of care for acute MI have greatly reduced the risk of post-MI thromboembolism.

Reperfusion therapy, when applied in a timely fashion, results in less extensive MI and less impairment of LV function. Patients with anterior MI treated with reperfusion therapy are less likely to have extensive apical akinesis, which is the breeding ground for mural thrombus. It is advised that patients treated for acute MI have an echocardiogram to assess for overall LV function; in the case of anterior MI, the presence of apical mural thrombus can be detected by echocardiography. If LV mural thrombus is present, the patient should receive therapeutic anticoagulation with unfractionated or LMW heparin while oral anticoagulation with warfarin is initiated. Warfarin therapy should be continued for 6 months after MI when LV apical mural thrombus is detected. Early ambulation after MI, along with the use of compression stockings and subcutaneous heparin prophylaxis (unfractionated or LMW) for deep venous thrombosis, has greatly diminished the threat of pulmonary embolism.

PROGNOSIS

Risk Stratification after Myocardial Infarction

Key to understanding an individual patient's risk for future coronary events or mortality related to MI is a thorough assessment of drivers for those risks: status of LV function and its impact on clinical functional status, residual myocardial ischemia, and spontaneous or exercise-induced arrhythmias. Appropriate predischarge assessments provide a comprehensive picture of the patient's risk status and prognosis.

Electrocardiographic Monitoring

Patients are routinely monitored by telemetry systems that capture arrhythmic events in the first 48 hours after MI. Late ventricular arrhythmias such as VF or sustained VT identify patients who are likely to benefit from ICD therapy. This is particularly true if EF is reduced to less than 40%. ICD implantation is also indicated for patients with persistently reduced EF (<30%).

Cardiac Catheterization and Noninvasive Testing

Predischarge risk stratification may involve cardiac catheterization, submaximal predischarge exercise stress testing (on days 4 to 6), or maximal exercise stress testing after discharge (at 2 to 6 weeks). The presence or absence of high-risk coronary anatomy is demonstrated for patients who have undergone primary PCI at the time presentation. Many patients who have been treated with thrombolytic therapy undergo coronary angiography before discharge to determine the extent and severity of underlying CAD as well as the status of the culprit lesion. If coronary angiography is not performed, predischarge submaximal exercise testing (up to 70% of maximal predicted heart rate) is done to identify those who are at increased risk for postdischarge coronary ischemic events. Patients who undergo submaximal exercise stress testing in lieu of coronary angiography frequently have a follow-up maximal exercise stress test within 2 to 6 weeks after discharge. During stress testing, positive results that suggest the need for coronary angiography include exercise-induced angina, ST changes of ischemia (ST depression), exercise-induced hypotension, exercise-induced ventricular arrhythmias, and low functional capacity. The sensitivity and specificity of stress testing after MI is enhanced by the use of imaging modalities such as stress echocardiography or nuclear perfusion imaging. All patients should have their EF assessed, typically by echocardiography, before discharge.

Secondary Prevention, Patient Education, and Rehabilitation

The goal of secondary prevention is to reduce the risk of recurrent MI and cardiovascular mortality. Risk factor modification is key to the secondary prevention strategy. All patients should have their lipid status assessed at the time of admission, but statin therapy is warranted in patients with acute MI at presentation. The target LDL level is less than 100 mg/dL, preferably closer to 70 mg/dL. Smoking cessation is of critical importance because it can reduce the risk of reinfarction, and ongoing smoking can double the risk of recurrent MI or mortality in the first year after MI. Structured smoking cessation programs and the use of pharmacologic aids (e.g., nicotine patches or gum, bupropion, varenicline) can increase the success of smoking cessation efforts.

Antiplatelet therapy with aspirin (75 to 162 mg/day) is given indefinitely to all patients after MI. Regardless of whether primary PCI has been performed, patients will benefit from the use of clopidogrel 75 mg/day for the first year after MI. Those patients who have received a stent during primary PCI should continue either clopidogrel 75 mg/day or prasugrel 10 mg/day for a duration appropriate for the type of stent employed. At the least, patients should receive dual antiplatelet therapy (aspirin + thienopyridine) for 1 month for a bare metal stent, 3 months for a sirolimus-based stent, and 6 months for a paclitaxel-eluting stent. Most patients who have been treated with any type of stent receive dual antiplatelet therapy for the first year after their MI. The use of antiplatelet therapy in any form should be tempered by an individual patient's risk of hemorrhagic complications.

The use of warfarin anticoagulation (target international normalized ratio [INR], 2.0 to 3.0) is indicated for patients with persistent or paroxysmal AF, guided by their CHADS-2 score (congestive heart failure, hypertension, age ≥75 years, diabetes mellitus, and stroke). Patients who have experienced pulmonary or systemic thromboembolism also warrant warfarin therapy. Patients who are at high risk for thromboembolism after acute MI, such as those with low EF related to anterior MI, should also be considered for warfarin. The concomitant use of dual antiplatelet therapy along with warfarin requires careful monitoring for bleeding complications.

Acute anterior MI that has resulted in significant injury to the ventricle with an EF of less than 40% places the patient at risk for future negative remodeling of the left ventricle and potential heart failure. ACE-inhibitor therapy has been shown to reduce the risk of negative remodeling and the occurrence of heart failure in such patients. This group of patients also experiences a reduction in future recurrent MI risk with the use of ACE-inhibitor therapy. This observation does not appear to carry over to patients with stable CAD. ACE-inhibitor therapy (captopril, ramipril, lisinopril) is indicated for all patients after MI. The use