



Primary Percutaneous Coronary Intervention in ST-segment Elevation Myocardial Infarction

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ABSTRACT

Globally, one of the most important cardiovascular diseases is acute coronary syndrome and is one of the leading causes of death, and its incidence is increasing among the elderly. Older adults are disproportionately affected by coronary ischemic heart disease. Indeed, it is believed that patients 65 years of age or older account for more than 60% of cases of ST-segment elevation myocardial infarctions (STEMIs). For many patients who report with STEMI, 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. Variations in the chronology of patients' presentation and diagnosis can exacerbate these delays, which are detrimental to patient outcomes.

Keywords: Acute myocardial infarction; Myocardial infarction; Primary percutaneous coronary intervention; ST-segment elevation myocardial infarction

INTRODUCTION

Myocardial infarction is the acute form of coronary artery disease that is associated with great morbidity and mortality, so it necessitates early and rapid management. It is categorized into several types based on the underlying mechanisms and severity. The most critical type is ST-elevation myocardial infarction (STEMI), which is characterized by a complete blockage of a coronary artery, leading to substantial damage to the heart muscle. Non-ST-elevation myocardial infarction (NSTEMI), on the other hand, involves a partial blockage, resulting in less

extensive damage but still requiring prompt medical intervention (Figure 1). Type 2 myocardial infarction occurs due to an imbalance between oxygen supply and demand in the heart, often caused by conditions such as severe anemia or arrhythmias rather than a direct blockage (Figure 2). Each type of myocardial infarction requires specific diagnostic and therapeutic approaches to optimize patient outcomes and minimize heart damage [1].

Figure (1): Diagrams showing type 1 MI
Myocardial Infarction Type 2

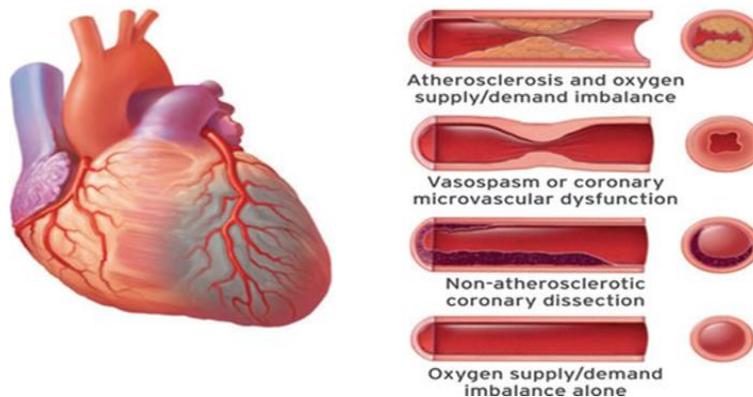


Figure (2): Diagrams showing type 2 MI

Primary percutaneous coronary intervention (PCI) is the preferred course of therapy for patients with acute STEMI, given the numerous studies that have proved its advantages over thrombolysis, including superior results both now and in the future [1].

The concept of PCI began in the late 1970s, when Andreas Gruentzig performed the first balloon angioplasty, marking the beginning of a new era in interventional cardiology [2]. This technique, initially used for stable angina, was soon adapted for acute myocardial infarction, leading to the development of primary PCI as a treatment strategy.

In the 1990s, primary PCI began to gain prominence over thrombolytic therapy for acute MI. Landmark studies, such as the PRIMARY PCI versus Thrombolysis (PPCI) trials, demonstrated that primary PCI was superior to thrombolysis in reducing mortality and improving outcomes in ST-elevation myocardial infarction (STEMI) patients [3,4]. The early 2000s saw further advancements with the introduction of drug-eluting stents and improved antiplatelet therapy, which significantly enhanced the efficacy and safety of PCI [5].

Today, primary PCI remains the gold standard for treating STEMI, supported by evidence from numerous clinical trials and guidelines. It has become the preferred method due to its

ability to rapidly restore coronary blood flow, reduce infarct size, and improve survival rates [6,7]. The ongoing evolution in techniques, devices, and pharmacological adjuncts continues to refine and enhance the outcomes of primary PCI.

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 [8].

The relevance of shortening the interval between the start of symptoms and reperfusion is crucial, as evidenced by recent guidelines. The European Society of Cardiology (ESC) advises primary PCI reperfusion as soon as possible for patients with STEMI who come within 12 hours after symptom onset and show persistent ST-segment elevation on a 12-lead ECG (class I guideline, level of evidence A) [9].

In all STEMI patients, the primary PCI should occur within two hours of the initial medical contact (FMC)(Figure 3). Patients who arrive within two hours after the onset of symptoms or who have an extensive anterior STEMI with minimal bleeding risk should have a 90-minute wait (class I recommendation, level of evidence B)[10].

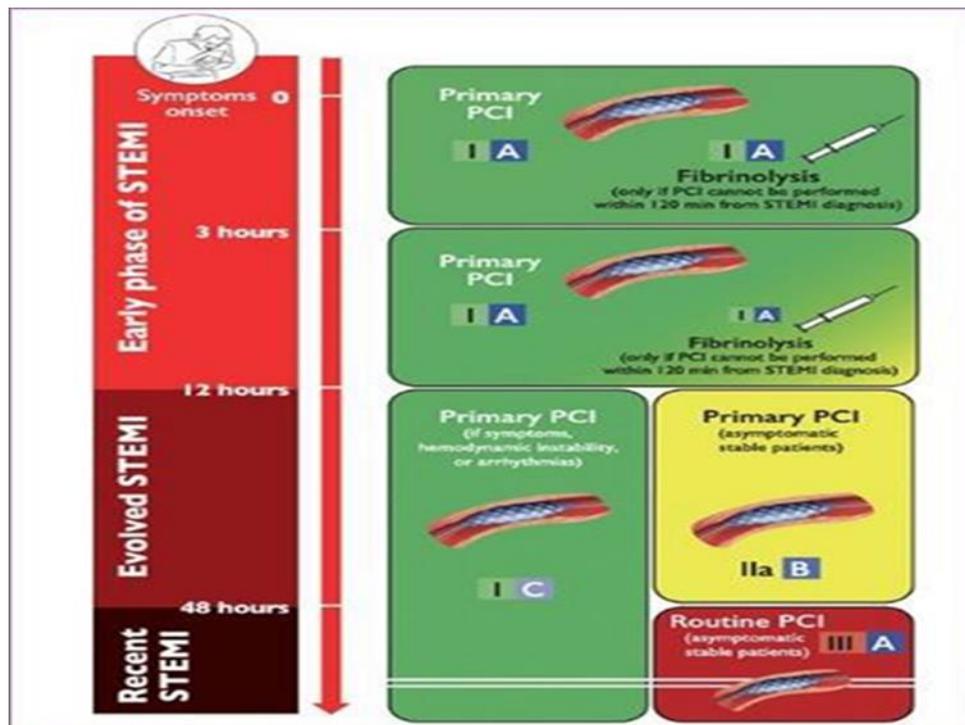


Figure (3): Indication for a reperfusion strategy in IRA according to the time from symptoms onset

Definition

When it comes to STEMI, primary PCI is defined as angioplasty and stenting (DES or BMS) without previous or concurrent fibrinolytic therapy. When possible, it is the optimal therapeutic alternative executed by a team of experienced professionals. Centers with significant mortality rates are lower when the volume of PCI procedures is high among patients undergoing primary PCI [11]. Stents have been shown to be essential during the initial three months following PCI; thereafter, the artery can remain open independently [12].

The possibility of repeating target vessel revascularization during initial PCI is reduced by DES in comparison to BMS [13]. Primary PCI ensures a more comprehensive and long-lasting restoration of perfusion to the infarct-related artery (IRA) than fibrinolysis [14].

Indications and advantages of primary PCI over thrombolytic therapy

It seems safe and effective to use primary PCI in high-risk STEMI patients in hospitals that do not offer cardiac surgery [15].

For the immediate treatment of STEMI, numerous randomized controlled trials have demonstrated the superiority of primary PCI over intravenous thrombolysis (better clinical

outcome including strokes, less recurrent MI, less coronary restenosis, less recurrent myocardial ischemia and more effective restoration of coronary patency). Primary PCI rather than thrombolysis is especially beneficial for women and older individuals. [16].

Throughout the 6-to 18-month prolonged monitoring period, primary PCI continued to yield better results than thrombolytic therapy. Specifically, the combined outcomes of stroke, non-fatal re-infarction, and death were reduced by 19.2% as opposed to non-fatal MI outcomes of 10.0 vs. 4.8%, mortality outcomes of 9.6%, and MI outcomes of 12.8 vs. 9.6% [17].

Access Type: Femoral vs. Radial Access

An angiogram can be performed by employing a radial or femoral approach to access the coronary arteries. In general, the radial artery approach is the recommended strategy to reduce the risk of bleeding at the access site. This is due to the fact that compared to the femoral artery, the radial artery is more prone to compression on the radial bone. Nevertheless, the radial artery's diminutive size necessitates a higher level of expertise and experience.

The palmar arch circulation should be

assessed before access is gained through the radial artery to prevent ischemia of the hand as a result of complications during the procedure. In 2016, 24 trials involving individuals with acute coronary syndrome were the subject of a meta-analysis, including STEMI RADIAL, MATRIX, RIVAL, and RIFLE-STEACS. Patients who underwent the procedure via the radial approach experienced a decrease in major hemorrhage, all-cause mortality, and severe adverse cardiovascular events [18].

Technique

Techniques employed during primary PCI aim to promptly restore blood flow in the

occluded coronary artery. Balloon angioplasty is typically the initial step, involving the inflation of a small balloon at the site of the blockage to dilate the artery. Following this, stent placement is performed to maintain vessel patency. Drug-eluting stents (DES) are often favored over bare-metal stents (BMS) due to their lower rates of restenosis and the need for repeat revascularization procedures. The deployment of these stents releases antiproliferative drugs, reducing the likelihood of neointimal hyperplasia and subsequent re-narrowing of the artery (Figure 4,5) [19,20].

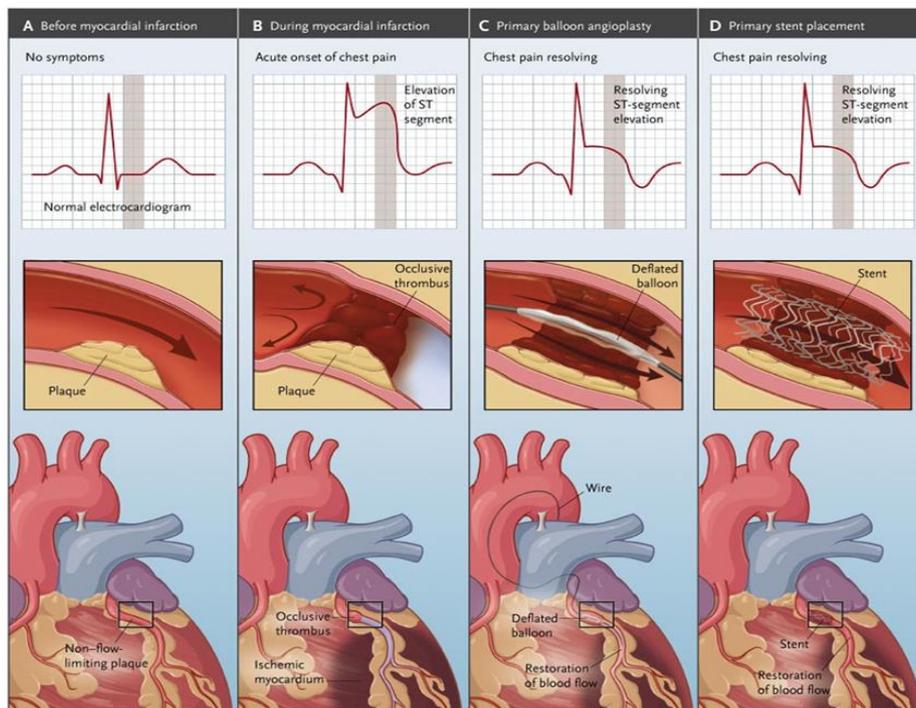


Figure (4):Diagrams showing ST elevation myocardial infarction before, during and after PCI

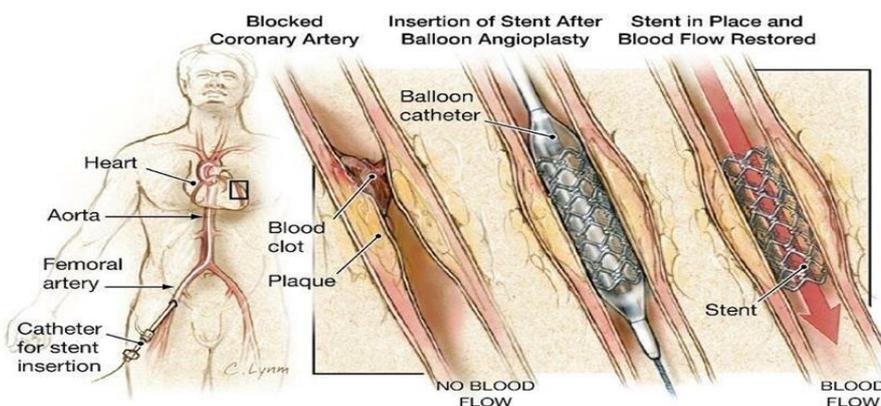


Figure (5):Illustrating the procedure of percutaneous coronary interventions

Specific techniques such as the proximal optimization technique (POT), the kissing balloon technique, and the Culotte stenting technique are employed to optimize stent placement, especially in complex lesions. The POT involves inflating a balloon within the stent's proximal end to ensure it is well-opposed to the vessel wall, which is crucial in bifurcation lesions (Figure 6)[21]. The kissing balloon technique uses two balloons simultaneously to open bifurcational lesions (Figure 7), ensuring both branches are

adequately treated [22]. Culotte stenting, on the other hand, involves deploying stents in both branches of a bifurcation in a manner that ensures complete lesion coverage and minimal overlap[23]. But in acute settings, provisional stenting is often favored over complex bifurcation techniques to reduce procedural time and complications. These straightforward approaches ensure rapid intervention and stabilization, which are crucial for improving patient outcomes in emergency situations[24].

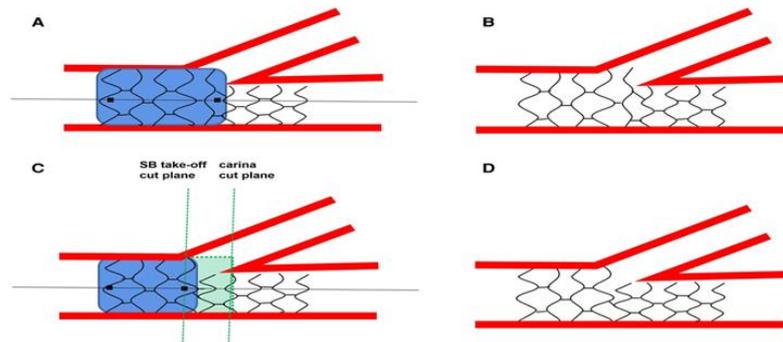


Figure (6): Illustrating proximal optimization technique proposed by the European bifurcation club consensus

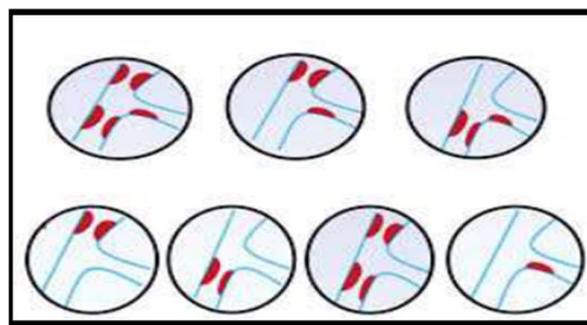


Figure (7): Illustrating true bifurcation lesions

Advanced imaging techniques are integral to the success of primary PCI. Intravascular ultrasound (IVUS) and optical coherence tomography (OCT) provide high-resolution images of the coronary arteries, facilitating precise stent placement and optimizing procedural outcomes. Additionally, thrombectomy devices such as mechanical thrombectomy, aspiration thrombectomy, and rotational thrombectomy are each designed to

physically remove or fragment clots. These devices are particularly beneficial in acute settings like STEMI in patients with a high thrombus burden to enhance the success of the intervention [25,26].

This procedure involves the use of various medications to optimize outcomes and prevent further ischemic events. Antiplatelet therapy is fundamental, with aspirin and P2Y12 inhibitors such as clopidogrel,

prasugrel, or ticagrelor commonly administered to inhibit platelet aggregation and prevent thrombus formation. Additionally, anticoagulants like unfractionated heparin or bivalirudin are crucial during the procedure to prevent clot formation within the coronary arteries. Glycoprotein IIb/IIIa inhibitors, including abciximab and eptifibatid, may be utilized in patients with high thrombotic risk [27,28].

Post-PCI Medications

Following primary percutaneous coronary intervention (PCI), a structured approach to medication management is critical for optimizing patient outcomes and preventing complications. Antiplatelet therapy is foundational, with aspirin and a P2Y12 inhibitor (e.g., clopidogrel, prasugrel, ticagrelor) used to reduce the risk of stent thrombosis and recurrent myocardial infarction. Aspirin is generally continued indefinitely, while the P2Y12 inhibitor is prescribed for 6-12 months based on the type of stent and patient risk factors [29].

Patients who have not undergone revascularization within 24 to 48 hours should be managed with anticoagulants such as enoxaparin or fondaparinux to prevent thrombotic complications. The duration of anticoagulation therapy typically does not exceed 8 days unless there is a clear indication for longer treatment [30].

ACE inhibitors play a crucial role in post-PCI management, particularly for patients with left ventricular dysfunction, diabetes, or hypertension. These medications, such as enalapril or lisinopril, help lower blood pressure, prevent cardiac remodeling, and improve heart function, thereby enhancing long-term cardiovascular outcomes [31,32]. Beta-blockers (e.g., metoprolol, carvedilol) are also important, especially in patients with heart failure symptoms or those at risk for arrhythmias. They work by reducing heart

rate, lowering myocardial oxygen demand, and improving survival rates [33,34].

Statins are prescribed to manage dyslipidemia and stabilize atherosclerotic plaques. Statins like atorvastatin or rosuvastatin lower low-density lipoprotein (LDL) cholesterol levels, significantly reducing the risk of future cardiovascular events [35,36]. For patients experiencing residual angina post-PCI, antianginal medications such as nitrates, beta-blockers, or calcium channel blockers can be used to alleviate symptoms and improve quality of life by reducing myocardial oxygen demand and controlling angina episodes [37,38].

Anti-failure medications are introduced when heart failure symptoms or left ventricular dysfunction are evident. ACE inhibitors and beta-blockers are key components in managing heart failure, while mineralocorticoid receptor antagonists (e.g., spironolactone), SGLT2 inhibitors, and diuretics may be added to control fluid retention and prevent further heart failure progression [39,40]. Combining these with antianginal therapies ensures comprehensive management, addressing both symptoms of heart failure and ongoing angina effectively.

Adverse Effects

Occasionally, complications arise as a consequence of primary PCI. Hematomas, bleeding, pseudoaneurysms, and arteriovenous fistulae at the access site are all examples of local vascular complications. These events occur in 2 to 3% of patients, about two-thirds of whom require transfusion [41].

Approximately 7% of patients who undergo the procedure experience major hemorrhage, which includes bleeding at the access site. The decrease in hemorrhage rates is likely due to the use of smaller catheters and lower heparin dosages, as well as the growing expertise of ancillary personnel and interventional cardiologists. Primary PCI

results in a substantially lower incidence of intracranial hemorrhage than fibrinolytic therapy (0.05% vs. 1%, $P < 0.001$) [42].

Up to 2% of patients experience severe nephropathy following PCI, which is at least partially caused by radiographic contrast material. It is most frequently observed in individuals who are advanced in age and have cardiogenic shock or underlying renal insufficiency [43]. Rarely may radiographic contrast materials cause anaphylactic responses [44].

Approximately 3% of patients have elective balloon angioplasty, and those who have primary balloon angioplasty may be at even higher risk of experiencing an abrupt closure of the infarct-related artery during or shortly after the urgent bypass surgery. The infarct-related artery is stented, which reduces the incidence of precipitous closure to approximately 1%. In the opinion of certain investigators, this procedure eliminates the necessity for on-site surgical capability and reduces the necessity for emergent bypass surgery [45].

Thus, when the coronary anatomy is suitable, stenting is the best initial intervention. The risk of re-stenosis is also reduced by stents, as previously mentioned. This effect is further accentuated by the use of drug-eluting stents [46]. In the majority of stenting trials, less than 1.5% of patients who received a drug-eluting or bare-metal stent during the first year experienced stent thrombosis. Severe cardiovascular events are uncommon in patients undergoing primary PCI [47].

The rates of in-hospital mortality and emergency cardiac surgery were 4.3% and 2.5%, respectively, in a report covering 4366 interventional procedures. Patients who do not have perfusion restored experience these events at a significantly higher rate [48].

PCI options for patients with STEMI and multi-vessel disease include:

1) Primary PCI is for the culprit artery exclusively, while PCI of non-perpetrator arteries is reserved for spontaneous ischemia or intermediate or high-risk indications on pre-discharge noninvasive imaging. 2) Multi-vessel PCI during primary PCI. 3) Primary PCI of the perpetrator artery alone, followed by elective PCI of the non-culprit artery.

Randomized controlled trials (RCTs), observational studies, and meta-analyses comparing culprit artery-only PCI with multi-vessel PCI were used to produce contradicting results [49].

Multi-vessel PCI during acute STEMI is only appropriate for patients with numerous truly significant ($\geq 90\%$ diameter) stenosis or highly unstable lesions (angiographic signals of potential thrombus or plaque breakup), as well as those with persisting ischemia after PCI of the hypothesized culprit lesion. However, regular stent placement in non-culprit lesions without significant stenosis is not advised in patients with cardiogenic shock and multi-vessel disease [50].

Guidelines from the past recommended avoiding primary PCI on non-culprit arterial lesions in individuals with hemodynamically stable STEMI [51]. Several observational studies and meta-analyses have shown that patients who had multi-vessel initial PCI had significantly worse outcomes statistically [52].

Operators who are responsible for performing primary PCIs in STEMI should acknowledge the importance of selecting an appropriate stent size. It is advisable to administer nitrates intracoronary prior to determining the appropriate stent size, as the majority of patients with STEMI experience some degree of coronary spasm. In real-world practice, re-stenosis or stent thrombosis are frequently brought on by stent undersizing, which can also be caused by the presence of a thrombus [53].

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