Acute STEMI and Complications of Myocardial Infarction

Definition and Epidemiology

Sustained myocardial ischemia, regardless of its cause, can result in myocardial necrosis, which underlies the clinical syndrome of MI. MI represents a spectrum of myocardial necrosis, from relatively small amounts of muscle in the case of demand ischemia, to more extensive subendocardial MI that characterizes NSTEMI, to typically large transmural MIs commonly manifesting as STEMI. The current accepted definition of acute MI accounts for clinical setting and mechanism. STEMI represents the range of large MIs that are almost always caused by total occlusion of an epicardial coronary artery resulting in extensive transmural myonecrosis (Fig. 8-8). In contrast, NSTEMI reflects subtotal coronary occlusion leading to subendocardial myonecrosis. Whereas both NSTEMI and STEMI are life-threatening, their different underlying mechanisms mandate different therapeutic strategies and affect the urgency with which they are applied.

One half of all deaths in the United States and developed countries are related to cardiovascular disease. In the United States, there are approximately 1.2 million nonfatal or fatal MIs each year. CAD plays a role in 650,000 deaths each year, and 250,000 deaths are caused by acute MI. One half of patients with acute MI at presentation die within 1 hour of onset, before therapy can be instituted. Of the 5 million patients who come to emergency rooms with chest pain, 1.5 million are admitted to hospital with ACS. In this group of patients, the presence of ST elevation on ECG or an LBBB indicates the diagnosis of STEMI and the need for prompt intervention to open an occluded coronary artery. STEMI accounts for 30% of all MIs, but this mechanism of MI is associated with the highest immediate mortality risk, prompting the need for urgent therapeutic intervention.

Pathology

Lipid-rich coronary plaques are subject to inflammation incited by the response to oxidation of LDL-cholesterol within the plaque. A sequence of inflammatory events leads to macrophage accumulation and the elaboration of metalloproteinases that degrade collagen in the fibrous cap of the plaque. Thinning of the fibrous cap makes the plaque vulnerable to rupture and exposure of blood to thrombogenic stimuli, resulting in platelet aggregation and activation, thrombin generation, and the evolution of fibrin-based thrombus. If the occlusion is total, transmural myocardial ischemia and necrosis ensue and the ECG demonstrates ST elevation. In contrast, partially occlusive thrombus can result in unstable angina or NSTEMI (subendocardial MI). The presence of coronary collaterals can limit the extent of ischemia and necrosis in either scenario. Both STEMI and NSTEMI can set the stage for arrhythmias and LV dysfunction. Whereas coronary thrombosis is the cause of most MIs, there are patients who develop MI related to coronary embolization, coronary vasospasm, vasculitis, coronary anomalies, dissection of the aorta or a coronary artery, or trauma.

One key feature of the pathology of MI is its time-dependent nature. Experimental and clinical studies have documented that coronary occlusion leads to ischemia and myonecrosis in a wavefront manner, from endocardium to epicardium. Restoration of flow to the vessel within 6 hours after occlusion is associated with limitation of infarct size and a favorable effect on mortality risk. The principle of time dependency of MI drives the need to aggressively reperfuse occluded coronary arteries, and this is the cornerstone of contemporary therapy for STEMI.

Clinical Presentation

Patients with acute MI usually have a combination of chest discomfort, ECG changes (ST elevation in contiguous leads or LBBB), and elevation in biomarkers such as CK-MB and troponin. The high sensitivity and high specificity of troponin have made it the preferred biomarker in the diagnosis of MI. The chest discomfort associated with MI is similar to angina pectoris but more severe in nature. It is usually described as substernal pressure, tightness, or fullness. Patients may have symptoms of discomfort that radiate to the neck, jaw, one or both arms, or the back. Not uncommonly, patients with symptoms of acute MI also experience nausea, vomiting, diaphoresis, apprehension, dyspnea, or weakness. In contrast to angina pectoris associated with stable CAD, acute MI symptoms last longer than 20 to 30 minutes (up to hours).

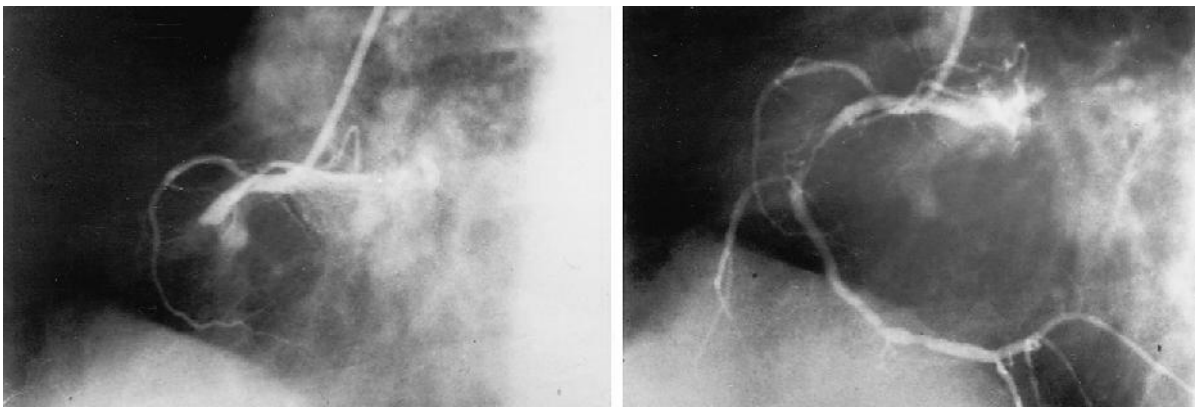

FIGURE 8-8 Right coronary artery angiogram in a patient with acute inferior myocardial infarction. The *left panel* demonstrates total occlusion of the right coronary artery. The *right panel* depicts restoration of flow 90 minutes after the intravenous administration of tissue-type plasminogen activator.