Occasionally, patients only have symptoms in the non-chest areas usually associated with radiation. Up to 20% of patients, particularly the elderly and diabetics, do not have typical chest discomfort at presentation. The index of suspicion for acute MI should be high in these groups if the patient exhibits profound weakness, acute dyspnea or pulmonary edema, nausea, vomiting, ventricular arrhythmias, or hypotension. The differential diagnosis for patients with chest discomfort suspicious for acute MI includes aortic dissection, pulmonary embolism, chest wall pain, esophageal reflux, acute pericarditis, pleuritis, and panic attacks. Given the life-threatening nature of aortic dissection and pulmonary embolism, these diagnoses should always be paramount, along with acute MI, in patients presenting with chest discomfort.

Physical Examination

A comprehensive examination should be undertaken if acute MI is suspected. Attention must be paid to vital signs, because patients may be either hypertensive or hypotensive during the course of an MI. In some cases, such as inferior MI, profound bradycardia may be present. Auscultation of the heart may reveal an S_4. In the case of a large MI, the patient may have symptoms and signs of heart failure such as dyspnea, rales, elevated central venous pressure, and an S_3. Severe heart failure may lead to cardiogenic shock with hypotension and vasoconstriction causing the extremities to be cool to touch. Patients with acute MI are also subject to mechanical problems such as mitral regurgitation due to papillary muscle dysfunction.

Electrocardiogram

The ECG is an important tool in the diagnosis of acute MI. ST elevation of 1 mm or greater in contiguous leads is seen in most patients with acute MI. The initial ECG may be nondiagnostic, so it is important to obtain serial tracings no more than 20 minutes apart to detect the evolutionary changes characteristic of STEMI. The first stage of ECG presentation is ST elevation that subtends the region of the heart affected by transmural ischemia. ST depression may be present in opposing leads, and these are termed *reciprocal changes* (see Chapter 73, "ST Elevation Acute Myocardial Infarction and Complications of Myocardial Infarction," in *Goldman-Cecil Medicine,* 25th Edition). The presence of reciprocal changes may indicate a larger and more threatening MI. As the MI progresses, ST elevation gives way to T wave inversion. Varying degrees of resolution of ST and T wave changes occur over time, but patients with transmural MI develop pathologic Q waves in the leads subtending the infarcted muscle. Other causes of ST elevation include pericarditis and a chronic repolarization finding of "early repolarization." The presence of either cause of ST elevation can confound the early ECG diagnosis of acute MI.

Approximately 30% of acute MIs originate from the circumflex coronary artery on the posterior wall of the heart. This type of MI appears on the ECG as precordial ST depression. The presence of precordial ST depression should raise suspicion of the presence of "true posterior MI," and additional leads placed through the axilla to the back may reveal the presence of posterior ST elevation. Echocardiography demonstrating posterior hypokinesis is also useful in discriminating true posterior MI. Acute inferior MI due to occlusion of the right coronary artery can also be associated with right ventricular infarction if the right coronary artery's acute marginal branch is compromised. Right ventricular infarction can lead to some challenging management issues, and its diagnosis is aided by the use of right precordial leads to detect ST elevation.

LBBB can mask ST elevation due to acute MI. Patients with clinical features of acute MI who have an LBBB (particularly a new LBBB) should be presumed to have STEMI and treated appropriately. Right bundle branch block (RBBB) does not mask the ST elevation of STEMI.

Differential Diagnosis

The diagnosis of STEMI is usually straightforward based on symptoms and ECG findings, but a number of conditions can mimic the ST elevation of STEMI and confound the diagnosis. The ECG changes of early repolarization, Takotsubo's syndrome, acute myocarditis, or pericarditis can be difficult or impossible to distinguish from those of STEMI. In the face of ST elevation and chest discomfort, it may be necessary to perform coronary angiography in patients who ultimately are diagnosed with a condition other than STEMI so as to not miss this critical diagnosis.

Diagnostic Testing

Cardiac troponins (cTnI and cTnT) are sarcomere proteins that, when measured in blood, are specific for myocardial injury. The troponin level becomes elevated 2 to 4 hours after the onset of injury, and the abnormal elevation can persist for up to 2 weeks after the event. The CK-MB isomere is not as specific for heart injury as troponin, but it can still be useful in documenting the presence of MI. CK-MB is found elevated within 4 hours after an acute MI, but it clears more rapidly than troponin. In the case of persistently elevated troponin, a measurable increase in CK-MB may herald another episode of myocardial necrosis. Chronic renal insufficiency is associated with false-positive elevations of troponin T, more so than troponin I. In addition to biomarkers of myocardial injury, other laboratory studies obtained in patients with acute MI include a complete blood count, blood chemistries, lipid panel, prothrombin time (PT), and partial thromboplastin time (PTT). Leukocytosis is a common finding in acute MI, reflecting the inflammatory nature of myocardial necrosis.

At the time of admission, chest radiographs are obtained to assess for the presence of pulmonary edema or mediastinal widening suspicious for dissection. Echocardiography is important in delineating the extent of MI and assessing EF. In cases of diagnostic ambiguity, early use of echocardiography can demonstrate the presence of regional wall motion abnormalities consistent with acute MI. Echocardiography with color Doppler is also helpful in diagnosing complications of acute MI such as infarct-related mitral regurgitation or ventricular septal defect (VSD), pericardial effusion, or evidence of pseudoaneurysm as a result of myocardial rupture. Follow-up echocardiography in the months after acute MI can also reveal recovery of LV function. Radionuclide tracer studies are not useful in diagnosing acute MI. CT, cardiac MRI, and transesophageal echocardiography are all useful in diagnosing aortic dissection when there is an increased index of suspicion. Cardiac MRI can also distinguish myopericarditis.