ESC European Society of Cardiology

European Heart Journal (2017) **00**, 1–8
doi:10.1093/eurheartj/ehx393

ESC GUIDELINES

2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation – Web Addenda

The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC)

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Councils: Council on Cardiovascular Nursing and Allied Professions (CCNAP), Council for Cardiology Practice (CCP).
Working Groups: Cardiovascular Pharmacotherapy, Cardiovascular Surgery, Coronary Pathophysiology and Microcirculation, Myocardial and Pericardial Diseases, Thrombosis.

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The disclosure forms of all experts involved in the development of these guidelines are available on the ESC website www.escardio.org/guidelines

Keywords Guidelines • Acute myocardial infarction • ST-segment elevation • Acute coronary syndromes • Ischaemic heart disease • Reperfusion therapy • Primary percutaneous coronary intervention • Antithrombotic therapy • Secondary prevention • Fibrinolysis • Evidence • Emergency medical system • Antithrombotics • Risk assessment • Quality indicators • MINOCA.

Web Contents

7. Long-term therapies for ST-segment elevation myocardial infarction

7.2. Antithrombotic therapy

7.2.1 Aspirin

Long-term maintenance aspirin treatment is indicated in all post-STEMI patients.[1] The CURRENT–OASIS 7 randomized trial failed to demonstrate a difference in hard clinical outcomes within 30 days when comparing low (75–100 mg/day) and higher doses (300–325 mg/day) of aspirin.[2] However, there were fewer gastrointestinal bleeds with lower doses.[2] Previous meta-analyses also failed to show a benefit for patients taking a higher than 100 mg maintenance regimen, whereas bleeding risk was increased. For long-term prevention, low doses (75–100 mg) are indicated. Patients with a history of hypersensitivity to aspirin can undergo desensitization and continue therapy indefinitely.[3] Patients who are truly intolerant to aspirin should instead receive clopidogrel monotherapy (75 mg/day) as long-term secondary prevention.[4] The use of ticagrelor monotherapy as a replacement for aspirin for secondary prevention after DAPT discontinuation is being investigated and no recommendations can be formulated at the present time.

7.2.2 Duration of dual antiplatelet therapy and antithrombotic combination therapies

As presented in the main text, 12 months DAPT is recommended in STEMI patients who underwent primary PCI or fibrinolysis with subsequent PCI.[5,6] For patients undergoing fibrinolysis without subsequent PCI and for those not reperfused, 1 month DAPT is recommended and prolongation up to 12 months should be considered. The choice of the $P2Y_{12}$ inhibitor agent in each scenario is presented in the main text

The traditional 12-month duration of DAPT that was recommended in previous guidelines, based on the protocols of large pivotal trials post-ACS and from consensus, has been challenged by the results of multiple studies of patients receiving DES for different clinical indications, comparing 12 months with either shorter or longer treatment durations.[7–9] Altogether, these studies suggest that there is room for individualizing DAPT duration according to bleeding and ischaemic risks,[10] particularly beyond 12 months.

To date, there has not been a dedicated study evaluating optimal DAPT duration in patients at high bleeding risk. Several studies have shown that shortening DAPT from 12 months (or longer) to

6 months reduces the risk of major bleeding complications, with no apparent trade-off in ischaemic events.[10] Within the PROlonging Dual Antiplatelet Treatment After Grading stent-induced Intimal hyperplasia studY (PRODIGY) population (n = 2013), which comprised 33% of STEMI patients, individuals at high bleeding risk based on a Can Rapid risk stratification of Unstable angina patients Suppress ADverse outcomes with Early implementation of the ACC/AHA guidelines (CRUSADE) bleeding score of > 40 showed a greater absolute risk of major bleeding and transfusion and no ischaemic benefit if treated with 24 months vs. 6 months of DAPT, whereas no such bleeding liability was observed in patients with a CRUSADE bleeding score of ≤ 40.[11]

The benefit of extending clopidogrel or prasugrel beyond 12 months was evaluated in the DAPT study,[12] but in this trial only 10% of patients presented with STEMI.

The benefit of extending ticagrelor beyond 12 months was evaluated in the Prevention of Cardiovascular Events in Patients with Prior Heart Attack Using Ticagrelor Compared to Placebo on a Background of Aspirin–Thrombolysis in Myocardial Infarction 54 (PEGASUS-TIMI 54) trial. This study examined two doses of ticagrelor (60 mg and 90 mg *b.i.d.*) vs. placebo in patients on aspirin and with a history of MI (53% STEMI) 1–3 years before and with high-risk features; the study showed a reduction in MACE with ticagrelor for 90 mg (HR 0.85, 95% CI 0.75–0.96; P = 0.008) and for 60 mg (HR 0.84, 95% CI 0.74–0.95; P = 0.004).[13] Total mortality was not different, and there was a borderline signal towards reduced cardiovascular mortality when both ticagrelor doses were pooled (HR 0.85, 95% CI 0.71–1.00; P = 0.06), consistent with the reduction in non-fatal outcomes.[13] Stroke incidence was significantly reduced in the 60 mg ticagrelor dose compared with aspirin monotherapy (HR 0.75, 95% CI 0.57–0.98; P = 0.03). The incidence of bleeding was significantly increased in the ticagrelor groups compared with aspirin monotherapy (HR 2.32, 95% CI 1.68 to 3.21; P < 0.001, and HR 2.69, 95% CI 1.96–3.70; P < 0.001 in the 60 mg and 90 mg ticagrelor groups). Regulatory agencies have approved the 60 mg ticagrelor regimen for the treatment of post-MI patients beyond 1 year. A subgroup analysis has shown consistent results in patients who presented with STEMI vs. NSTEMI.[13]

Gastric protection with a PPI is recommended for some patients (see main text). There is no pharmacokinetic interaction between PPIs and ticagrelor or prasugrel, and no clear evidence that the pharmacokinetic interaction of clopidogrel with some PPIs has meaningful clinical consequences.

The Anti-Xa Therapy to Lower cardiovascular events in Addition to Standard therapy in subjects with Acute Coronary Syndrome–Thrombolysis In Myocardial Infarction 51 (ATLAS ACS 2–TIMI 51) trial (n = 15 526, 50% STEMI) tested the addition of rivaroxaban, a factor Xa antagonist, to aspirin and clopidogrel following ACS.[14] In that trial, a low dose of rivaroxaban (2.5 mg *b.i.d.*) reduced the composite primary endpoint of cardiovascular death, MI, or stroke, but also all-cause mortality, over a mean follow-up of 13 months. Stent thrombosis was reduced by one-third. However, this was associated with a three-fold increase in non-CABG-related major bleeding, and intracranial haemorrhage. Importantly, the high dose of rivaroxaban (5 mg twice daily) was not associated with a reduction of death from either cardiovascular causes or any cause, but was associated with a major increase in the risk of bleeding.[14]

In the Thrombin Receptor Antagonist in Secondary Prevention of Atherothrombotic Ischemic Events – Thrombolysis In Myocardial Infarction 50 (TRA 2P–TIMI 50) trial, patients (n = 26 449) with a history of MI, ischaemic stroke, or peripheral artery disease were randomly assigned to receive either vorapaxar 2.5 mg daily or placebo, in addition to standard-of-care therapy, including aspirin, clopidogrel, or both (DAPT in 58% of patients).[15] After a median follow-up of 30 months, vorapaxar significantly reduced the primary endpoint (death from cardiovascular causes, MI, or stroke) driven by a reduction in the rate of MI. However, the ischaemic benefit of vorapaxar was hampered by a significant increase in the rate of Global Utilisation of Strategies To open Occluded arteries (GUSTO)-defined moderate or severe bleeding and a two-fold increase in intracranial bleeding.

8. Complications following ST-segment elevation myocardial infarction

8.1 Myocardial dysfunction

LV dysfunction may occur during the acute and subacute phases of STEMI. This can be transient (i.e. myocardial stunning) or persistent depending on the duration of ischaemia and completeness of reperfusion. Improvement in ventricular function usually occurs following early successful myocardial reperfusion, but it may take weeks and does not always happen.

8.1.1 Left ventricular dysfunction

LV systolic dysfunction: This is the most frequent consequence of STEMI and is still a powerful independent predictor of mortality.[16,17] It is caused by myocardial loss or ischaemic dysfunction (stunning), in some cases worsened by the presence of arrhythmias, valvular dysfunction, or mechanical complications. LV dysfunction may be clinically silent or cause heart failure. Diagnosis is made by clinical and imaging techniques, most frequently echocardiography.

LV aneurysm: Less than 5% of patients with a large transmural MI undergo adverse remodelling with subsequent development of LV aneurysm. Patients frequently develop heart failure, which should be treated according to specific guidelines.[18] Surgical aneurysmectomy seems of no benefit.[19] However, surgery may be considered in patients with large aneurysms and uncontrolled heart failure or recurrent ventricular arrhythmias not amenable to ablation.[20]

LV thrombus: LV thrombus formation is a frequent complication in patients with anterior MI, even in the absence of apical aneurysm.[21–25] For mural thrombi, once diagnosed, oral anticoagulant therapy should be considered for up to 6 months, guided by repeated echocardiography and with consideration of bleeding risk and need for concomitant antiplatelet therapy. However, prospective randomized data on the best anticoagulation regimen, duration, and strategy of combination with antiplatelet agents are lacking. The clinical experience with direct-acting oral anticoagulants in this setting is limited. Recommendations for the concomitant use of antiplatelet agents and anticoagulants in other settings have been published.[26]

Secondary mitral valve regurgitation: LV remodelling with lateral and apical displacement of the papillary muscles, leaflet tethering, and annular dilatation are a common cause of secondary (functional)

mitral regurgitation.[27] This is more often a late complication but may also occur in the subacute setting in patients with extensive infarction, especially in the posterior–lateral region of the LV, causing significant dysfunction of the posteromedial papillary muscle.[28] While transthoracic echocardiography is fundamental for the initial diagnosis, transoesophageal echocardiography may be needed for better definition of the mechanism and severity of mitral regurgitation.[29] The severity of mitral regurgitation may improve with reperfusion and aggressive medical treatment, including diuretics and arterial vasodilators. In non-responders with severe mitral regurgitation and refractory heart failure or haemodynamic instability, urgent or emergency mitral valve surgery is indicated. In these patients, mitral valve replacement is associated with improved survival and LV function compared to medical therapy alone,[30] although the overall mortality rate is relatively high.[31]

8.1.2 Right ventricular involvement

RV involvement most frequently occurs with inferior wall STEMI. Diagnosis can be made by the presence of elevation of the ST-segment ≥1 mm in leads aVR, V1, and/ or in the right precordial leads (V_3R and V_4R), which should be sought routinely in patients with inferior STEMI. Echocardiography is commonly used to confirm the diagnosis of RV involvement, but RV infarcts are also well assessed by CMR.[32] Patients with RV infarction may have an uncomplicated course or develop the typical triad of hypotension, clear lung fields, and increased jugular venous pressure. They also present more frequently with ventricular arrhythmias, AV block, mechanical complications, low cardiac output, and shock.[33] The management of RV ischaemia includes early reperfusion, with particular care in opening the RV branches,[34,35] which may result in a rapid haemodynamic improvement,[36] avoidance of therapies that reduce pre-load (i.e. nitrates and diuretics), and correction of AV dyssynchrony (correction of AF) and/or AV block, with sequential pacing if needed.

8.2 Heart failure

8.2.1 Clinical presentations

Heart failure is the most frequent complication and one of the most important prognostic factors in patients with STEMI.[37,38] Diagnosis during the acute phase of STEMI is based on typical symptoms, physical examination, and chest X-ray. Risk assessment is based on Killip classification. Contrary to chronic heart failure, natriuretic peptides are of limited value for the diagnosis of acute heart failure following MI due to the lack of definite cut-off values for diagnosis in these patients. Determining the mechanism of heart failure in STEMI patients is essential. Although LV systolic dysfunction is the most frequent cause, haemodynamic as well as rhythm disturbances, mechanical complications, and valve dysfunction should be ruled out. Therefore, early evaluation by transthoracic echocardiography is mandatory to assess the extent of myocardial damage, assess LV systolic and diastolic functions and volumes, valve function, and detect mechanical complications. Any unexpected deterioration of the patient's clinical status, with evidence of haemodynamic compromise, should trigger a clinical re-evaluation including a repeat echocardiographic examination, specifically searching for evidence of progressive LV dysfunction, mitral regurgitation, or mechanical complications.[39]

Pulmonary congestion: This may range from mild–moderate (Killip class 2) to overt pulmonary oedema (Killip class 3), resolve early after reperfusion and medical therapy, or evolve to chronic heart failure, which should be managed according to current guidelines.[18]

Hypotension: This is defined as persistent SBP <90 mmHg. It may be due to different causes, including LV or RV dysfunction, low cardiac output, rhythm disturbances, mechanical complications, valvular dysfunction, hypovolaemia, or excess medication. Hypotension may be asymptomatic or lead to clouding of consciousness or syncope. If prolonged, hypotension may cause acute renal dysfunction or other systemic complications. Therefore, severe hypotension should be reversed as soon as possible.

Low cardiac output states: These are characterized by persistent hypotension and signs of poor peripheral perfusion, including renal dysfunction and reduced urinary output. Isolated low cardiac output is most frequently seen in patients with severe RV infarction but can be present in patients with LV dysfunction, mitral regurgitation, or mechanical complications. Doppler echocardiography is essential in the early diagnosis of the mechanism causing this complication.[39]

Cardiogenic shock: Cardiogenic shock is defined as persistent hypotension (SBP <90 mmHg) despite adequate filling status with signs of hypoperfusion. It complicates 6–10% of all cases of STEMI and remains a leading cause of death, with in-hospital mortality rates ≥50%.[40] Cardiogenic shock does not often present before admission; in half of the cases it develops in the first 6 h, and in 75% within the first 24 h.[40] Patients typically present with hypotension, evidence of low cardiac output (e.g. resting tachycardia, altered mental status, oliguria, and cool periphery), and pulmonary congestion. Haemodynamically, it is characterized by cardiac index <2.2 L/min/m^2, wedge pressure >18 mmHg, and diuresis usually <20 mL/h. Shock is also considered to be present if i.v. inotropes and/or mechanical support are needed to maintain an SBP >90 mmHg. In STEMI patients presenting with cardiogenic shock in which PCI-mediated reperfusion is estimated to occur in > 120 min, immediate fibrinolysis and transfer to a PCI centre should be considered. In these cases, upon arrival at the PCI centre, emergent angiography is indicated, regardless of the ST resolution and the time from fibrinolysis administration. It is usually associated with extensive LV damage, but may occur in RV infarction. Mortality appears to be associated with initial LV systolic dysfunction and the severity of mitral regurgitation.[41] Other parameters, such as serum lactate and creatinine levels, predict mortality.[42] The presence of RV dysfunction on early echocardiography is also an important predictor of an adverse prognosis, especially in the case of biventricular dysfunction.[43] Therefore, cardiogenic shock characterization and management do not necessarily need invasive haemodynamic monitoring, but LVEF and associated mechanical complications should be urgently evaluated by transthoracic echocardiography.[39,41,43–45]

8.3 Management of arrhythmias and conduction disturbances in the acute phase

Management of arrhythmias and conduction disturbances in the context of STEMI is presented in the main document.

8.4 Mechanical complications

Mechanical complications may occur in the first days following STEMI, although the incidence has fallen significantly in the era of

primary PCI. Mechanical complications are life-threatening and need prompt detection and management. Sudden hypotension, recurrence of chest pain, new cardiac murmurs suggestive of mitral regurgitation or ventricular septal defect, pulmonary congestion, or jugular vein distension should raise suspicion. Immediate echocardiographic assessment is needed when mechanical complications are suspected

8.4.1. Free wall rupture

Rupture of the LV free wall may occur in < 1% of patients during the first week following transmural infarction and may present with sudden pain and/or cardiovascular collapse, with or without electromechanical dissociation. Older age, lack of reperfusion, or late fibrinolysis appear to be associated with an increased incidence of cardiac rupture. The development of haemopericardium and tamponade, leading to sudden profound shock, is usually rapidly fatal. The diagnosis is confirmed by echocardiography. Because the rupture is characteristically serpiginous through the different layers of the ventricular wall, partial sealing of the ruptured site by thrombus formation and the pericardium may permit time for pericardiocentesis and haemodynamic stabilization followed by immediate surgery.[46] Ventricular repair with pericardial patch (or other materials) is recommended. Mortality rates are in the order of 20–75%,[47] depending on the condition of the patient and of the size and morphology of the rupture. In suitable patients, CMR can complement the diagnosis by identifying the contained cardiac rupture and its anatomical features to guide surgical intervention.[48,49]

8.4.2 Ventricular septal rupture

Ventricular septal rupture usually presents as rapid-onset clinical deterioration with acute heart failure or cardiogenic shock, with a loud systolic murmur occurring during the subacute phase. It may occur within 24 h to several days after MI and with equal frequency in anterior and posterolateral MI. The diagnosis is confirmed by echocardiography and Doppler, which will differentiate this from acute mitral regurgitation, and define the rupture and its size, and quantify the left to right shunt,[50] which can be more precisely confirmed by a Swan–Ganz catheter. The shunt may result in signs and symptoms of acute, new-onset right heart failure. IABP may stabilize patients in preparation for angiography and surgery. Intravenous diuretics and vasodilators should be used with caution in hypotensive patients. Surgical repair may be required urgently, but there is no consensus on the optimal timing for surgery.[51] Early surgery is associated with a high mortality rate, reported as 20–40%, and a high risk of recurrent ventricular rupture, while delayed surgery allows easier septal repair in scarring tissue but carries the risk of rupture extension and death while waiting for surgery. For this reason, early surgery should be performed in all patients with severe heart failure that does not respond rapidly to aggressive therapy, but delayed elective surgical repair may be considered in patients who respond well to aggressive heart failure therapy. Percutaneous closure of the defect with appropriately designed devices may soon become an alternative to surgery.[52]

8.4.3 Papillary muscle rupture

Acute mitral regurgitation may occur 2–7 days after AMI due to rupture of the papillary muscle or chordae tendineae. The rupture may be complete or involve one or more of the heads and is 6–12 times more frequent in the posteromedial papillary muscle because of its single artery blood supply.[53,54] Papillary muscle rupture usually presents as sudden haemodynamic deterioration with acute dyspnoea, pulmonary oedema, and/or cardiogenic shock. A systolic murmur is frequently underappreciated. Emergency echocardiography is diagnostic. Immediate treatment is based on afterload reduction to reduce regurgitant volume and pulmonary congestion. Intravenous diuretic and vasodilator/inotropic support, as well as IABP, may stabilize patients in preparation for angiography and surgery. Emergency surgery is the treatment of choice although it carries a high operative mortality (20–25%). Valve replacement is often required, but cases of successful repair by papillary muscle suture have been increasingly reported and appear to be a better option in experienced hands.[55]

8.5 Pericarditis

Three major pericardial complications may occur: early infarct-associated pericarditis, late pericarditis, or post-cardiac injury (Dressler syndrome) and pericardial effusion.

8.5.1. Early and late (Dressler syndrome) infarct-associated pericarditis

Early post-MI pericarditis usually occurs soon after the STEMI and is transient, whereas late infarct-associated pericarditis (Dressler syndrome) typically occurs 1–2 weeks after STEMI and is of presumed immune-mediated pathogenesis triggered by initial damage to pericardial tissue caused by myocardial necrosis. Both early and late pericarditis are rare in the primary PCI era and are often related to late reperfusion or failed coronary reperfusion, as well as to larger infarct size.[56] Diagnostic criteria do not differ from those for acute pericarditis including two of the following criteria: (i) pleuritic chest pain (85–90% of cases); (ii) pericardial friction rub (≤33% of cases); (iii) ECG changes (≤60% of cases), with new widespread ST-segment elevation, usually mild and progressive, or PR depression in the acute phase; and (iv) pericardial effusion (≤60% of cases and generally mild).[57]

Anti-inflammatory therapy is recommended in post-STEMI pericarditis as in post-cardiac injury pericardial syndromes for symptom relief and reduction of recurrences. Aspirin is recommended as first choice of anti-inflammatory therapy post-STEMI at a dose of 500–1000 mg every 6–8 h for 1–2 weeks, decreasing the total daily dose by 250–500 mg every 1–2 weeks in keeping with 2015 ESC Guidelines for the diagnosis and management of pericardial diseases.[57] Colchicine is recommended as first-line therapy as an adjunct to aspirin/non-steroidal anti-inflammatory drug therapy (3 months) and is also recommended for the recurrent forms (6 months).[57] Corticosteroids are not recommended due to the risk of scar thinning with aneurysm development or rupture.[57] Pericardiocentesis is rarely required, except for cases of haemodynamic compromise with signs of tamponade.

8.5.2. Pericardial effusion

Post-STEMI patients with pericardial effusion who fulfil pericarditis diagnostic criteria should be managed as having pericarditis (see section 8.5.1). Patients without inflammatory signs in whom circumferential pericardial effusion >10 mm is detected or those who become symptomatic for suspected tamponade, should be investigated for a possible subacute rupture by echocardiography or by CMR if

echocardiography is inconclusive.[57] Pericardiocentesis is rarely required. Echocardiography will detect and quantify the size of effusion. If it is blood and re-accumulates fast, exploratory surgery is recommended.

10. Assessment of quality of care

Traditionally, performance measures based on guideline treatment recommendations have been selected. They define the minimum standard of care that might be expected for all patients who meet certain criteria and have no contraindications for a given healthcare intervention. A broader approach is recommended here, including also the evaluation of the organization (structural measures), of key clinical results (outcomes), and of the feedback from the patients' experience (patient-reported outcomes). A comprehensive description of core indicators for the evaluation of quality, benchmarking, and quality initiative implementation for patients with AMI has been developed by the ESC Acute Cardiovascular Care Association.[58] An inverse relationship between the level of compliance with these quality indicators at a hospital level and 30-day mortality has been reported.[59] For indicators to have maximal impact on the healthcare system, clinicians, hospitals, and networks need to commit adequate time and resources for measuring their performance on the indicators and developing strategies for achieving optimal performance.

Local organizations caring for STEMI patients should evaluate the quality of the organization at a system level, starting by belonging to a formal network specifically designed to quickly and effectively manage STEMI patients. These include the existence of written protocols covering: (1) ease of initial contact by patients; (2) capability of immediate diagnosis with pre-hospital interpretation of ECG and decision for immediate transfer to PCI centres; (3) pre-hospital activation of the catheterization laboratory with a single emergency telephone number; and (4) transportation facilities adapted to the environment and distances (e.g. ambulance, helicopter, and/or fixed-wing aeroplanes) equipped with ECG-defibrillators. System participants should systematically record the key times to reperfusion (see *Figure 2* in the Full Text) and have periodical audits for quality assessment by the coordinating centre.

As timely reperfusion therapy is the cornerstone of STEMI treatment, performance measures should include the proportion of patients arriving within the first 12 h who receive reperfusion therapy, and the speed at which reperfusion is achieved according to guideline recommended times depending on mode of entry into the system: EMS, PCI centres, or non-PCI centres. Time delays should be recorded systematically and the proportion of patients receiving primary PCI within recommended times (wire-crossing of IRA within 90 min after STEMI diagnosis or within 60 min of arrival when patients present directly to PCI-capable hospitals) periodically audited. Note that these target delays for implementation of primary PCI are quality indicators and they differ from the maximal PCI-related delay of 120 min, which is useful in selecting primary PCI over immediate fibrinolysis as the preferred mode of reperfusion. Ideally, these should be subjected to national audits, which is not the case in the majority of European countries.

Other performance measures include the proportion of patients receiving appropriate $P2Y_{12}$ inhibition during hospitalization, high-intensity statins, beta-blocker, or ACE inhibitors at discharge in patients with LVEF equal or less to 40% or clinical evidence of heart failure, counselling for smoking cessation at discharge, and recommendation for enrolment in a secondary prevention/cardiac rehabilitation programme. The inclusion of outcomes as quality indicators is a matter for debate due to the different factors influencing outcomes, such as mortality, which are not quality-related (e.g. age and initial clinical situation). However, analysis of key outcomes such as 30 day risk-adjusted mortality and readmission rates may be helpful in gaining a global perspective of the quality of the system, and pointing out the need for quality improvement, particularly when marked differences between comparable centres are found. Finally, the patient perspective should be considered. Input about the management of their pain, quality of explanations received by doctors and nurses during hospitalization related to the disease, the benefit/risk of treatments, and the quality and accuracy of information provided before discharge related to self-care, lifestyle advice (including smoking cession and diet counselling), rehabilitation programmes, secondary prevention drugs, and medical follow-up are potential tools for global quality improvement.

Opportunity-based composite quality indicators analyse the performance of sets of different quality indicators as all-or-none responses.[60] For STEMI, composite quality indicators are calculated for patients with LVEF >40% and no evidence of heart failure (proportion of patients receiving low-dose aspirin, a $P2Y_{12}$ inhibitor, and high-intensity statins) and for patients with LVEF ≤40% and/or clinical evidence of heart failure (same as previous plus ACE inhibitors, or ARBs, and beta-blockers). The relationship between these composite quality indicators and mortality after AMI has been validated in different populations.

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