

## Diagnosis, Management, and Prognosis of Myocardial Infarction (MI)

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### Abstract

Myocardial infarction (MI) is one of the most frequently encountered reasons for hospital admission and is commonly seen in all populations worldwide. For that, an extensive literature search of the Medline, Cochrane and EMBASE databases on has been conducted using the medical subject headings (MeSH) terms. Papers discussing diagnosis, management, and prognosis of MI were screened for relevant information. There were no limits on date, language, age of participants or publication type. The clinical approach to a patient suspected to have an acute MI consists of detailed history taking, electrocardiogram (ECG) changes, cardiac biochemistry evidence and imaging. In the same context. the initial management of acute MI consists of restoring the balance between oxygen supply and demand to prevent further myocardial ischemia, pain relief and prevention of treatment complications. This should be followed by percutaneous coronary intervention (PCI), coronary angiography, thrombolytic therapy, or emergency coronary artery bypass graft (CABG); according to the condition of the patients and the type of MI. Following an acute MI, patients are at risk of further adverse cardiovascular events such as recurrent infarct, death, stroke and heart failure. These outcomes vary depending on the clinical profile and the associated comorbidities.

**Keywords:** Myocardial Infarction; Diagnosis; Management; Prognosis

### Introduction

Myocardial infarction (MI) is one of the most frequently encountered reasons for hospital admission and is commonly seen in all populations worldwide [1]. Theoretically speaking, infarction of the myocardial muscle can be detected based on certain pathological features like coagulation necrosis causing loss of muscle cells (myocytes). When such a pathological change occurs, it triggers inflammation

which subsequently leads to fibrosis and healing with a scar [2]. The clinical approach to a patient suspected to have an acute MI consists of detailed history taking, electrocardiogram (ECG) changes, cardiac biochemistry evidence and imaging [3]. The reliability of each of these modalities as a diagnostic tool is dependent on multiple factors, the most significant being the window period between the time of infarction and the time the patient seeks medical attention [4].

The current approach to patients presenting with typical or atypical features of myocardial ischemia or infarction starts with making the provisional diagnosis of the acute coronary syndrome. Then, depending on the changes seen on the twelve lead ECG, the acute coronary syndrome is classified into ST-elevation MI (STEMI) or non-ST elevation MI (NSTEMI) [5]. In spite of major leaps in the diagnostic tools and treatment of MI, ST-segment elevation MI persists as a leading cause for ill health in both the developed and developing world [6]. The prevalence of coronary disease and infarction is on the rise in the developing countries, with further worsening of adverse cardiovascular events due to disadvantages like inadequate primary prevention policies and limited availability of medical help [7]. The meteorical advances in acute coronary care and resuscitation since the twentieth century has led to considerable decline in mortality and morbidity rates from STEMI. In the early parts of the twentieth century, therapy was centered around passive observation and monitoring than active intervention. Significant advances have opened the gates to the current reperfusion therapy, which along with intensive hemodynamic monitoring has improved the standards of acute coronary care and emergency management. The approach to ST-elevation MI is increasingly leaning towards practice guidelines and evidence-based medicine [8].

The simplest and oldest diagnostic tool for MI is the 12 lead electrocardiograph. In daily practice, even though the ECG remains an important test in diagnosis and detection of progression of the disease, there still remains ample potential for its role as a prognostic marker. The fundamental advantage that stands to be gained by realizing this potential is the time it saves for immediate intervention. In addition, it can serve as an instant and cost-effective method for risk assessment instead of waiting for biochemical and angiography results [9].

### Purpose of the Study

The purpose of this study was to provide an overview of the diagnosis, management and prognosis of MI.

### Methods

We performed an extensive literature search of the Medline, Cochrane, and EMBASE databases on 10 December 2019 using the medical subject headings (MeSH) terms “myocardial infarction [MeSH Terms]”. Papers discussing diagnosis, management, and prognosis of MI were screened for relevant information. There were no limits on date, language, age of participants or publication type.

### Diagnosis of MI

#### Symptoms of MI

The most recent guidelines indicate that symptoms of MI include different combinations of jaw, chest, upper limb, and epigastric pain/discomfort on exerting effort or at rest [10]. This discomfort/pain is usually diffuse and lasts for about 20minutes with no effect on the movement or the position on the pain [11].

Other ischemic symptoms may also accompany MI; including shortness of breath and/or fatigability [10]. Noteworthy, chest pain/discomfort is the most common symptom of MI [10]. However, 44% of the Non-ST Segment Elevation MI (NSTEMI) patients and 27% of the ST-Segment Elevation MI (STEMI) patients; had no chest pain/discomfort [12]. Table 1 shows the frequency of different myocardial infarction symptoms [13].

Symptom	Frequency (%)
Chest pain	80.0%
Breathlessness	35.3%
Diaphoresis	17.6%
Syncope	2.4%
Back pain	1.2%
Epigastria pain	20.0%
Jaw pain	9.4%
Shoulder pain	36.5%
Cold sweating	57.6%

**Table 1:** The frequency of myocardial infarction symptoms among patients [13].

Some studies have shown that women are more likely than men to experience greater numbers of atypical AMI symptoms such as nausea, dizziness, fatigue, sweating, indigestion, and numbness in the hands and palpations [11,14] (Table 2).

Presenting symptom*	Type 1 myocardial infarction		Type 2 myocardial infarction	
	Frequency in Men (%)	Frequency in women (%)	Frequency in Men (%)	Frequency in women (%)
Chest pain, n (%)	93%	93%	87%	82%
Dyspnea, n (%)	32%	39%	45%	36%
Palpitation, n (%)	2%	9%	16%	23%
Syncope, n (%)	1%	1%	11%	5%
Other, n (%)	8%	7%	3%	8%
<b>Symptom feature</b>				
Typical nature†, n (%)	64%	81%	61%	72%
Typical location‡, n (%)	90%	93%	82%	82%
Radiation (any), n (%)	65%	72%	45%	54%
Associated symptoms, n (%)	51%	62%	55%	59%
<b>Symptom feature</b>				
Typical nature §, n (%)	59%	77%	55%	62%
Typical location   , n (%)	41%	23%	45%	38%

**Table 2:** Comparison of symptoms frequency among men and women in MI [14].

\*: Patients reporting more than one symptom were counted for all symptoms reported.

†: Typical nature is pain with descriptors of dull, heavy, tight, pressure, ache, squeezing, crushing or gripping.

‡: Typical location is chest, arm or jaw.

§: Typical pain classified in any patient who described the pain of chest, arm or jaw, with descriptors of dull, heavy, tight, pressure, ache, squeezing, crushing or gripping.

||: Atypical pain classified in any patient who described epigastric or back pain, or pain that was burning, stabbing, indigestion like, or any other pain description, or presentation.

**Cardiac biomarkers**

A variety of biomarkers have been used to evaluate patients with suspected acute myocardial infarction (MI). The cardiac troponins I and T, as well as the MB isoenzyme of creatine kinase (CK-MB), are the most frequently used. Values  $\geq$  99 percentile of the upper reference limit should be considered abnormal [10]. This value for troponin and CK-MB will vary depending on the assay used. An elevation in the concentration of troponin or CK-MB is required for the diagnosis of acute MI [10]. If both are measured and the troponin value is normal but the CK-MB is elevated, MB is likely due to release from noncardiac tissue. Follow-up on such individuals reveals that they do extremely well without subsequent events [15].

Troponin is the preferred marker for the diagnosis of myocardial injury for all diagnostic categories because of its increased specificity and better sensitivity compared to CK-MB [16,17] (Table 3). However, an elevation in cardiac troponins must be interpreted in the context of the clinical history and electrocardiogram (ECG) findings since it can be seen in a variety of clinical settings and is therefore not specific for an acute coronary syndrome (ACS). The new guidelines endorse the concept that if there are elevations of cardiac troponin (cTn) in a situation where ischemia is not present, the term “myocardial injury” should be used [10].

Test type	Cardiac marker	NPV (%)	PPV (%)	FTBP (hours)	Peak (hours)	Specificity (%)	Sensitivity (%)
Single assay	CK	90	20	3 - 8	12 - 24	80	35
	CK-MB	90	25	4 - 6	12 - 24	85	35
	TnI* and TnT*	91	56	4 - 10	—	96	35
Serial assay	CK	99	30	—	—	68	95
	CK-MB	99	73	—	—	95	95
	TnI** and TnT**	98	72	—	8 - 28	95	89

**Table 3:** Cardiac biomarkers used in the diagnosis of AMI [17].

\*: Measured 4 hours after onset of chest pain; \*\*: Measured 10 hours after onset of chest pain; AMI: Acute Myocardial Infarction; FTBP: First Test Becomes Positive; PPV: Positive Predictive Value; TnI: Troponin I; TnT: Troponin T; NPV: Negative Predictive Value.

**Electrocardiographic detection of myocardial infarction**

The electrocardiogram (ECG) is an integral part of the diagnostic workup of patients with suspected MI and should be acquired and interpreted promptly (i.e. target within 10 minutes) after first medical contact [18]. Pre-hospital ECGs reduce the time to diagnosis and treatment and can facilitate the triage of STEMI patients to hospitals with PCI capability if within the recommended time interval (120 minutes from STEMI diagnosis) [19]. Acute myocardial ischemia is often associated with dynamic changes in ECG waveform and serial ECG acquisition can provide critical information, particularly if the ECG at the initial presentation is non-diagnostic.

Recording several standard ECGs with fixed electrode positions at 15 - 30 minutes intervals for the initial 1 - 2h, or the use of continuous computer-assisted 12-lead ECG recording (if available) to detect dynamic ECG changes, is reasonable for patients with persistent or recurrent symptoms or an initial non-diagnostic ECG [20]. Serial or continuous ECG recordings may be helpful in determining reperfusion or reocclusion status. Reperfusion is usually associated with a large and prompt reduction in ST-segment elevation [21]. Table 4 lists ST-segment-T wave (ST-T) criteria suggestive of acute myocardial ischemia that may or may not lead to MI.

<b>ST-elevation</b>
New ST-elevation at the J-point in two contiguous leads with the cut-point: $\geq 1$ mm in all leads other than leads V2-V3 where the following cut-points apply: $\geq 2$ mm in men $\geq 40$ years; $\geq 2.5$ mm in men $< 40$ years, or $\geq 1.5$ mm in women regardless of age
<b>ST-depression and T wave changes</b>
New horizontal or down sloping ST-depression $\geq 0.5$ mm in two contiguous leads and/or T inversion $> 1$ mm in two contiguous leads with prominent R wave or R/S ratio $> 1$

**Table 4:** Electrocardiographic manifestations suggestive of acute myocardial ischemia (in the absence of left ventricular hypertrophy and bundle branch block) [10].

**Reciprocal changes in MI**

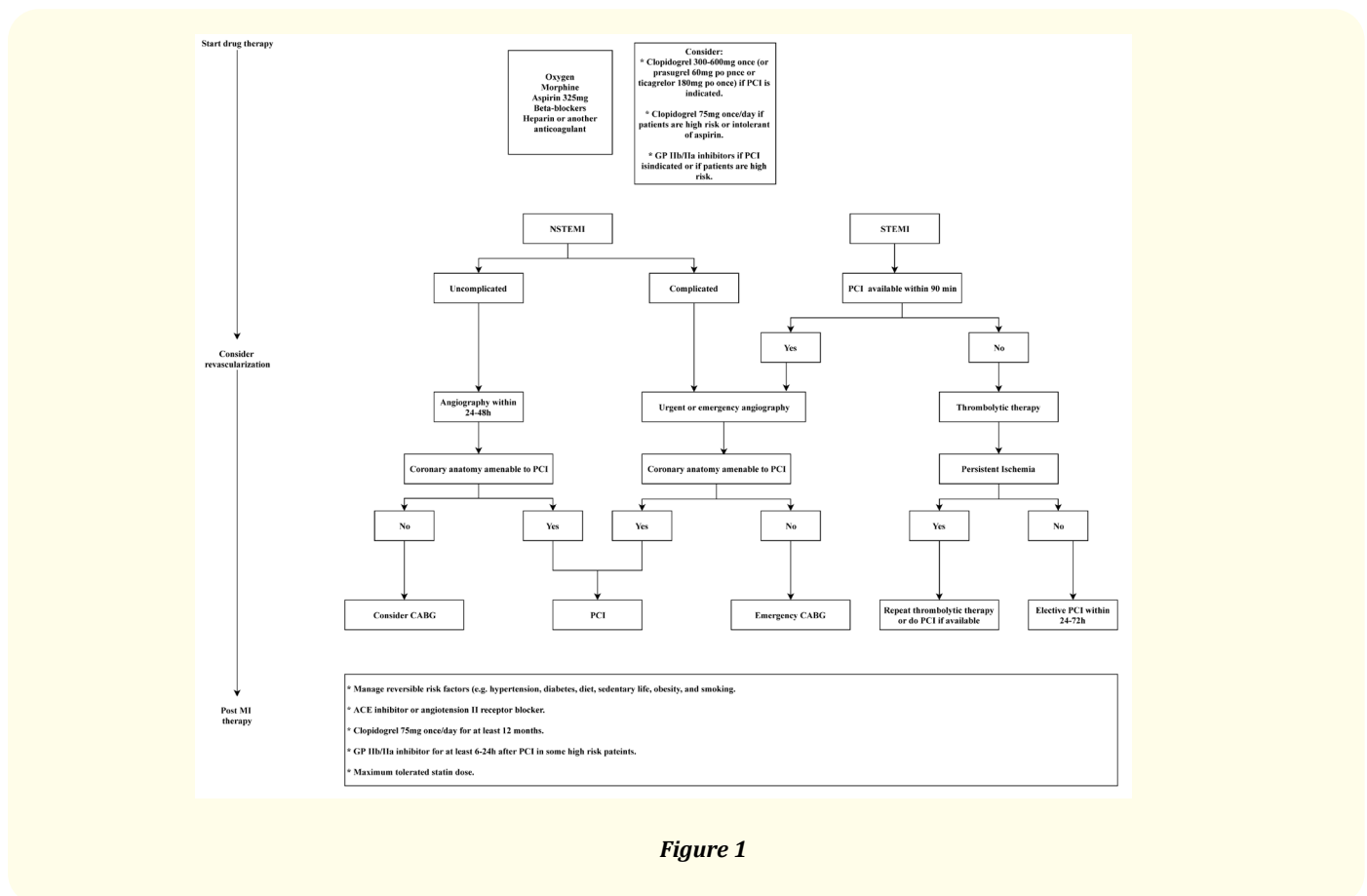
Reciprocal change is defined as ST-segment depression in leads separate and distinct from leads that reflect ST-segment elevation. The concept of reciprocal change cannot be used in patients with abnormal intraventricular conduction, including left bundle-branch block, ventricular paced rhythm and, to a lesser extent, left ventricular hypertrophy via voltage criteria [22]. Reciprocity means that the inferior and anterior ECG leads display inversely proportional changes [23] (Table 5). So, in case of an inferior wall STEMI, the inferior leads II, III and aVF will show ST elevation with reciprocal changes in the form of ST-segment depression in anterior leads and vice versa in an anterior wall infarction [24].

Location of Infarction	Arterial Supply	Indicative Changes	Reciprocal Changes
Anterior	LAD	V1-V4	II, III, aVF
Inferior	RCA	II, III, aVF	I, aVL
Lateral	Circumflex	I, aVL, V5, V6	V1
Posterior	Posterior Descending (RCA)	None	V1, V2
Septal	Septal Perforating (LAD)	Loss of R wave in V1, V2, or V3	None
	Posterior Descending (RCA)		

**Table 5:** Different reciprocal changes in MI [23].

**Management of MI**

Figure 1 summarizes the most recent guidelines for the management of MI [25,26].



**Figure 1**

### Initial management

The initial management of acute MI consists of restoring the balance between oxygen supply and demand to prevent further myocardial ischemia, pain relief, and prevention of treatment complications. Oxygen has been used in acute MI treatment for over 100 years and was first advocated by Steele [27]. The justification for which is that it increases the oxygen delivery to ischemic myocardium thereby reducing the size of MI and improving clinical outcomes. The routine use of supplemental oxygen for treatment of acute MI is recommended by international clinical guidelines [25,26].

However, a Cochrane review in 2013 demonstrated no advantage of oxygen over room air for patients with suspected MI [28]. Subsequently, the Air Versus in Myocardial Infarction (AVOID) trial confirmed this [29]. Therefore, the more recent guideline suggests that oxygen should be administered when blood oxygen saturation is less than 90% or if the patient is in respiratory distress [25,26]. Pain relief approaches are important for patient comfort and also since pain is associated with sympathetic activation, it could increase the cardiac workload. Therefore, intravenous morphine, intravenous beta-blockers, and nitrates are considered for MI patients when there are no contraindications [25,26].

### Percutaneous coronary intervention (PCI)

In 1977, Gruentzig, *et al.* introduced one of the most important therapeutic advances of 20<sup>th</sup>-century medicine by the re-opening of severely stenosed coronary artery in humans using balloon angioplasty [30,31]. Adoption of this technique was widespread, and a number of technological advances have evolved the procedure to what is now referred to as percutaneous coronary intervention (PCI), a collective term used for coronary angioplasty, thrombus extraction and stenting. While PCI is intensive and more difficult to undertake than the administration of thrombolysis, it offers better clinical outcomes. A meta-analysis of 23 randomized trials with 7,739 acute MI patients demonstrated PCI resulted in reduced mortality rate, non-fatal re-infarction, and stroke compared to thrombolysis [32]. However, the benefit of PCI is only evident when patients are treated early after the onset of symptoms [33]. Efficient and effective clinical systems that are able to deliver timely and consistent reperfusion allow for the advantage of primary PCI.

### Thrombolytic therapy

Although Tillet, *et al.* [34] fortuitous research on thrombolytic agents laid the groundwork for the use of thrombolytic therapy using streptokinase in the early 1930s, the intracoronary infusion of streptokinase was initiated only in late 1970s following DeWood, *et al.* [35] angiographic study. The first randomized multicenter trial, Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico (GISSI), in 1986 [36], validated streptokinase as an effective therapeutic method and established a fixed protocol for its use in acute MI, and thus the thrombolytic era began. Following streptokinase, tissue-plasminogen activators (t-PA) evolved as an optimal method. The Global Utilization of Streptokinase and Tissue plasminogen activator for Occluded coronary arteries (GUSTO-1), a landmark trial, demonstrated optimal vessel patency at 90 minutes after the administration of intravenous t-PA administration, which resulted in 15% reduction in mortality rate in comparison to streptokinase [37].

### Prognosis of acute MI

Following an acute MI, patients are at risk of further adverse cardiovascular events such as recurrent infarct, death, stroke and heart failure. These outcomes vary depending on the clinical profile and comorbidities; thus risk stratification models should be applied in predicting prognosis [38]. Many reports have shown that short term (in-hospital and one month) and long term (greater than six months) mortality rates following acute MI have been decreasing over the last three decades in developed countries [39]. These improvements have been attributed to the increasingly widespread use of revascularization procedures, effective acute treatment, and long-term secondary prevention [40-42].

Registries examining rates and trends in the mortality rates of STEMI have reported decreasing mortality over the last 30 years; however, most of these studies are presented with data collected prior to 2005 - prior to the implementation of current management and secondary prevention strategies [43,44]. More contemporary patient registries also continue to report decreasing mortality in STEMI [45]. The Registry of Information and Knowledge about Swedish Heart Intensive Care, Sweden, reported that in-hospital mortality decreased from 11.8% to 5.1% and one-month mortality reduced from 14.2% to 6.3% from 1996 to 2007 [46]. In contrast, a North California based health database reported from that 1999 to 2008 there was no significant reduction in mortality rate (odds ratio 0.93; 95% confidence interval 0.71 to 1.20) [45]. This may be attributed to the differences in clinical practices between countries [47]. Short-term mortality is lower in patients with NSTEMI (2% to 4%) compared to patients with STEMI (3% to 8%) treated with primary PCI within two hours of hospital arrival [48]. Better short-term outcomes for patients with NSTEMI have also been noted in other studies (e.g. in-hospital mortality 5% to 7% compared with 7% to 9.3% with STEMI in the GRACE and European Heart registries) [49,50].

### Conclusion

MI is a very complicated disease with various atypical presentations; thus, a structures approach for diagnosis is a must. Further assessment and risk stratification is the key for better management and a better prognosis, accordingly.

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### Conflicts of Interest

No conflicts related to this work.

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