Treatment

Acute STEMI is caused by occlusion of the epicardial coronary artery by thrombus after rupture of a vulnerable plaque. The process of myocardial necrosis is time dependent, so diagnosis and treatment of STEMI to preserve myocardium must occur as quickly as possible. More than half of deaths occur within 1 hour after onset of symptoms, before the patient can be reached for emergency care. Patients often delay seeking care for symptoms of acute MI despite efforts to alert the public to the risk of ignoring symptoms of chest discomfort. Emergency medical personnel who respond to patients with possible MI begin to institute initial therapy in the field. Patients are monitored with ECG for rhythm disturbances such as ventricular tachycardia (VT) or ventricular fibrillation (VF) that require prompt cardioversion or defibrillation. Oxygen is administered via nasal cannula, and intravenous access is established. Aspirin (162 to 325 mg) is administered to the patient, and sublingual nitroglycerin may also be given in attempt to relieve chest discomfort. Some emergency response systems perform 12-lead ECGs and telemeter the results to the emergency department, allowing for early diagnosis of STEMI and early decision making regarding revascularization strategies.

Once the patient arrives in the emergency department, an ECG, if not already available, will be preformed within 5 minutes. If the ECG is nondiagnostic, a second study is obtained no more than 20 minutes after presentation. A diagnosis of STEMI triggers decision making regarding reperfusion strategies that are used by the particular institution (see Chapter 73, "ST Elevation Acute Myocardial Infarction and Complications of Myocardial Infarction," in *Goldman-Cecil Medicine,* 25th Edition). Hospitals that are capable of performing emergency cardiac catheterization for the purpose of reperfusion therapy have an established rapid response system to activate the catheterization laboratory for this urgent therapy. There is evidence that primary PCI therapy for STEMI is superior to fibrinolytic therapy, but its use depends on the timely availability of a well-trained catheterization team. The quality of primary PCI is signified by a so-called door-to-balloon time of less than 90 minutes. Likewise, the standard for fibrinolytic therapy is a door-to-needle time of less than 30 minutes. Regardless of the means of reperfusion, it is important for the hospital treating patients with STEMI to have a structured protocol for timely diagnosis, decision making, and initiation of therapy.

In addition to aspirin, the patient should be given a loading dose of thienopyridine (clopidogrel 600 mg or prasugrel 60 mg), assuming he or she will be treated with primary PCI. Unfractionated heparin in a dose of 60 IU/kg should be administered (no more than 4000 IU bolus) with a drip rate of 12 IU/kg/hour (maximum dose, 1000 IU/hour). LMW heparin may also be used (enoxaparin 30 mg IV bolus with 1 mg/kg subcutaneously every 12 hours for patients younger than 75 years of age who have normal renal function). Other agents such as glycoprotein IIb/IIIa inhibitors or bivalirudin are administered depending on the protocols of the catheterization laboratory.

Intravenous morphine (2 to 4 mg, repeated every 5 to 15 minutes as needed) is frequently used for pain control. Patients also are commonly given sublingual nitroglycerin 0.4 mg (repeat every 5 minutes for no more than three total doses), which may help to diminish chest discomfort. Intravenous nitroglycerin may be helpful for control of both pain and hypertension if present. Intravenous β-blockers such as metoprolol (5-mg bolus every 10 minutes for a total dose of 15 mg) is indicated in the treatment of STEMI, but it should be avoided in the face of heart failure, severe COPD, hypotension, or bradycardia. β-Blockers (metoprolol, propranolol, atenolol, timolol, and carvedilol) have been shown to significantly reduce the risk of future MI and cardiovascular mortality. Statin therapy, as mentioned for NSTEMI, is recommended for all patients with STEMI as a presenting symptom regardless of their history of hypercholesterolemia. Other adjunctive measures include bedrest for the first 12 hours, ongoing oxygen by nasal cannula with pulse oximeter monitoring, continuous rhythm monitoring, anxiolytic agents as needed, and stool softeners. Atropine is kept in reserve for the treatment of hemodynamically significant bradycardia, which may occur with inferior MI.

ACE-inhibitor therapy also plays an important role in the long-term survival of patients after STEMI. ACE-inhibitor therapy has been shown to reduce the incidence of heart failure, recurrent MI, and long-term mortality after STEMI. ACE inhibitors commonly used for this purpose include lisinopril, captopril, enalapril, and ramipril. The decision to initiate ACE-inhibitor therapy is directed by the patient's tolerance. Care is warranted early after STEMI, because the patient may be prone to hypotension related to ACE-inhibitor therapy. A low dose should be administered first, with gradual upward titration.

Aldosterone receptor blockade with eplerenone (25 to 50 mg/day) reduces cardiovascular mortality after MI in patients with heart failure and a reduced EF of less than 40% or diabetes. Spironolactone also reduces mortality in patients with heart failure and a history of remote MI.

Reperfusion Therapy

Timely reperfusion therapy, either thrombolytic therapy or primary PCI, is critical to limiting the extent of MI and reducing the risks of future morbidity and mortality. Primary PCI has been shown to have advantages over thrombolytic therapy, with higher immediate and long-term vessel patency. Primary PCI depends on the availability of cardiac catheterization facilities and staff to conduct the reperfusion procedure quickly (see earlier discussion). If the patient has not had access to a catheterization facility for longer than 2 hours after presentation, thrombolytic therapy is a reasonable alternative.

In the randomized, placebo-controlled Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto (GISSI) study, thrombolytic therapy with intravenous streptokinase was shown to reduce the risk of mortality in patients with STEMI if it was administered early after presentation. The time-dependent nature of therapy was also demonstrated, in that patients treated more than 12 hours after the onset of symptoms had no measurable benefit from thrombolysis. The next generation of thrombolytic agents, recombinant tissue-type plasminogen activators (rt-PA), improved on mortality reduction when compared with streptokinase (30-day mortality rate, 7.3% with streptokinase vs. 6.3% with rt-PA). The advantage of rt-PA appeared to be related to enhanced vessel patency at 90 minutes after administration (80% with rt-PA vs. 53% to 60% with streptokinase). Subsequent forms