Acute Coronary Syndrome and Myocardial Infarction

ACS is an emergent situation characterized by an acute onset of myocardial ischemia that results in myocardial death (ie, MI) if definitive interventions do not occur promptly. (Although the terms coronary occlusion, heart attack, and MI are used synonymously, the preferred term is MI.) The spectrum of ACS includes unstable angina, non–ST-segment elevation MI (NSTEMI), and ST-segment elevation MI (STEMI).

Pathophysiology

In unstable angina, there is reduced blood flow in a coronary artery, often due to rupture of an atherosclerotic plaque, but the artery is not completely occluded. This is an acute situation that is sometimes referred to as preinfarction angina because the patient will likely have an MI if prompt interventions do not occur.

In an MI, an area of the myocardium is permanently destroyed, typically because plaque rupture and subsequent thrombus formation result in complete occlusion of the artery. Vasospasm (sudden constriction or narrowing) of a coronary artery, decreased oxygen supply (eg, from acute blood loss, anemia, or low blood pressure), and increased demand for oxygen (eg, from a rapid heart rate, thyrotoxicosis, or ingestion of cocaine) are other causes of MI. In each case, a profound imbalance exists between myocardial oxygen supply and demand.

The area of infarction develops over minutes to hours. As the cells are deprived of oxygen, ischemia develops, cellular injury occurs, and the lack of oxygen results in infarction, or the death of cells. The expression “time is muscle” reflects the urgency of appropriate treatment to improve patient outcomes. Each year in the United States, nearly 1 million people have acute MIs; one fourth of these people die as a result (AHA, 2007). Half of those who die never reach a hospital.

Various descriptions are used to further identify an MI: the type (NSTEMI, STEMI), the location of the injury to the ventricular wall (anterior, inferior, posterior, or lateral wall), and the point in time within the process of infarction (acute, evolving, or old). The differences between NSTEMI and STEMI are diagnostic and are explained later in this chapter.

The ECG usually identifies the type and location of the MI, and other ECG indicators such as a Q wave and patient history identify the timing. Regardless of the location, the goals of medical therapy are to prevent or minimize myocardial tissue death and prevent complications. The pathophysiology of CAD and the risk factors involved were discussed earlier in this chapter.

Clinical Manifestations

Chest pain that occurs suddenly and continues despite rest and medication is the presenting symptom in most patients with ACS. Some of these patients have prodromal symptoms or a previous diagnosis of CAD, but about half report no previous symptoms (AHA, 2007). Patients may present with a combination of symptoms, including chest pain, shortness of breath, indigestion, nausea, and anxiety. They may have cool, pale, and moist skin. Their heart rate and respiratory rate may be faster than normal. These signs and symptoms, which are caused by stimulation of the sympathetic nervous system, may be present for only a short time or may persist. In many cases, the signs and symptoms of MI cannot be distinguished from those of unstable angina; hence, the evolution of the term ACS.

Assessment and Diagnostic Findings

The diagnosis of ACS is generally based on the presenting symptoms (Chart 28-6); the 12-lead ECG and laboratory tests (eg, serial cardiac biomarkers) are performed to clarify whether the patient has unstable angina, NSTEMI, or STEMI. The prognosis depends on the severity of coronary artery obstruction and the presence and extent of myocardial damage. Physical examination is always conducted, but the examination alone does not confirm the diagnosis.

Patient History

The patient history includes the description of the presenting symptom (eg, pain), the history of previous cardiac and other illnesses, and the family history of heart disease. The history should also include information about the patient’s risk factors for heart disease.

Electrocardiogram

The 12-lead ECG provides information that assists in ruling out or diagnosing an acute MI. It should be obtained within 10 minutes from the time a patient reports pain or arrives in the emergency department. By monitoring serial ECG changes over time, the location, evolution, and resolution of an MI can be identified and monitored.

The ECG changes that occur with an MI are seen in the leads that view the involved surface of the heart. The classic ECG changes are T-wave inversion, ST-segment elevation, and development of an abnormal Q wave (Fig. 28-5). Because infarction evolves over time, the ECG also changes over time. The first ECG signs of an acute MI occur as a result of myocardial ischemia and injury. Myocardial injury causes the T wave to become enlarged and symmetric. As the area of injury becomes ischemic, myocardial repolarization is altered and delayed, causing the T wave to invert. The ischemic region may remain depolarized while adjacent areas of the myocardium return to the resting state. Myocardial injury also causes ST-segment changes. The injured myocardial cells depolarize normally but repolarize more rapidly than normal cells, causing the ST segment to rise at least 1 mm above the isoelectric line (the area between the T wave and the next P wave is used as the reference for the isoelectric line) when measured 0.06 to 0.08 seconds after the end of the QRS, a point called the J point (Fig. 28-6). This elevation in the ST segment in two contiguous leads is a key diagnostic indicator for MI (ie, STEMI).

The appearance of abnormal Q waves is another indication of MI. Q waves develop within 1 to 3 days because there is no depolarization current conducted from necrotic tissue. The lead system then views the flow of current from other parts of the heart. A new and significant Q wave is 0.04 seconds or longer and 25% of the R-wave depth (provided the R wave exceeds a depth of 5 mm). An acute MI may also cause a significant decrease in the height of the R wave. During an acute MI, injury and ischemic changes are usually present. An abnormal Q wave may be present without ST-segment and T-wave changes, which indicates an old, not acute, MI. For some patients, there are no persistent ECG changes, and the MI is diagnosed by blood levels of cardiac biomarkers.

Using the above information, patients are diagnosed with one of the following forms of ACS:

- **Unstable angina:** The patient has clinical manifestations of coronary ischemia, but ECG and cardiac biomarkers show no evidence of acute MI.
- **STEMI:** The patient has ECG evidence of acute MI with characteristic changes in two contiguous leads on a 12-lead ECG. In this type of MI, there is significant damage to the myocardium.
- **NSTEMI:** The patient has elevated cardiac biomarkers but no definite ECG evidence of acute MI.

During recovery from an MI, the ST segment often is the first ECG indicator to return to normal (1 to 6 weeks). The T segment and T-wave changes, which indicates an old, not acute, MI. For some patients, there are no persistent ECG changes, and the MI is diagnosed by blood levels of cardiac biomarkers.

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wave becomes large and symmetric for 24 hours, and it then inverts within 1 to 3 days for 1 to 2 weeks. Q-wave alterations are usually permanent. An old STEMI is usually indicated by an abnormal Q wave or decreased height of the R wave without ST-segment and T-wave changes.

**Echocardiogram**

The echocardiogram is used to evaluate ventricular function. It may be used to assist in diagnosing an MI, especially when the ECG is nondiagnostic. The echocardiogram can detect hypokinetic and akinetic wall motion and can determine the ejection fraction (see Chapter 26).

**Laboratory Tests**

Cardiac enzymes and biomarkers are used to diagnose an acute MI. Cardiac biomarkers, which include myoglobin and troponin, can be analyzed rapidly, expediting an accurate diagnosis. These tests are based on the release of cellular contents into the circulation when myocardial cells die. Figure 28-7 shows the time courses of cardiac enzymes and biomarkers.

**Creatine Kinase and Its Isoenzymes**

There are three creatine kinase (CK) isoenzymes: CK-MM (skeletal muscle), CK-MB (heart muscle), and CK-BB (brain tissue). CK-MB is the cardiac-specific isoenzyme; it is found mainly in cardiac cells and therefore increases only when there has been damage to these cells. Elevated CK-MB assessed by mass assay is an indicator of acute MI; the level begins to increase within a few hours and peaks within 24 hours of an MI. If the area is reperfused (eg, due to thrombolytic therapy or PCI), it peaks earlier.

**Myoglobin**

Myoglobin is a heme protein that helps transport oxygen. Like CK-MB enzyme, myoglobin is found in cardiac and skeletal muscle. The myoglobin level starts to increase within 1 to 3 hours and peaks within 12 hours after the onset of symptoms. An increase in myoglobin is not very specific in indicating an acute cardiac event; however, negative results are an excellent parameter for ruling out an acute MI.

**Troponin**

Troponin, a protein found in the myocardium, regulates the myocardial contractile process. There are three isomers of troponin: C, I, and T. Troponins I and T are specific for cardiac muscle, and these biomarkers are currently recognized as reliable and critical markers of myocardial injury (Carreiro-Lewandowski, 2006). An increase in the level of troponin in the serum can be detected within a few hours during acute MI. It remains elevated for a long period, often as long as 3 weeks, and it therefore can be used to detect recent myocardial damage.

**Medical Management**

The goals of medical management are to minimize myocardial damage, preserve myocardial function, and prevent complications. These goals are facilitated by the use of guidelines developed by the American College of Cardiology (ACC) and the AHA (Chart 28-7). These goals may be achieved by reperfusing the area with the emergency use of thrombolytic medications or by PCI. Minimizing myocardial damage is also accomplished by reducing myocardial oxygen demand and increasing oxygen supply with medications, oxygen administration, and bed rest. The resolution of pain and ECG changes indicate that demand and supply are in equilibrium; they may also indicate reperfusion. Visualization of blood flow through an open vessel in the catheterization laboratory is evidence of reperfusion.

**Pharmacologic Therapy**

The patient with suspected MI is given aspirin, nitroglycerin, morphine, an IV beta-blocker, and other medications as indicated while the diagnosis is being confirmed. Patients should continue the beta-blocker throughout hospitalization and after discharge because long-term therapy with beta-blockers can decrease the incidence of future cardiac events. Unfractionated heparin or an LMWH is prescribed along with platelet-inhibiting agents to prevent further clot formation. Nonsteroidal anti-inflammatory drugs (NSAIDS)