalization and highlights a limitation to the sole use of E/A ratios for diagnosis. This problem can be overcome by altering the loading conditions on the myocardium, for example, with Valsalva or glycerinetrinitrate administration during echocardiography.

2-Pulmonary venous flow (PVF): the pulsatile PVF pattern is generated by the x and y descents of the LAP tracing. Atrial relaxation (x-descent) and LV diastole (y-descent) cause forward PVF. During atrial systole, there is normally a small amount of retrograde PVF. In DD, PVF reversal associated with atrial contraction

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becomes progressively more pronounced as LAP increases.

3-Deceleration time (DT): the rate of dissipation of the transmitral pressure gradient is also a function of LV compliance. The faster the LV pressure decreases the shorter the DT. Normal DT is 180–240 ms. Again, the prolongation of the DT seen in early DD is reversed in moderate to severe disease, as there is a progressive compensatory increase in LAP **(16).**

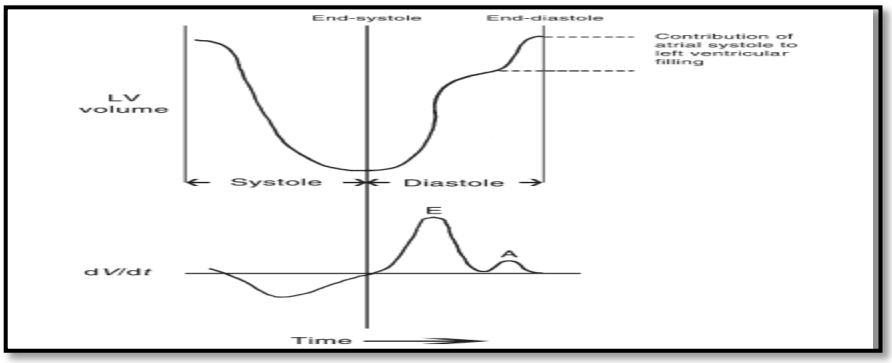

Figure (1): Normal left ventricular haemodynamics. The upper panel shows the change in LV volume through a single cardiac cycle. The lower panel shows the rate of change of the LV volume (dV/dt). The cardiac cycle begins at end-diastole. With the onset or systole, LV volume decreases until end-systole. dV/dt reaches its maximum during early diastole and obtains a secondary peak filling rate with atrial systole. It is these two peak filling rates, E and A, that we use for echocardiographic evaluation of DD.

Coronary angiography

Coronary angiography most often combines diagnosis with percutaneous coronary intervention (PCI) ie, angioplasty, stent placement. When possible, emergency coronary angiography and PCI are done as soon as possible after the onset of acute MI (primary PCI). Frequently, the infarction is actually aborted when the time from pain to PCI is short (<3 to 4h) **(1).** It obtained urgently for patients with STEMI, patients with persistent chest pain despite maximal medical therapy, and patients with complications, patients with uncomplicated NSTEMI whose symptoms have resolved typically undergo angiography within the first 24 to 48 h of hospitalization to detect lesions that may require treatment. Some experts also recommend that angiography be done before hospital discharge in STEMI patients with inducible ischemia on stress imaging or an ejection fraction < 40% **(1).**

Prehospital Care and Initial Management of AMI:

Specific prehospital care includes the following intravenous access, pulse oximetry, supplemental oxygen if the oxygen saturation is less than 90% on room air,immediate administration of oral Aspirin, nitroglycerin for active chest pain, given sublingually or by spray, and prehospital electrocardiography (ECG), if available**(Figure 2)(17)**.

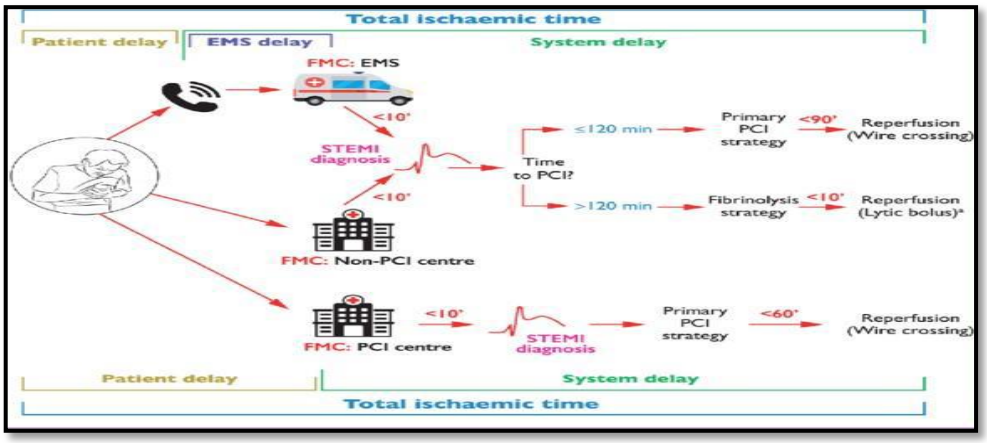

Fig.2: Modes of patient presentation, components of ischemic time and flow chart for reperfusion strategy selection (17).

Additional objectives of prehospital care include adequate analgesia (generally achieved with Morphine); pharmacologic reduction of excessive sympathoadrenal and vagal stimulation; treatment of hemodynamically significant or symptomatic ventricular arrhythmias (generally with Amiodarone and Lidocaine); and support of cardiac output, systemic blood pressure, and respiration. Prehospitalfibrinolytic therapy by the administration of tissue-type plasminogen activator (t-PA), Aspirin, and Heparin may be given to patients with MI, as guided by electrocardiographic findings, within 90 minutes of the onset of symptoms. This treatment improves outcomes, as compared with thrombolysis begun after the patient arrives at the hospital **(18).**

Emergency Department Care and In-Hospital Management:

All patients presenting to the emergency department with symptoms suggestive of acute myocardial infarction (MI) should be evaluated with a targeted history and focused physical examination. A 12-lead electrocardiogram (ECG) interpreted by an experienced physician should be completed within 10 minutes of arrival, in addition to establishing intravenous (IV) access. The initial management of the overall management plan for patients with acute MI has the following aims: restoration of the balance between oxygen supply and demand to prevent further ischemia, pain relief, and prevention and treatment of complications**(18)**.

- Continuous oxygen saturation monitoring by pulse oximetry is needed for all patients. Supplemental oxygen by a mask or nasal cannula is indicated only for patients who are breathless, hypoxic (oxygen saturation <90%), or who present with heart failure **(19).**
- **Aspirin:** all patients presenting with acute coronary syndrome should receive aspirin in a dose of at least 162 to 325 mg, unless there is a clear history of aspirin allergy. Chewed aspirin is preferred, as this promotes rapid absorption into the bloodstream to achieve faster therapeutic levels **(18).**
- **Reduction of cardiac pain:** Nitrates are usually given as a 0.4 mg dose in a sublingual tablet, if the initial dose is well tolerated, further nitrates can be administered. When chest pain persists or recurs, IV nitrates are indicated, usually started at a dose of 5 to 10 µg/min and gradually increased

until relief of chest pain is achieved. Nitrates not be used in patients presenting with marked hypotension or bradycardia, or if there is suspicion of right ventricular infarction. Special attention should be made in taking with concomitant use of phosphodiesterase inhibitors (eg, sildenafil) has occurred within the last 24 to 72 hours, because this drug combination may lead to life-threatening hypotension **(18).**

- **Analgesia:** refractory or severe pain should be treated symptomatically with IV morphine. The initial dose of morphine of 2-4 mg as an IV bolus can be given, with increments of 2-4 mg repeated every 5 to 10 minutes until the pain is relieved or intolerance is manifested by hypotension, vomiting, or depressed respiration. The use of other analgesic agents, such as non-steroidal anti-inflammatory drugs should be avoided if at all possible, as the use of these agents has been associated with adverse cardiovascular events **(19).**

ST-Elevation Myocardial Infarction

Management of ST-elevation myocardial infarction (MI) (STEMI) relies on two essential and key components: rapid recognition and timely reperfusion. Minimizing delays has been associated with improved overall outcomes as well as reduced mortality and long-term morbidity **(2).**

Reperfusion

Early mechanical intervention (primary PCI) or pharmacologic reperfusion should be performed as soon as possible for patients with clinical presentation of STEMI within 12 hours of symptom onset and who have persistent ST-segment elevation or new or presumed new left bundle branch block (LBBB). In addition, it is reasonable to consider an early reperfusion strategy for patients presenting after more than 12 hours, provided there is clinical and/or ECG evidence of ongoingischemia, with primary PCI being the preferred method in this population**(12)**.

For patients presenting to a non-PCI-capable hospital, if they cannot be transferred to a PCI-capable hospital within 120 minutes, it is very important to rapidly assess the following to reach a decision about administration of fibrinolytic therapy: the time from onset of symptoms, the risk of complications related to STEMI, the risk of bleeding with fibrinolysis therapy, the presence of shock or severe heart failure, and the time required for transfer to a PCI-capable hospital **(2).**

Primary percutaneous intervention

PCI achieves superior reperfusion outcomes and is associated with less complications, death, and long-term complications of STEMI when compared to fibrinolytic therapy **(21).**

Current guidelines strongly recommend performing primary PCI in patients presenting with symptoms of less than 12 hours duration, or those who present with cardiogenic shock or who develop acute severe heart failure, irrespective of time of delay from onset of symptoms. Guidelines also recommend considering primary PCI for patients who present between 12 and 24 hours after onset of symptoms, provided there is ongoing clinical or ECG evidence of myocardial ischemia **(22).**

Fibrinolysis

Fibrinolysis is an important reperfusion strategy, particularly in settings where primary PCI cannot be offered to STEMI patients within the recommended timelines. The benefit of fibrinolytic therapy in patients with STEMI

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is well established, with the largest benefit seen when administered early (within 12 hours after symptomatic onset) and in patients with the highest cardiovascular risk, including patients older than 75 years**(Table 1)**. Fibrinolytic therapy may not be beneficial in patients who present more than 12 hours after symptomatic onset, although current practice guidelines recommend consideration of fibrinolysis in symptomatic patients with a large area of myocardium at risk (based on ECG or cardiovascular imaging) or hemodynamic instability if PCI is unavailable. See absolute and relative contraindications to fibrinolytic therapy **(23)**.

Table 1: Absolute and relative contraindication for fibrinolytic therapy in patients with STEMI.

Absolute Contraindications*	Relative Contraindications*
• Any prior ICH • Known structural or cerebral vascular lesion (e.g., AVM, hemorrhagic stroke) • Known malignant intracranial neoplasm (primary or metastatic) • Ischemic stroke within three months, except acute ischemic stroke within 4.5 hours • Suspect aortic dissection as the primary diagnosis • Active bleeding or bleeding diathesis (excluding menses) • Significant closed-head or facial trauma within three months • Intracranial or intraspinal surgery within two months • Severe uncontrolled hypertension (unresponsive to emergency therapy)	• History of chronic, severe, poorly controlled hypertension • Significant hypertension on presentation (SBP > 180 mm Hg or DBP > 110 mm Hg) • History of ischemic stroke > three months • Dementia • Known intracranial pathology not covered in absolute contraindications • Traumatic or prolonged (> 10 min) CPR • Major surgery (< three weeks) • Recent (within two to four weeks) internal bleeding • Noncompressible vascular punctures • Pregnancy • Active peptic ulcer • Oral anticoagulant therapy
** Viewed as advisory for clinical decision making and may not be all-inclusive or definitive.* *CPR = cardiopulmonary resuscitation; DBP = diastolic blood pressure; SBP = systolic blood pressure*	

Coronary artery bypass grafting (CABG)

CABG is indicated for cardiogenic shock, failed PCI, high-risk anatomy, surgical repair of a mechanical complication of STEMI (eg, ventricular septal rupture, free-wall rupture, or severe mitral regurgitation from papillary muscle dysfunction or rupture) **(24).**

Antiplatelet agents

All patients with STEMI should receive an empiric loading dose of aspirin (150 to 325 mg) as early as possible and prior to reperfusion, regardless of the reperfusion method. A lifelong maintenance dose of (75 to 81 mg) daily should be prescribed to all patients after STEMI. Other antiplatelet agents used for dual antiplatelet therapy are the P2Y12 receptor inhibitors (eg, clopidogrel, ticagrelor, prasugrel); a loading dose of these agents is given before or at the time of reperfusion and an extended duration maintenance dose is administered thereafter, depending on the method of reperfusion**(12)**.

For patients undergoing primary PCI, a loading dose of 600 mg of clopidogrel, 180 mg of ticagrelor, or 60 mg of prasugrel should be given as early as possible or at the time of primary PCI. A maintenance dose of P2Y12 receptor inhibitors should be continued for at least 1 year for patients who receive a stent, either a BMS or a DES. A daily dose of 75 mg clopidogrel, 90 mg ticagrelor (twice daily), or 10 mg prasugrel is recommended **(25).**

Patients with refractory angina, clinical evidence of heart failure, or

hemodynamic or electrical instability who do not have serious comorbidities or contraindications to angiography/PCI should undergo an early invasive strategy. An immediate early invasive strategy is also recommended for patient who are stable but at a high risk for clinical events. It is reasonable to consider an early invasive strategy within 24 hours of admission in patients with intermediate/high risk. For patients who fall outside this category, a delayed invasive strategy within 25 to 72 hours of admission versus a conservative (ischemia-guided) strategy may be considered **(26).**

Beta blockers &**Calcium channel blockers**

Beta blockers are recommended to be given orally within the first 24 hours, preferably using one of the three drugs proven to reduce mortality in heart failure patients: metoprolol, carvedilol, or bisoprolol. These agents should also not be given to patients who have a contraindication to beta blockers (eg, first-degree heart block with a PR interval >240 ms, second or third degree heart block without a cardiac pacemaker, recent cocaine use, severe/advanced active reactive airway disease). In patients with chronic obstructive lung disease or chronic asthma, beta-1 selective beta blockers are preferred and should be initiated at low doses **(27).**

Non-dihydropyridine calcium channel blockers (eg, verapamil or diltiazem) should be given for recurrent myocardial ischemia only if there are contraindications to use beta blockers **(27).**

Anticoagulant therapy

Anticoagulant agents are recommended to be given to all patients with NSTEMI, regardless of the initial treatment strategy, in addition to antiplatelet therapy. The following agents may be considered as treatment options from this group of medications. Anticoagulant agents are an important adjunctive therapy for reperfusion therapy regardless of the strategy chosen. After primary PCI, unfractionated heparin (UFH), bivalirudin and low molecular weight heparin (LMWH) (eg, enoxaparin) are the available options **(2).**

Fondaparinux is not used in this setting because of the increased risk of catheter thrombosis.Enoxaparin is given at a dose of 1 mg/kg subcutaneously (SC) every 12 hours. It should be continued for the duration of hospitalization or until PCI is performed. A dose reduction is required for patients with impaired kidney function. Enoxaparin results in a more predictable and efficient anticoagulation compared to unfractionated heparin **(23).**

Bivahrudin is direct thrombin inhibitor that is given as 0.1 mg/kg loading dose, followed by 0.25 mg/kg per hour only in patients managed with an early invasive strategy. This regimen is continued until diagnostic angiography or PCI **(28).**

Fondaparinux is a selective factor X inhibitor. This agent is given as a once-daily SC injection of 2.5 mg, which is continued for the duration of hospitalization or until PCI is performed **(29).**

Additional Aspects of Management and Late Hospital Care

After the initial management and stabilization of the patient in the early and critical phase of acute myocardial infarction (MI), the goals of care for these patients is to restore normal activities, prevent long-term complications, as well as aggressively modify lifestyle and risk factors. This multifaceted goal is achieved with the implementation of important key elements, including the use of

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cardioprotective medications and cardiac rehabilitation, as well as physical activity, diet, and patient education**(12)**.

Cardio-protective medications:

- **Inhibitors of the renin-angiotensin-aldosterone (RAA) system& Statins:**

Initiate angiotensin-converting enzyme (ACE) inhibitors and continue administration indefinitely in all patients with a left ventricular ejection fraction that is less than 40% and in those with hypertension, diabetes mellitus, or stable chronic kidney disease, unless contraindicated. Angiotensin-receptor blockers (ARBs) are recommended in patients who are intolerant of ACE inhibitors. All patients with an acute MI should be started on high-potency statin therapy and continued indefinitely. Current clinical practice guidelines, high potency statins such as atorvastatin 40 mg or 80 mg, or rosuvastatin 20 mg are recommended **(26).**

- **Lifestyle modifications and cardiac rehabilitation**

Several lifestyle modifications have been strongly linked to a reduction in recurrent MI and prevention of further progression of cardiovascular disease. These modifications include dietary changes that adopt a low-fat and low-salt diet with dietary counseling, smoking cessation, up-to-date vaccination, and an increase in physical activity and exercise. The recommended frequency of regular exercise training is three or more times a week, for at least 30 minutes per session **(2).**

CONCLUSION:

Myocardial infarction is best managed in the subacute phase by enhancing the discharge planning process, initiating therapies early to prevent recurrent myocardial infarction, and minimising hospital readmission. Up to 25% of the time, evidence-based recommendations are not followed while treating individuals with acute coronary syndrome.

The cornerstone of medical therapy is antiplatelet therapy, which includes beta blockers, statins, renin-angiotensin-aldosterone system inhibitors, and. Early noninvasive stress testing is a vital risk assessment technique, especially for patients who do not receive revascularization.

The process leading up to discharge should include a review of prescription drugs, a recommendation for exercise-based cardiac rehabilitation, activity suggestions, education about lifestyle changes and the identification of cardiac symptoms, and a clear follow-up strategy. Due to the fact that medication nonadherence is frequent in patients who have had a myocardial infarction and is connected.

Conflict of interest: The authors declare no conflict of interest.

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