Overview of the nonacute management of ST-elevation myocardial infarction

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INTRODUCTION

Once the diagnosis of an acute ST-elevation myocardial infarction (STEMI) is made, the early management of the patient involves the simultaneous achievement of several goals including relief of ischemic pain, assessment of the hemodynamic state and correction of abnormalities that are present, initiation of reperfusion therapy with primary percutaneous coronary intervention or fibrinolysis, and initiation of antithrombotic therapy. (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department" and "Overview of the acute management of ST-elevation myocardial infarction".)

These early diagnostic and therapeutic interventions are followed by the initiation of short- and long-term interventions aimed at improving in-hospital and long-term outcomes. This topic will summarize the management of patients with acute STEMI in the hours and days following the very early decision-making period. (See "Overview of the acute management of ST-elevation myocardial infarction".)

The management of the patient with a non-ST-elevation or non-Q-wave MI or with a complication of an acute MI (eg, cardiogenic shock, mitral regurgitation, ventricular septal defect) is discussed separately. (See "Overview of the acute management of non-ST-elevation

acute coronary syndromes" and "Prognosis and treatment of cardiogenic shock complicating acute myocardial infarction" and "Acute myocardial infarction: Mechanical complications".)

FURTHER MEDICAL THERAPY

Aspirin and a platelet $P2Y_{12}$ receptor blocker are usually given as soon as the diagnosis of acute STEMI is made. (See "Overview of the acute management of ST-elevation myocardial infarction", section on 'Routine medical therapy'.)

Early initiation of the following therapies may be of benefit in patients hospitalized with STEMI. Many of them are useful long term. (See 'Preparing for discharge' below.)

Oral beta blockers — An oral cardioselective beta blocker should be continued or begun if the patient has not received early, oral beta blocker therapy. Patients who did not receive a beta blocker during the first 24 hours because of early contraindications should be reevaluated for beta blocker candidacy.

Most patients are continued on an oral beta blocker indefinitely. The efficacy of beta blocker therapy is related in part to the degree of heart rate slowing. These issues are discussed in detail separately. (See "Acute myocardial infarction: Role of beta blocker therapy".)

The concomitant use of beta blockers and angiotensin inhibitors is discussed separately. (See "Acute myocardial infarction: Role of beta blocker therapy", section on 'Contraindications'.)

Nitrates — Nitrates can be useful during the first 24 to 48 hours in patients with recurrent ischemia, hypertension, or heart failure (HF). However, nitrates should be avoided in patients with a right ventricular MI in whom the reduction in preload can lower the cardiac output. (See "Right ventricular myocardial infarction", section on 'Optimization of right ventricular preload'.)

Nitrate therapy after 48 hours is useful for the treatment of recurrent ischemia or HF as long as it does not preclude adequate therapy with agents proven to reduce mortality, such as beta blockers or angiotensin converting enzyme (ACE) inhibitors. (See "Nitrates in the management of acute coronary syndrome".)

Nitrates used for this purpose include isosorbide dinitrate, isosorbide mononitrate, or a transdermal nitroglycerin patch. (See "Nitrates in the management of chronic coronary syndrome", section on 'Nitrate tolerance'.)

Phosphodiesterase-5 inhibitors — The phosphodiesterase-5 inhibitors (sildenafil, vardenafil, and tadalafil) are contraindicated in patients taking nitrates because of the potential for

exaggerated vasodilation and hypotension and an increased risk of MI or death. (See "Sexual activity in patients with cardiovascular disease".)

Statin therapy — Intensive statin therapy should be initiated as early as possible in all patients with STEMI [1-5]. (See "Low-density lipoprotein-cholesterol (LDL-C) lowering after an acute coronary syndrome", section on 'Inpatient management'.)

Calcium channel blockers — Calcium channel blockers primarily serve as adjunctive therapy in patients with ongoing or recurrent symptoms of ischemia despite optimal therapy with beta blockers (with or without nitrates), in patients who are unable to tolerate adequate doses of one or both of these agents, or in patients with rapid atrial fibrillation when beta blockers are contraindicated.

No calcium channel blocker has been shown to reduce mortality in acute STEMI, and in certain patients they may be harmful, such as those with evidence of HF, left ventricular (LV) dysfunction, or atrioventricular block.

When a calcium channel blocker is used for the indications above, only diltiazem and verapamil are recommended in patients with STEMI. Immediate release nifedipine is contraindicated because of the reflex sympathetic activation, tachycardia, and hypotension associated with its use. (See "Major side effects and safety of calcium channel blockers", section on 'Possible increased mortality after acute myocardial infarction'.)

The second generation dihydropyridine calcium channel blockers, amlodipine and felodipine, appear to be safe in other forms of cardiovascular disease. Extrapolation from the results of these trials does not necessarily confirm that amlodipine or felodipine are safe in patients with an acute STEMI. Nonetheless, these agents, especially amlodipine, are often used when hypertension is not adequately controlled by other therapy of proven benefit (beta blockers and ACE inhibitors).

In-hospital glycemic control — The correction of hyperglycemia has become standard care for hospitalized patients, including those with acute MI. (See "Glycemic control in critically ill adult and pediatric patients".)

Venous thromboembolism prophylaxis — Prophylaxis for venous thromboembolism should be considered in medical patients who are immobilized. (See "Prevention of venous thromboembolic disease in acutely ill hospitalized medical adults".)

However, many patients with STEMI are now ambulatory within 24 hours, particularly if they have received reperfusion therapy and have had uncomplicated courses. These patients do not

require such prophylaxis. (See "Prevention of venous thromboembolic disease in acutely ill hospitalized medical adults", section on 'Summary and recommendations'.)

We suggest that in patients with uncomplicated STEMI, who will be at bed rest for less than 24 hours, venous thromboembolism prophylaxis is not necessary (unless indicated for some other reason). For STEMI patients in whom bed rest is anticipated for more than 24 hours and for whom anticoagulation is no longer required as part of the management of their acute coronary syndrome, we suggest low-dose unfractionated heparin (5000 units every 8 to 12 hours), low molecular weight heparin (eg, enoxaparin 40 mg/day), or fondaparinux 2.5 mg/day, all given subcutaneously, until the patient is ambulatory.

Red cell transfusion — As in the acute setting, red blood cell transfusion is generally reserved for severe or symptomatic anemia, such as hemoglobin <8 g/dL or hemoglobin 8 to 10 g/dL with hemodynamic instability or ongoing ischemia. Clinical judgment is required to determine if transfusion is likely to improve oxygen delivery or if there are other reasons to consider transfusion such as active bleeding or trauma. In deciding when to transfuse, individual patient characteristics such as the rate of blood loss and severity of myocardial ischemia need to be taken into account.

The rationale for using a restrictive transfusion strategy (limiting transfusions, transfusing for a lower rather than a higher hemoglobin level) includes avoiding risks of transfusion reactions or transfusion-transmitted infection and limiting burdens and costs while using evidence-based thresholds for optimal outcomes. While anemia correlates with worse outcomes, this is likely to be an association rather than causation. (See "Indications and hemoglobin thresholds for RBC transfusion in adults", section on 'Overview of our approach'.)

Supporting data for the thresholds used in acute MI are presented separately. (See "Indications and hemoglobin thresholds for RBC transfusion in adults", section on 'Acute MI'.)

Gastrointestinal prophylaxis — Patients with ACS who develop gastrointestinal bleeding after percutaneous coronary intervention have significantly worse outcomes. (See "Gastrointestinal bleeding in patients undergoing percutaneous coronary intervention", section on 'Outcomes'.)

The discussion of the primary prevention of gastrointestinal bleeding is found elsewhere. (See "Gastrointestinal bleeding in patients undergoing percutaneous coronary intervention", section on 'Prevention'.)

RECURRENT CHEST PAIN

Recurrent chest pain after acute STEMI is typically caused by either recurrent ischemia and reinfarction or by postinfarction pericarditis. Usually, recurrent chest pain within the first 12 hours of onset of MI is considered to be related to the original infarct and not evidence of reinfarction. Pericarditis is probably not responsible for significant chest discomfort in the first 24 hours.

A somewhat separate issue, chest pain or silent ischemia on stress testing before or soon after discharge, is discussed below. (See 'Stress testing' below.)

Recurrent ischemia and reinfarction — Among patients with an STEMI who have undergone apparently successful fibrinolysis by clinical criteria, early recurrence of ischemia (threatened reocclusion) has been observed in 20 to 30 percent of patients [6,7], thrombotic coronary reocclusion in 5 to 15 percent [8-10], and reinfarction in 3 to 5 percent [11-14]. Among patients treated with fibrinolysis, reinfarction occurs at a median of two to four days after therapy and is associated with increases in in-hospital and 30-day mortality compared to those patients without reinfarction [11,13]. As many patients who have undergone fibrinolysis are routinely referred for percutaneous coronary intervention (PCI), the frequency of recurrent ischemia after fibrinolysis is decreasing. (See "Percutaneous coronary intervention after fibrinolysis for acute ST-elevation myocardial infarction".)

Recurrent ischemia or reinfarction after primary PCI represents ischemia in a coronary distribution not revascularized or possible stent thrombosis.

Diagnosis — Within the first 18 hours of the initial MI, a recurrent elevation in cardiac biomarkers alone should **not** be relied upon to diagnose reinfarction, but should be accompanied by recurrent ST segment elevation on electrocardiogram and at least one other supporting criterion (such as recurrent chest pain or hemodynamic decompensation). For patients more than 18 hours from the initial MI, a biomarker rise and at least one additional criterion are sufficient for the diagnosis.

Management — Initial medical management of recurrent ischemia after STEMI should include escalation of therapy with beta blockers and nitrates to decrease myocardial oxygen demand and reduce ischemia. Intravenous anticoagulation should be initiated if it is not already being administered. For patients with hemodynamic instability, poor LV function, or a large area of myocardium at risk, insertion of an intraaortic balloon pump should also be considered.

PCI is the treatment of choice for patients with recurrent ischemia. Patients with recurrent ST elevation can also be treated or retreated with fibrinolytic therapy. However, an invasive strategy of angiography and revascularization is usually preferred. Patients should **not** be

retreated with streptokinase. (See "Diagnosis and management of failed fibrinolysis or threatened reocclusion in acute ST-elevation myocardial infarction".)

Infarction pericarditis — Acute pericarditis, generally defined as a pericardial friction rub with or without chest discomfort, may complicate the course of an acute MI. Chest pain is typically pleuritic. Pain in one or both trapezius ridges is especially consistent with the diagnosis. The evaluation and management of this complication are discussed separately. (See "Pericardial complications of myocardial infarction", section on 'Peri-infarction pericarditis'.)

PREPARING FOR DISCHARGE

Activity level in-hospital is determined by clinical status; most uncomplicated patients will be able to ambulate when the risk of bleeding from the arterial puncture site is low. Prior to discharge, the patient should undergo risk stratification, including noninvasive measurement of resting LV function and possibly exercise testing. (See 'Recurrent ischemia and reinfarction' above.)

Other components of predischarge planning include counseling about changes in behavior/lifestyle (smoking cessation, dietary improvement, and cardiac rehabilitation) and appropriate medical therapy to reduce the risk for further ischemic episodes. (See "Risk stratification after acute ST-elevation myocardial infarction".)

Early discharge after an uncomplicated MI — Early discharge (within three to five days) of a patient undergoing reperfusion who has had an uncomplicated acute STEMI (no angina or evidence of ischemia, bleeding, HF, or arrhythmia 72 hours after the event) is considered safe [15-18].

Risk stratification — Late risk stratification is performed before or sometimes after discharge. The main components are measurement of the LV ejection fraction (LVEF) and, in many patients, stress testing to detect possible residual ischemia. (See "Risk stratification after acute ST-elevation myocardial infarction".)

Evaluation of left ventricular function — Assessment of resting LV function is an important part of risk stratification in patients with acute MI and was given a strong recommendation in the 2013 American College of Cardiology Foundation/American Heart Association guideline for the management of STEMI [3,4]. Patients with LV systolic dysfunction have increased mortality at six months and one year (figure 1A-B) [19,20]. The increase in mortality is most pronounced in the 8 to 10 percent of patients with an LVEF ≤30 percent [19,20].

In the absence of a specific indication (eg, HF or suspected mechanical complication), the LVEF is usually measured before discharge. However, early measurements of LVEF may be misleading, since improvement in LVEF, beginning within three days and largely complete by 14 days, is common in patients who are reperfused, a presumed reflection, at least in part, of recovery from myocardial stunning [21,22].

Multiple imaging techniques for assessing LV function are available and each has equivalent prognostic value in the post-MI patient. In general, echocardiography should be used for routine assessment of LVEF after acute MI. It has the added advantage of detecting other factors that can be associated with a worse prognosis such as diastolic dysfunction, concurrent right ventricular dysfunction, left atrial enlargement, mitral regurgitation, and LV thrombus [23-27]. (See "Role of echocardiography in acute myocardial infarction" and "Left ventricular thrombus after acute myocardial infarction" and "Chronic secondary mitral regurgitation: General management and prognosis".)

Stress testing — A stress test, usually with exercise, is typically performed after STEMI to detect residual ischemia. Stress testing also permits assessment of the exercise capacity needed for the cardiac rehabilitation exercise prescription and can, in some patients, identify arrhythmias. While the prognostic value of stress testing has declined in the reperfusion era, such an assessment is standard practice in the United States and required of patients referred to a cardiac rehabilitation program. (See "Risk stratification after acute ST-elevation myocardial infarction", section on 'Stress testing'.)

Predischarge stress testing to detect residual ischemia is generally not performed in patients who have undergone percutaneous coronary intervention (PCI) or coronary artery bypass graft surgery and have been fully revascularized (eg, single vessel disease and successful PCI). Such patients often undergo exercise testing a few weeks or more after discharge as part of a cardiac rehabilitation program or for activity counseling.

Patients who have undergone partial revascularization or no revascularization, in contrast, are candidates for low level predischarge stress testing, which appears to be safe if:

- The patient has undergone in-hospital cardiac rehabilitation
- There have not been symptoms of HF or recurrent angina
- The electrocardiogram (ECG) has been stable for 48 to 72 hours prior to the exercise test

Stress testing should not be performed in patients with unstable postinfarction angina, decompensated HF, or life-threatening cardiac arrhythmias.

The appropriate stress protocol varies with physician preferences, patient characteristics, and local laboratory expertise. This is discussed in detail separately.

Accurate interpretation cannot be achieved in patients with baseline ECG abnormalities such as left bundle branch block, LV hypertrophy with ST-T wave changes, ventricular preexcitation, or ventricular pacing. In such patients, either exercise echocardiography or exercise radionuclide myocardial perfusion imaging can be performed. Pharmacologic stress is required in patients who cannot exercise, although such modalities provide less information than exercise testing because they are not able to assess functional capacity (algorithm 1). (See "Selecting the optimal cardiac stress test".)

For patients with symptomatic ischemia on post-infarct stress testing, we suggest the following approach:

- Coronary angiography followed by revascularization if appropriate is performed in patients with noninvasive evidence suggesting a moderate to large amount of myocardium at risk. Examples include ischemia during stage I or II of the Bruce protocol, a hypotensive blood pressure response or symptoms of pulmonary congestion, more than 2 mm of ST segment depression, or imaging studies suggesting involvement of either a large portion of the anterior wall or showing multiple areas of ischemia.

- Patients with only mild ischemia can be continued on medical therapy alone but patient preference may be important.

The management of silent ischemia detected on stress testing is similar and discussed separately. (See "Silent myocardial ischemia: Epidemiology, diagnosis, treatment, and prognosis", section on 'Revascularization'.)

Chronic anticoagulation — There is some evidence that initiating warfarin therapy following STEMI can reduce reinfarction and cerebrovascular accidents and may reduce mortality following acute MI. The discussion of the indications for chronic anticoagulation after MI is found elsewhere. (See "Acute coronary syndrome: Oral anticoagulation in medically treated patients".)

Ventricular arrhythmias — The role of implantable cardioverter defibrillators after MI is discussed separately. (See "Primary prevention of sudden cardiac death in patients with cardiomyopathy and heart failure with reduced LVEF".)

The management of late arrhythmias is discussed separately. (See "Ventricular arrhythmias during acute myocardial infarction: Incidence, mechanisms, and clinical features", section on

'Late arrhythmias'.)

Vaccination — We recommend influenza and pneumococcal vaccination as part of a comprehensive secondary prevention program in adults with coronary and other atherosclerotic vascular diseases. If indicated, we suggest vaccination for these during hospitalization for an acute coronary syndrome. (See "Pneumococcal vaccination in adults" and "Seasonal influenza vaccination in adults".)

Return to activities — It is important that patients be counseled about returning to prior activities. The majority of patients who remain asymptomatic after an uncomplicated acute MI can probably return to prior nonphysical activities, including work, safely within two weeks, although few data are available.

- Daily physical activity, such as walking, should be encouraged; the intensity of physical activity can be increased according to the cardiac rehabilitation plan.
- When sexual activity can be safely resumed is dependent upon a number of factors including whether or not the patient has been revascularized and, if not, if there is ischemia on stress testing (table 1). An important caveat is that phosphodiesterase-5 inhibitors (sildenafil, vardenafil, and tadalafil) cannot be taken within 24 to 48 hours of a nitrate. These issues are discussed in detail separately. (See "Sexual activity in patients with cardiovascular disease".)
- Driving can begin a week after discharge if the patient is judged to be in compliance with individual state laws.
- Since commercial aircrafts are pressurized to 7500 to 8000 feet, air travel should be undertaken only by stable patients (without a fear of flying) within the first two weeks and then only as long as they travel with companions, carry sublingual nitroglycerin, and request airport transportation to avoid rushing. (See "Approach to patients with heart disease who wish to travel by air or to high altitude", section on 'Air travel'.)

For patients with a complicated MI, return to usual activities should be delayed by two to three weeks.

DISCHARGE MEDICATIONS

Appropriate medical therapy can reduce the risk of subsequent ischemic events and mortality in patients who have had an acute MI. In addition to the interventions discussed in this section,

beta blockers, statins, nitrates, and calcium channel blockers may be useful long term and are discussed above. (See 'Further medical therapy' above.)

Medications upon discharge from the hospital should include those important for risk reduction, including antiplatelet drugs, a beta blocker, a statin, and possibly an angiotensin converting enzyme (ACE) inhibitor or an angiotensin receptor blocker (ARB).

Antiplatelet drugs — In the absence of an absolute contraindication, aspirin is recommended indefinitely in all patients who have had an acute MI. (See "Aspirin for the secondary prevention of atherosclerotic cardiovascular disease".)

Treatment with a $P2Y_{12}$ receptor blocker is indicated in all patients after acute STEMI for at least one year, regardless of the reperfusion strategy. The specific recommendations for the duration of therapy after MI or stent placement are found elsewhere. (See "Acute ST-elevation myocardial infarction: Antiplatelet therapy" and "Long-term antiplatelet therapy after coronary artery stenting in stable patients", section on 'Summary and recommendations'.)

Angiotensin inhibition — We start an ACE inhibitor in all STEMI patients prior to discharge. ACE inhibitors lead to improved 30-day mortality after acute STEMI, particularly in patients with an anterior infarct or reduced LV function, diabetes, or stable chronic kidney disease. An ARB is an alternative for patients unable to tolerate an ACE inhibitor (usually due to cough). (See "Angiotensin converting enzyme inhibitors and receptor blockers in acute myocardial infarction: Recommendations for use".)

Aldosterone antagonists — For patients with STEMI who are receiving an ACE inhibitor and a beta blocker, and who have a LVEF less than or equal to 40 percent and either HF or diabetes, we prescribe a mineralocorticoid receptor antagonist, also referred to as an aldosterone blocker or antagonist [3,4]. Therapy should be begun before discharge, since a mortality benefit is seen within 30 days [28]. The serum potassium should be monitored closely during treatment. Although uncommon, life-threatening hyperkalemia can occur due to the combination of aldosterone inhibition, reduced aldosterone secretion associated with ACE inhibitor therapy, and a progressive decline in renal perfusion induced due to HF. Older adult patients with renal insufficiency are at greatest risk. The use of aldosterone antagonists is discussed in detail elsewhere. (See "Primary pharmacologic therapy for heart failure with reduced ejection fraction", section on 'Mineralocorticoid receptor antagonist'.)

Although a 2018 meta-analysis found benefit to the addition of aldosterone antagonist in patients with STEMI without HF and with an ejection of more than 40 percent, we believe significant limitations to this study prevent us from recommending this therapy for these patients [29,30].

RISK FACTOR MODIFICATION

Risk factor modification using behavioral/lifestyle changes such as dietary modification, increase in activity level, and smoking cessation are associated with better outcomes after acute coronary syndromes (ACS). Patient education should be given at the time of discharge and referral to a cardiac rehabilitation program should be made. (See "Prevention of cardiovascular disease events in those with established disease (secondary prevention) or at very high risk", section on 'Lifestyle modifications' and 'Cardiac rehabilitation' below.)

Smoking cessation — Smoking cessation reduces the risk of recurrent MI. In one study of 564 postinfarction patients, for example, the risk of recurrent infarction fell by 50 percent within one year of smoking cessation and normalized to that of nonsmokers within two years [31]. (See "Cardiovascular risk of smoking and benefits of smoking cessation".)

We recommend education about smoking cessation during hospitalization and referral to a smoking cessation program or outpatient cardiac rehabilitation program. (See "Pharmacotherapy for smoking cessation in adults", section on 'Nicotine replacement therapy'.)

However, the issue of whether nicotine replacement can be started during hospitalization has not been well studied. The safety of this approach was evaluated in a retrospective analysis of 194 smokers admitted with an ACS who received transdermal nicotine replacement therapy during their hospitalization [32]. Compared to a propensity-matched cohort, there was no difference in mortality at 7 and 30 days and at one year in patients started on nicotine replacement in-hospital.

We suggest cautious initiation of nicotine replacement during hospitalization for those patients with ACS in whom the benefits appear to outweigh the risks.

Long-term glycemic control — We recommend a goal HbA1c of less than 7 percent, which is consistent with the general treatment goal for patients with type 2 diabetes. The supporting evidence and treatment goals are discussed separately. (See "Initial management of hyperglycemia in adults with type 2 diabetes mellitus", section on 'Glycemic management' and "Prevention of cardiovascular disease events in those with established disease (secondary prevention) or at very high risk".)

Thiazolidinediones have been used in the treatment of patients with diabetes mellitus. A concern has been raised about the use of rosiglitazone in patients with coronary artery disease. (See "Thiazolidinediones in the treatment of type 2 diabetes mellitus", section on 'Cardiovascular effects'.)

Hypertension — Hypertension should be treated in patients who have had an MI. The optimal goal blood pressure and choice of antihypertensive drugs in such patients are discussed in detail separately. (See "Choice of drug therapy in primary (essential) hypertension" and "Goal blood pressure in adults with hypertension".)

Diet — Good dietary habits and use of a prudent diet can reduce the risk of coronary heart disease. (See "Healthy diet in adults".)

Exercise — An increase in activity level in patients with established cardiovascular disease is associated with better outcomes. This topic is discussed in detail separately. (See "Exercise and fitness in the prevention of atherosclerotic cardiovascular disease".) An in-hospital assessment of exercise capacity should be made (phase I) and followed by transition into an outpatient phase II cardiac rehabilitation program. (See "Cardiac rehabilitation: Indications, efficacy, and safety in patients with coronary heart disease".)

Stress management — Psychosocial stress factors are associated with a poor prognosis after acute MI, and reducing emotional stress may improve outcomes. (See "Psychosocial factors in acute coronary syndrome".)

Cardiac rehabilitation — Multifactorial cardiac rehabilitation, which includes efforts to address all of the above issues, can produce significant long-term reductions in both total and cardiovascular mortality. All patients should be considered for referral to a cardiac rehabilitation facility. (See "Cardiac rehabilitation: Indications, efficacy, and safety in patients with coronary heart disease".)

THERAPIES OF UNCLEAR BENEFIT

Colchicine — We do not routinely treat patients with acute MI with colchicine. Trials that evaluated the efficacy of colchicine arrived at different conclusions:

- In the COLCOT trial, 4745 patients with MI within 30 days and receiving optimal medical therapy were randomly assigned to colchicine 0.5 mg daily or placebo [33]. Treatment began at a median of 14 days after ACS. The risk of the primary composite endpoint (death from cardiovascular causes, resuscitated cardiac arrest, MI, stroke, or urgent hospitalization for angina leading to coronary revascularization) was lower in the colchicine group (hazard ratio [HR] 0.77, 95% CI 0.61-0.96) after a median follow-up of nearly two years. This result was driven principally by lower risks of angina and stroke. Adverse events were generally similar in the two groups. A subsequent analysis of the impact of time-to-treatment initiation found that earlier (between days 0 and 3 after

randomization) but not later initiation was associated with benefit [34]. Limitations of this study include the absence of improvement in hard endpoints such as cardiovascular death or MI and a relatively high discontinuation rate (about 18.5 percent in both groups).

- The Australian COPS trial randomly assigned 795 patients with ACS to colchicine (0.5 mg twice daily for one month, then 0.5 mg daily for 11 months) or placebo during the index hospitalization [35]. Over 12 months of follow-up, there was no difference in the rate of the primary composite outcome (all-cause death, ACS, ischemia-driven urgent revascularization, and noncardioembolic ischemic stroke) in the two groups (24 and 38 events, respectively; 6.1 versus 9.5 percent; HR 0.65, 95% CI 0.38-1.09). There were more all-cause deaths in the colchicine group (eight versus one). The results of this trial need to be interpreted in view of its relatively small size.

The role of colchicine in chronic coronary disease is discussed separately. (See "Prevention of cardiovascular disease events in those with established disease (secondary prevention) or at very high risk", section on 'Colchicine'.)

Hematopoietic stem cell therapy — Hematopoietic stem cell therapy remains investigational; trials have not established the efficacy of stem cell therapies in patients with ACS [36].

PROGNOSIS

Patients presenting with a non-ST-elevation MI have a lower in-hospital mortality than those with a STEMI, but a similar or perhaps worse long-term outcome. This topic is discussed in detail separately. (See "Prognosis after myocardial infarction".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: ST-elevation myocardial infarction (STEMI)".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, “The Basics” and “Beyond the Basics.” The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given

condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on “patient info” and the keyword(s) of interest.)

- Basics topic (see "Patient education: Heart attack (The Basics)" and "Patient education: Heart attack recovery (The Basics)" and "Patient education: Medicines after a heart attack (The Basics)" and "Patient education: Coping with high drug prices (The Basics)")
- Beyond the Basics topic (see "Patient education: Heart attack (Beyond the Basics)" and "Patient education: Heart attack recovery (Beyond the Basics)" and "Patient education: Coping with high prescription drug prices in the United States (Beyond the Basics)")

SUMMARY

- **Acute management** – In patients with ST-elevation myocardial infarction (STEMI), early diagnostic and therapeutic interventions, such as antiplatelet therapy and emergency reperfusion, are associated with improved early outcomes. (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department" and "Overview of the acute management of ST-elevation myocardial infarction".)
- **Nonacute management** – After the patient has been stabilized and early interventions have been carried out, the nonacute management of STEMI typically includes:
  - Beta blockers. (See 'Oral beta blockers' above.)
  - Long-term dual antiplatelet therapy with aspirin and a $P2Y_{12}$ receptor blocker. (See 'Antiplatelet drugs' above.)
  - Statins (high-intensity). (See 'Statin therapy' above.)
  - Aggressive management of recurrent myocardial ischemia or infarction. (See 'Recurrent chest pain' above.)

- Angiotensin converting enzyme inhibitors in patients with diabetes, heart failure (HF), a left ventricular ejection fraction (LVEF) <40 percent, and hypertension. (See 'Angiotensin inhibition' above.)
- Risk factor modification of factors that include smoking, hypertension, diabetes, and dyslipidemia. (See 'Risk factor modification' above.)
- Assessment of LV function. (See 'Evaluation of left ventricular function' above.)
- Long-term risk stratification. (See 'Risk stratification' above.)

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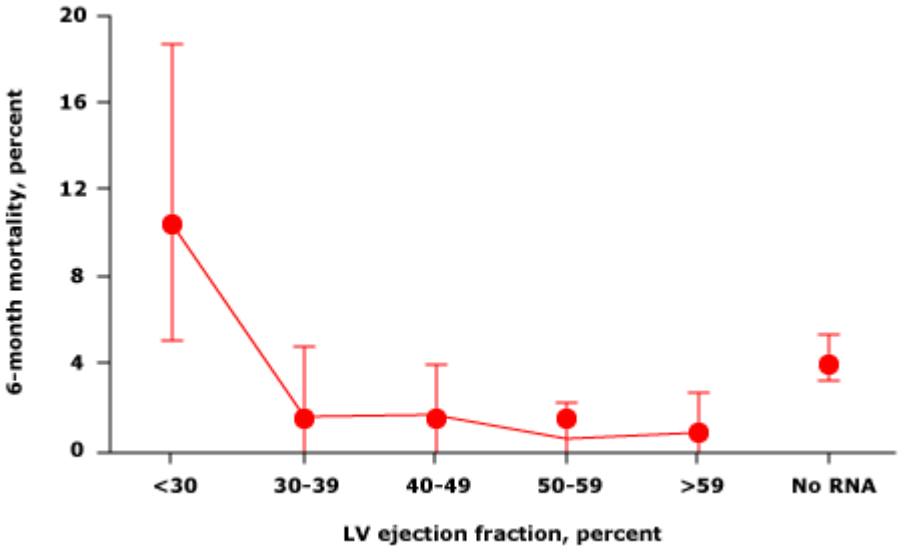
Topic 67 Version 76.0

GRAPHICS

LVEF predicts six-month mortality after STEMI

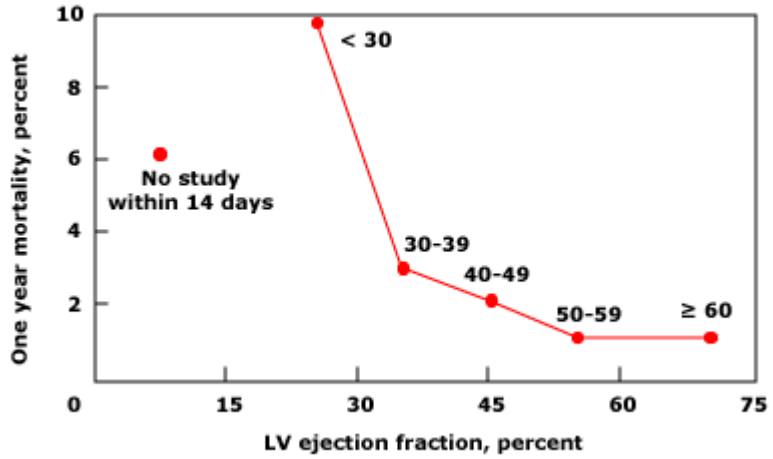


In a study of 1194 patients with an acute ST segment elevation myocardial infarction (STEMI) treated with thrombolysis, the resting left ventricular ejection fraction (LVEF) measured by radionuclide angiography (RNA) was a powerful predictor of six-month mortality. The highest risk group was those with an LVEF <30 percent.

Data from Burns RJ, Gibbons RJ, Yi Q, et al. The relationships of left ventricular ejection fraction, end-systolic volume index and infarct size to six-month mortality after hospital discharge following myocardial infarction treated by thrombolysis. J Am Coll Cardiol 2002; 39:30.

Graphic 75743 Version 5.0

LVEF predicts one-year mortality after STEMI

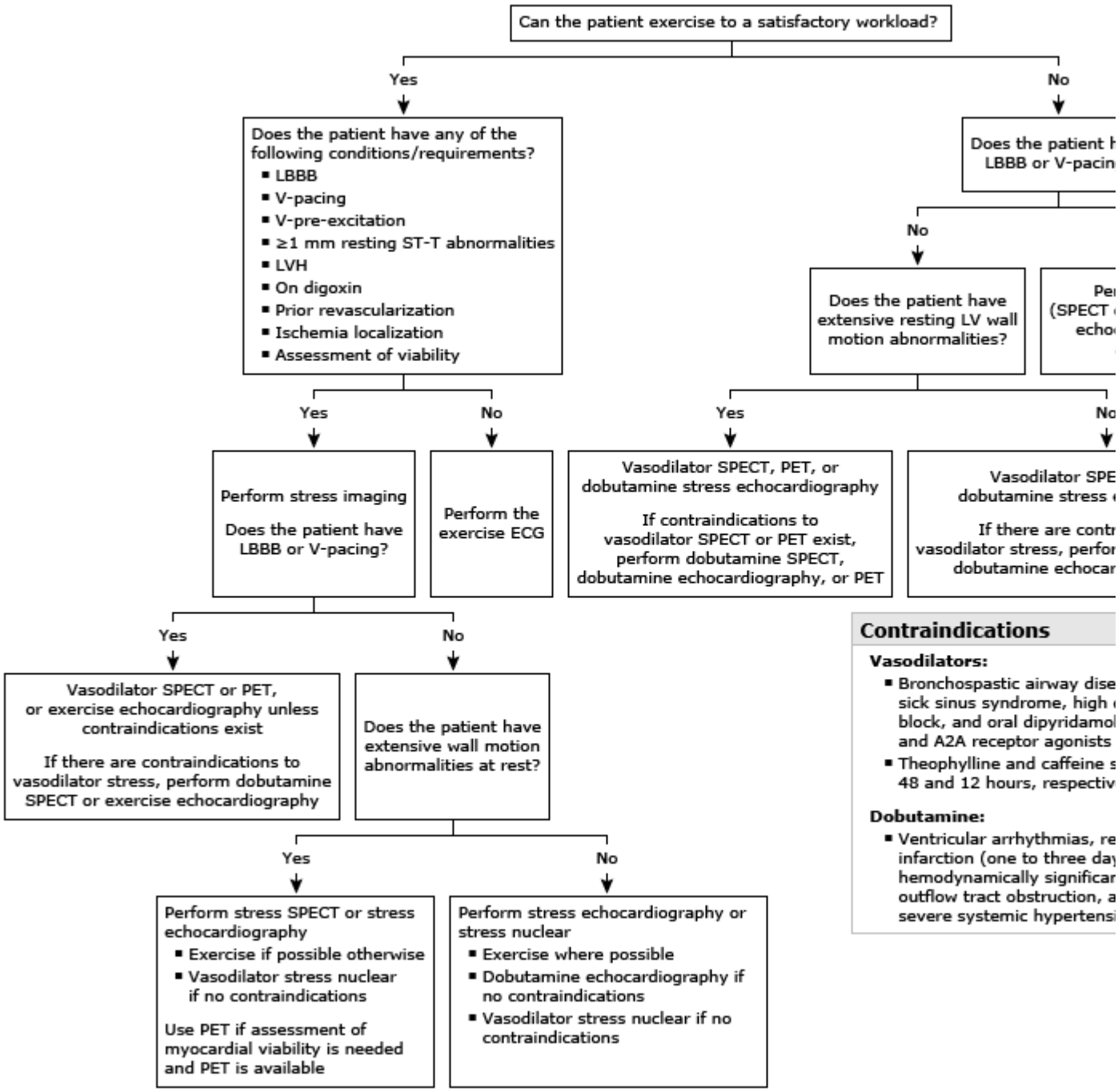


Among 3197 patients with an acute ST elevation myocardial infarction (STEMI) who were treated with thrombolytic therapy in the TIMI II trial, the one-year mortality correlated with the resting left ventricular ejection fraction (LVEF) measured by radionuclide ventriculography obtained within 14 days of the event. The mortality was highest in patients with an LVEF <30 percent (9.9 percent). Patients not undergoing study for any reason had an intermediate mortality of 6.6 percent.

Data from Zaret BL, Wackers FJ, Terrin ML, et al. J Am Coll Cardiol 1995; 26:73.

Graphic 68071 Version 3.0

Algorithm for choosing the optimal cardiac stress test



LBBB: left bundle branch block; V: ventricular; LVH: left ventricular hypertrophy; LV: left ventricular; SPECT: single photon emission computed tomography; PET: positron emission tomography; ECG: electrocardiogram.

Graphic 87155 Version 5.0

Second Princeton consensus panel recommendations for risk assignment for sexual activity and management of sexual dysfunction

Grade of risk	Criteria
CAVEATS	Phosphodiesterase-5 inhibitors contraindicated with any nitrate preparation should not be taken with alpha-blocking agents
	Nitrates should not be administered within 24 hours (or longer in patients with renal or hepatic dysfunction) of sildenafil, 24 hours of vardenafil, or up to 48 hours of tadalafil
Low risk	No symptoms and less than three major cardiovascular risk factors (excluding gender)
	Controlled hypertension
	Mild, stable angina
	Post-successful coronary revascularization
	Uncomplicated MI more than six to eight weeks previously in patients who do not have exercise-induced ischemia or who have undergone coronary revascularization
	Mild valvular disease
	Asymptomatic LV dysfunction
	Management
	Patients at low risk can safely initiate or resume sexual activity and can be treated for sexual dysfunction
Intermediate or indeterminate risk	No symptoms and three or more major cardiovascular risk factors (excluding gender)
	Moderate, stable angina
	Recent MI (more than two but less than six weeks previously); in patients who have not undergone revascularization, the risk can be assessed with stress testing
	Asymptomatic LV dysfunction with LV ejection fraction <40 percent or NYHA class II heart failure
	Noncardiac sequelae of atherosclerotic disease, such as peripheral vascular disease or prior stroke or transient ischemic attack
	Management
	Patients at intermediate or indeterminate risk should undergo further cardiologic evaluation, such as stress testing (particularly in patients with a sedentary lifestyle), in an attempt to restratify the patient into the high risk or low risk category
High risk	Unstable or refractory angina

	Uncontrolled hypertension
	NYHA class III/IV heart failure
	MI within the past two weeks
	High-risk arrhythmias
	Obstructive hypertrophic cardiomyopathy
	Moderate to severe valvular disease, particular aortic stenosis
	Management
	Patients at high risk should be stabilized by appropriate therapy before resumin sexual activity

MI: myocardial infarction; LV: left ventricular; NYHA: New York Heart Association.

Adapted from Kostis B, Jackson G, Rosen R, et al. Am J Cardiol 2005; 96:313.

Graphic 71511 Version 4.0