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Contemporary Management of ST-Segment Elevation Myocardial Infarction

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Abstract: Coronary heart disease is the leading cause of death worldwide. In the United States, approximately 1 of every 6 deaths in 2007 was caused by coronary heart disease. Clinical presentation in the acute setting is mostly due to atherosclerotic plaque rupture leading to flow limitation in the affected vessel, and myocardial ischemia and infarction. ST-segment elevation myocardial infarction is usually associated with complete occlusion of the coronary artery and carries the worst prognosis in terms of in-hospital mortality. Despite various advances in treatment options, including percutaneous coronary intervention, ischemic heart disease still carries a significant morbidity and mortality. In this article, we aim to provide a summary of a few key advances in the management of ST-segment elevation myocardial infarction.

Keywords: coronary heart disease; STEMI; percutaneous coronary intervention; fibrinolysis; P2Y12 inhibitors; stent

Introduction
Coronary heart disease (CHD) is the leading cause of death worldwide, and in the United States, approximately 1 of every 6 deaths in 2007 was caused by CHD. Acute coronary syndrome (ACS) encompasses unstable angina and acute myocardial infarction (AMI). The current method of ACS classification for purposes of management is based on clinical presentation, serum biochemical markers of cardiac damage, and echocardiographic (ECG) changes. Acute myocardial infarction is classified as either ST-segment elevation myocardial infarction (STEMI) or non–ST-segment elevation myocardial infarction (NSTEMI) according to the ECG changes on presentation. In the case of NSTEMI, confirmation is determined by the finding of elevated serum cardiac biochemical markers, such as cardiac-specific troponin I or T. ST-segment elevation myocardial infarction carries the worst prognosis in terms of early mortality of all presentations of CHD. It requires urgent assessment and treatment in specialist centers that are equipped to manage such emergencies. Rapid recognition and management of STEMI can be life saving. In this article, we aim to summarize some of the key evidence behind the management of STEMI. We discuss various topics, including oxygen therapy, β-blockers, antiplatelet therapy and, most importantly, reperfusion therapy.

Pathophysiology and Reperfusion Therapy
Acute myocardial infarction results from complete or nearly complete lack of perfusion to an area of myocardium. This is usually a result of thrombosis secondary to rupture or erosion of coronary artery plaque, known as atherothrombosis, leading to activation of platelets and the coagulation cascade. ST-segment elevation myocardial infarction is often the result of complete occlusion of the artery by a thrombus, leading to myocardial
necrosis as early as 15 minutes after occlusion, particularly in the absence of any protective collateral circulation. The infarct territory typically extends gradually outward from the inner subendocardium toward the epicardium. The ST-segment elevation seen in STEMI is caused by the loss of adenosine triphosphate–dependent transmembrane ion gradients. Resolution of the ST-segment elevation indicates reperfusion and is one of the best predictive measures of cellular response to reperfusion. Resolution of > 50% of the ST-segment elevation correlates with recanalization of the coronary artery while persistent ST-segment elevation indicates either persistent occlusion of the epicardial coronary vessel or microvascular obstruction secondary to embolism and infarction. Prompt and complete restoration of myocardial perfusion is paramount in the management of STEMI. Myocardium may remain viable up to 4 hours after the onset of ischemia, and therefore may be salvaged with reperfusion, with recovery occurring after 20 minutes of reperfusion. Myocardial reperfusion therapy, consisting of either fibrinolytic therapy or percutaneous coronary intervention (PCI), or both, therefore plays a key role in the management of STEMI. In particular, the more widespread availability of PCI facilities is revolutionizing the management of STEMI.

Fibrinolytic Therapy

The commonly used fibrinolytics are streptokinase, urokinase, and recombinant tissue plasminogen activator. Streptokinase was one of the first fibrinolytic agents used in AMI, achieving reperfusion in about 70% of patients. It was noted that improved patency of the infarct-related artery (IRA) was associated with better left ventricular ejection fraction. The Gruppo Italiano per la Sperimentazione della Streptochinasi nell’Infarto Miocardico (GISSI-1) and Second International Study of Infarct Survival (ISIS-2) studies showed that thrombolysis with streptokinase significantly reduced mortality in hospital and at 1 year. The benefit was found to be greatest with earlier administration of fibrinolytics, within the first few hours after onset of symptoms. The ISIS-2 study also demonstrated that aspirin alone was effective at reducing mortality within the first 30 days of STEMI, and that the addition of aspirin to streptokinase resulted in additive and sustained benefit lasting up to 10 years. Subsequently, the Clopidogrel as Adjunctive Reperfusion Therapy (CLARITY) study showed that dual antiplatelet therapy with clopidogrel plus aspirin was more effective than aspirin alone in achieving sustained patency of the IRA, and the Clopidogrel and Metoprolol in Myocardial Infarction Trial (COMMIT) indicated that dual antiplatelet therapy was superior for reducing mortality in this setting without any significant increase in risk of major bleeding.

Recombinant tissue plasminogen activator is another commonly used agent, which is identical to endogenous tissue plasminogen activator. Recombinant tissue plasminogen activator is less antigenic and more fibrin-specific than streptokinase. It produces rapid and dose-dependent coronary thrombolysis. The Thrombolysis in Myocardial Infarction-1 (TIMI-1) trial demonstrated the superiority of tissue plasminogen activator (alteplase) over streptokinase in opening up occluded coronary arteries. After 90 minutes, reperfusion occurred in 62% of the alteplase-treated patients compared with 31% of streptokinase-treated patients. Similar results were seen in the Global Use of Strategies to Open Occluded Coronary Arteries-1 (GUSTO-1) and TIMI-4 trials, in which there was early TIMI grade 3 reperfusion without an increase in re-occlusion rates and adverse events. The improved perfusion resulted in improved left ventricular function, reduced incidence of arrhythmia, and reduced mortality.

Despite the mortality benefits of fibrinolytic therapy, the most serious concern with this mode of reperfusion therapy is the associated risk of major bleeding, including intracranial hemorrhage, which carries a high mortality risk. This safety concern and the lack of consistent restoration of patency of the IRA has led to the search for more effective reperfusion strategies and the development of PCI as the preferred strategy for treating STEMI.

PCI

Primary PCI (PPCI) is defined as angioplasty and/or stenting as the initial mode of treatment to achieve reperfusion, without the use of fibrinolytic therapy. Percutaneous coronary intervention may also be performed in the case of failure to achieve reperfusion with fibrinolytic therapy (rescue PCI) or performed in a routine manner early after administration of fibrinolytic therapy (facilitated PCI). The rationale for facilitated PCI is to achieve earlier reperfusion in some cases, and prevent re-occlusion of the IRA, which may occur when fibrinolytic therapy is used without PCI. Both rescue PCI and facilitated PCI can be referred to as pharmacoinvasive strategies.

Primary PCI is currently the preferred therapeutic option for the management of STEMI. A meta-analysis of studies comparing fibrinolysis with PPCI showed that PPCI was better than fibrinolytic therapy at reducing overall short-term mortality (7% vs 9%; P = 0.0002), nonfatal reinfarction (3%
vs 7%; \( P < 0.0001 \)), stroke (1% vs 2%; \( P = 0.0004 \)), and the combined endpoint of death, nonfatal reinfarction, and stroke (8% vs 14%; \( P < 0.0001 \)). Primary PCI remained better than fibrinolytic therapy during long-term follow-up and is associated with improved IRA patency, better left ventricular function, and lower incidence of recurrent myocardial ischemia.\(^{15}\) The major hurdle for implementation of PPCI has been the “door-to-balloon” time, as not all hospitals have PCI capabilities. This issue was considered in the Primary Angioplasty in Patients Transferred From General Community Hospitals to Specialized PTCA Units with or without Emergency Thrombolysis-2 (PRAGUE-2) trial\(^ {16}\) and the Danish Trial in Acute Myocardial Infarction-2 (DANAMI-2),\(^ {17}\) which both showed that PCI is the better option compared with fibrinolysis even after interhospital transfer for PCI. The PRAGUE-2 trial compared intravenous streptokinase with transfer for PCI in patients with STEMI who presented to hospitals without PCI capabilities. The 30-day mortality was 10% in the streptokinase group compared with 6.8% in the PCI group (\( P = 0.12 \)), and death/reinfarction/stroke at 30 days was 15.2% in the streptokinase group compared with 8.4% in the PCI group (\( P < 0.003 \)). In DANAMI-2, the primary endpoint (combination of death/clinical reinfarction/disabling stroke) occurred in 8.5% of the patients transferred for PCI compared with 14.2% of those in the streptokinase group (\( P < 0.002 \)). In the PCI-capable centers, 6.7% of the PCI group reached the primary endpoint compared with 12.3% in the fibrinolysis group (relative risk reduction of 45% in the centers with PCI capabilities; \( P < 0.05 \)).

The era of stents has led to an increasing number of patients now undergoing PCI for coronary artery disease (CAD), when many would have previously undergone coronary artery bypass graft (CABG) surgery. Bare-metal stents have been limited by stent thrombosis and restenosis, and newer drug-eluting stents (DES) have been designed to reduce the incidence of restenosis. The drugs eluted by these stents inhibit neointimal growth resulting from smooth muscle cell proliferation. Neointimal hyperplasia most often occurs secondary to inflammation; consequently, immunosuppressive and antiproliferative drugs, such as sirolimus and paclitaxel, are used to inhibit this process. The first successful trials were of sirolimus-eluting stents in 2002 and paclitaxel-eluting stents soon after. Drug-eluting stents reduce the need for target-lesion revascularization compared with bare-metal stents, particularly in the case of patients with diabetes or long-stenotic lesions.\(^ {18}\) New-generation DES, including everolimus and zotarolimus, are also available. The Clinical Evaluation of the XIENCE V\(^ {\text{®}}\) Everolimus Eluting Coronary Stent System IV (SPIRIT IV) trial\(^ {19}\) confirmed the superiority of everolimus DES when compared with paclitaxel DES. Target-lesion failure was significantly reduced in the everolimus DES group (4.2% vs 6.8%; \( P = 0.001 \)). The 1-year rates of myocardial infarction and stent thrombosis were also lower with everolimus DES (1.9% vs 3.1%; \( P = 0.02 \)) for myocardial infarction and 0.17% vs 0.85%; \( P = 0.004 \) for stent thrombosis). Similar results were observed in the Comparison of the Everolimus-Eluting Xience-V Stent with the Paclitaxel-Eluting Taxus Liberté Stent (COMPARE) trial.\(^ {20}\) In a noninferiority trial,\(^ {21}\) zotarolimus DES were shown to be noninferior to everolimus DES. Target-lesion failure occurred in 8.2% of patients in the zotarolimus DES group and 8.3% of those in the everolimus DES group (\( P < 0.001 \)). However, the rate of stent thrombosis was noted to be higher in the zotarolimus group (1.2% vs 0.3%; \( P = 0.08 \)).

Although DES reduce the risks of restenosis and the need for target-lesion revascularization, some long-term trials have demonstrated a paradoxical increase in rates of late stent thrombosis. Delayed arterial wall healing and/or late inflammatory reaction to the polymer coating with DES might explain this.\(^ {22}\) To reduce the risk of stent thrombosis, dual antiplatelet therapy is recommended for \( \geq 12 \) months following DES implantation. In patients with increased risk of life-threatening bleeding, a bare-metal stent should be used, and dual antiplatelet therapy is recommended for \( \geq 1 \) month.\(^ {21}\)

The role of CABG surgery in STEMI is limited to emergencies when urgent surgical intervention is needed. These urgent circumstances might include ongoing ischemia secondary to complicated left main CAD, ischemic valvular dysfunction, ventricular septal defect, and ventricular rupture.\(^ {24}\) However, PCI is becoming a suitable alternative to CABG surgery in some centers in the stable population with multivessel CAD and in those with left main CAD. A collaborative analysis of trials comparing PCI with CABG surgery in patients with multivessel CAD showed similar mortality rates except among patients with diabetes and patients aged \( > 65 \) years, in whom CABG surgery was associated with reduced mortality.\(^ {25}\) Another meta-analysis of 4 trials with a total of 1533 patients randomly assigned to CABG surgery and 1518 randomly assigned to PCI showed that the rates of death, myocardial infarction, and stroke were similar in both groups. However, the need for repeat revascularization in the CABG surgery group was significantly less than that in the PCI group.\(^ {26}\)
Thrombosis Aspiration During PCI

There is growing evidence to suggest that myocardial damage occurs due to distal embolization of the thrombus, either spontaneously or secondary to PCI, resulting in distal blockage of and impairment to the microvascular circulation. Thrombus aspiration in patients with STEMI improves reperfusion, thereby improving clinical outcome when compared with PCI alone, irrespective of baseline clinical and angiographic characteristics. Studies using catheters for thrombus aspiration showed that they significantly lowered the incidence of left ventricular remodeling at 6 months in patients with anterior STEMI. In the Thrombus Aspiration During Percutaneous Coronary Intervention in Acute Myocardial Infarction Study (TAPAS), patients with STEMI were assigned to treatment with thrombus aspiration using 6F Export Aspiration Catheter (Medtronic, Inc.) or balloon angioplasty before stent implantation in the IRA. Complete resolution of ST-segment elevation occurred in 56.6% of patients in the thrombus aspiration and in 44.2% of patients in the conventional PCI group ($P < 0.001$). Combined outcome of death and reinfarction at 1 year was significantly less in the thrombus aspiration group compared with the conventional PCI group (6.7% vs 11.6%, respectively; $P = 0.016$).

Adjunctive Therapy and Secondary Prevention

Adjunctive therapy and secondary prevention now play a vital role in the management of STEMI. The agents with evidence for cardiovascular event reduction include antiplatelets/anticoagulants, lipid-lowering agents, angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers, and β-blockers.

Unfractionated Heparin Versus Low-Molecular-Weight Heparin and Fondaparinux

Unfractionated heparin (UFH) has an established role in treatment of patients with STEMI receiving tissue plasminogen activator or undergoing PCI. However, in a meta-analysis of trials assessing UFH and low-molecular-weight heparin (LMWH) with aspirin and thrombolysis, UFH compared with placebo did not reduce reinfarction (3.5% vs 3.3%) or death (4.8% vs 4.6%), and did not increase incidence of major bleeding during hospitalization. On the other hand, LMWH compared with placebo reduced the risk of reinfarction (1.6% vs 2.2%), with a number needed to treat of 167. It also reduced the risk of death (7.8% vs 8.7%), with a number needed to treat of 111. However, LMWH had an increased risk of major bleeding (1.1% vs 0.4%), with a number needed to harm of 143. The benefits of LMWH remained evident at 30 days.

The Sixth Organization to Assess Strategies in Acute Ischemic Syndromes (OASIS-6) trial compared fondaparinux, a factor Xa inhibitor, with UFH. Fondaparinux reduced the risk of death or reinfarction significantly, with an absolute risk reduction of 1.5% at 30 days ($P = 0.008$). This benefit was not noted in patients undergoing PCI. A lower incidence of severe bleeding was noted with fondaparinux, and the incidence of cardiac tamponade was reduced (0.8% vs 0.5%; $P = 0.02$).

Bivalirudin and Glycoprotein IIb/IIIa Inhibitors

Bivalirudin is a selective (direct) thrombin inhibitor. In a pilot study assessing its pre-hospital use in the management of STEMI, it was shown to reduce the need for glycoprotein IIb/IIIa inhibitors following PCI. Thrombolysis in myocardial infarction flow, before and after reperfusion therapy, and bleeding complications were the same in patients receiving UFH and bivalirudin.

The Harmonizing Outcomes with Revascularization and Stents in Acute Myocardial Infarction (HORIZON-AMI) study also showed that bivalirudin given to patients with STEMI undergoing PCI significantly reduced 30-day rates of major bleeding (4.9% vs 8.3%; $P < 0.001$) and the net adverse clinical events compared with heparin plus glycoprotein IIb/IIIa inhibitors (9.2% vs 12.1%; $P = 0.005$). There was also a significant reduction in the mortality rates at 30 days for both cardiovascular-related (1.8% vs 2.9%; $P = 0.03$) and all-cause mortality (2.1% vs 3.1%; $P = 0.047$).

Clopidogrel and Prasugrel

Clopidogrel is a prodrug that is converted to an active metabolite that binds to and irreversibly antagonizes the platelet P2Y$_{12}$ receptor, which is 1 of 2 platelet receptors for adenosine diphosphate. The addition of clopidogrel to aspirin has been extensively studied and has been proven to reduce mortality and recurrent ischemia. Studies such as the CLARITY study have demonstrated the benefit of clopidogrel in reducing cardiovascular-related mortality. In the CLARITY study, patients were given a 300-mg clopidogrel loading dose followed by 75 mg daily in addition to aspirin up to and including the day of angiography, with a maximum of 8 days (mean duration, 3 days). There was a reduction in the composite endpoint of death from cardiovascular causes, recurrent myocardial infarction, or recurrent
ischemia at 30 days (21.7% vs 15%; P < 0.001). The need for urgent revascularization was reduced from 14.1% to 11.6% (P = 0.03) without an increase in major or minor bleeding complications. Similar benefits were noted in COMMIT. Clopidogrel has 2 key limitations that may impair its efficacy in the management of STEMI by PPCI. First, its onset of action is relatively slow because it takes several hours for a loading dose of clopidogrel to achieve its maximal effect, which may be suboptimal in preventing acute stent thrombosis in the highly thrombogenic situation of STEMI and PPCI. Second, the maximal individual response to clopidogrel varies widely, such that some individuals have a poor pharmacodynamic response, leading to high platelet reactivity that drives an increased risk for stent thrombosis. This interindividual variation in response is partly related to genetic variation, particularly in the gene for cytochrome P450 2C19, which plays an important role in clopidogrel active metabolite formation and has numerous loss-of-function alleles, the most common being the *2 allele. Other contributors to variability in clopidogrel response are drug interactions and disease states (eg, diabetes), while other factors remain to be discovered. These limitations have been overcome to a large extent by the introduction of prasugrel, which is a prodrug like clopidogrel, but has a more rapid onset of action and more potent and consistent irreversible platelet inhibition.

The Therapeutic Outcomes by Optimizing Platelet Inhibition with Prasugrel–Thrombolysis in Myocardial Infarction (TRITON-TIMI) 38 study compared prasugrel with clopidogrel in patients with STEMI and NSTEMI. The composite endpoint of death from cardiovascular-related causes, nonfatal myocardial infarction, or nonfatal stroke occurred less often in prasugrel-treated patients compared with clopidogrel-treated patients (9.9% vs 12.1%; P = 0.0004). Significant reductions in myocardial infarction (7.4% vs 9.7%; P < 0.001) and stent thrombosis (1.1% vs 2.4%; P < 0.001) were also noted. Major bleeding was noted in 2.4% of the prasugrel-treated group compared with 1.8% in the clopidogrel-treated group (P = 0.03). Patients with a history of transient ischemic attack or ischemic stroke had net harm (hazard ratio, 1.02–2.32; P = 0.04). Patients aged ≥ 75 years and patients who weighed < 60 kg had no net benefit with prasugrel, as these groups had higher rates of bleeding with prasugrel. To minimize the risk of bleeding in these groups, some experts have suggested a lower maintenance dose (5 mg) of prasugrel. The clinical efficacy of this lower dose in comparison with clopidogrel has not yet been established.

The TRITON-TIMI 38 STEMI subgroup analysis found no excess bleeding episodes in patients with STEMI undergoing PCI. Prasugrel significantly reduced cardiovascular-related death, nonfatal myocardial infarction, and nonfatal stroke (9.5% vs 6.5%; P = 0.0017). The reduction in secondary endpoints, such as death from cardiovascular-related causes, myocardial infarction, and urgent target-vessel revascularization at 30 days, was also significant.

**Ticagrelor**

Ticagrelor is a reversibly binding P2Y12 inhibitor that belongs to the class cyclopentyltriazolo-pyrimidine, distinct from thienopyridines, and does not require hepatic activation. In the Platelet Inhibition and Patient Outcomes (PLATO) study, patients with ACS with or without ST-segment elevation were randomly assigned to receive either ticagrelor (180-mg loading dose, 90-mg twice-daily dose thereafter) or clopidogrel (300- to 600-mg loading dose, 75-mg dose thereafter) in addition to aspirin (unless intolerant). This resulted in reduction of the primary endpoint, a composite of death from vascular causes, myocardial infarction, or stroke. The primary endpoint occurred in 9.8% of the ticagrelor-treated group versus 11.7% of the clopidogrel-treated group (P < 0.001). There was consistency of effect of ticagrelor evident in the STEMI subgroup analysis. It was noted that there was a 15% relative risk reduction in the primary endpoint for the ticagrelor-treated group compared with the clopidogrel-treated group, with no significant interaction according to ACS subtype. No significant difference was noted in overall rates of major bleeding between ticagrelor and clopidogrel (11.6% vs 11.2%; P = 0.43). Ticagrelor was associated with higher rates of dyspnea (13.8% vs 7.8%; P < 0.001) and, as a result, more ticagrelor-treated patients discontinued study medication (7.4% vs 6%; P < 0.001).

**Statins**

Statins reduce mortality, especially when given early in AMI. The Treating to New Targets (TNT) trial demonstrated an relative risk reduction of 22% in composite primary outcome (death from cardiovascular-related disease, nonfatal myocardial infarction, resuscitation after cardiac arrest, fatal or nonfatal stroke) with intensive lipid-lowering therapy in the stable coronary disease population. The absolute risk reduction was 2.2%.

Evidence suggests that an intensive lipid-lowering regimen helps to reduce mortality and major cardiovascular events when compared with a standard regimen in patients with ACS. Similarly, the Pravastatin or Atorvastatin Evaluation and Infection Therapy (PROVE-IT). Myocardial
Ischemia Reduction with Aggressive Cholesterol Lowering (MIRACL), and Incremental Decrease in End Points Through Aggressive Lipid Lowering (IDEAL) studies all showed that treatment of patients with ACS treated with high-dose atorvastatin reduced the composite endpoint, including death from cardiovascular disease, myocardial infarction, revascularization, and stroke. The secondary endpoints of myocardial infarction, revascularization, and death due to CAD were also reduced.

β-Blockers
β-Blockers have been known to reduce the incidence of cardiac mortality even in primary prevention. Although numerous studies have suggested that the use of β-blockade in the first 12 hours of myocardial infarction reduces infarct size, much controversy is associated with this practice. This is mainly secondary to the results of COMMIT, which showed an increase in the risk of cardiogenic shock in patients receiving early intravenous β-blocker therapy (5% vs 3.9%). However, high-risk patients presenting with pulmonary congestion were not excluded. Moreover, a high dose of metoprolol (15 mg intravenously followed by 200 mg orally) was used.

The Valsartan in Acute Myocardial Infarction (VALIANT) trial showed that the use of β-blockers reduced 3-year mortality rate in patients with heart failure or systolic left ventricular dysfunction after AMI. However, long-term treatment with β-blockers may not be necessary in patients with preserved left ventricular function following AMI.

Angiotensin Pathway Inhibitors
Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers have been studied fairly extensively and have proven to be beneficial in treating myocardial infarction. There is enough evidence to suggest that ACE inhibitors help to reduce the recurrence of myocardial infarction and also reduce the incidence of left ventricular dysfunction/cardiac failure. Studies looking at the long-term use of ACE inhibitors in post–myocardial infarction patients with asymptomatic left ventricular dysfunction showed a reduced recurrence of myocardial infarction independent of left ventricular ejection fraction. Angiotensin-converting enzyme inhibitors also reduced the need for cardiac revascularization. Similar results were seen in studies looking at unselected patients; early administration of ACE inhibitors reduced early deaths, specifically those due to cardiac rupture. This early beneficial effect persisted for 5 years.

A review of several large-scale studies, such as Left Ventricular Remodelling, Neurohormonal Activation and Early Treatment with Enalapril (CONSENSUS-II), GISSI-3, ISIS-4, Chinese Cardiac Study 1 (CCS-1), and Survival of Myocardial Infarction Long-Term Evaluation (SMILE), have shown that ACE inhibitors improved the 30-day mortality, with proportional reduction in 30-day mortality of 2% to 11%. There was greater benefit in high-risk groups with heart failure, tachycardia (heart rate ≥ 100 bpm), and in anterior myocardial infarction.

Studies looking at the use of an angiotensin receptor blocker (valsartan) and captopril showed that valsartan was as effective as captopril in patients who were at high risk for cardiovascular-related events after myocardial infarction. However, combining valsartan with captopril only increased the rate of adverse events without improving survival.

Oxygen Controversy
Oxygen used to be widely recommended as part of the initial treatment of patients presenting with AMI, the rationale being that it might increase the oxygenation of blood flowing to the coronary arteries, and thus reduce myocardial ischemia. However, others have hypothesized that oxygen might play a paradoxical role and reduce coronary blood flow. A recent Cochrane systematic review could identify 3 randomized controlled trials that were unable to demonstrate any benefit of oxygen therapy. Moreover, the meta-analysis found a statistically insignificant increase in mortality in patients treated with oxygen. Due to the lack of trials assessing oxygen therapy after the advances in reperfusion therapy, there is lack of consensus on this issue except in the management of patients with hypoxemia.

Conclusion
The management of STEMI is a complex topic, and we have tried to cover a few key areas. The mainstay of treatment of STEMI is urgent reperfusion therapy, with PPCI being the gold standard. Pharmacoinvasive approaches are appropriate when facilities for PPCI are not readily available. Antiplatelet therapy with aspirin and a P2Y12 inhibitor should be initiated as soon as a diagnosis of STEMI is confirmed. Both prasugrel and ticagrelor are new P2Y12 inhibitors that have been proven to be superior to clopidogrel in patients undergoing PPCI. Drug-eluting stents should be considered in patients without high risk of life-threatening bleeding, as they require 6 to 12 months of dual antiplatelet therapy. Thrombus aspiration prior to
PCI has been shown to provide better clinical outcomes. There is much controversy surrounding early initiation of β-blockers. β-Blockers should be initiated after PCI as part of secondary prevention together with a statin and an ACE inhibitor. In patients having thrombolysis with no evidence of pulmonary edema, a small dose of an intravenous β-blocker might be appropriate. The use of fondaparinux is only appropriate in patients receiving fibrinolysis or no reperfusion therapy.

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Conflict of Interest Statement

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