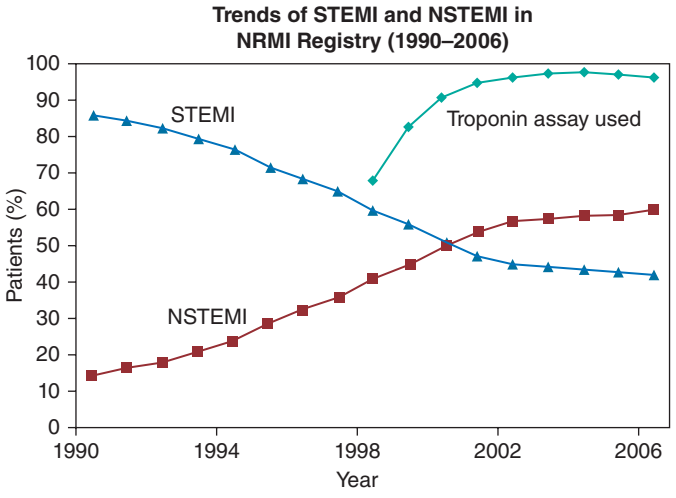

FIGURE 294-1 Trends of incidence of ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI) and of frequency of use of troponin assay to diagnose acute myocardial infarction. NRMI, National Registry of Myocardial Infarction. *(From N Arora, RG Brindis, CP Cannon: Acute coronary syndrome in North America, in Theroux P [ed]: Acute Coronary Syndromes, 2nd ed. Philadelphia: Elsevier, 2011.)*

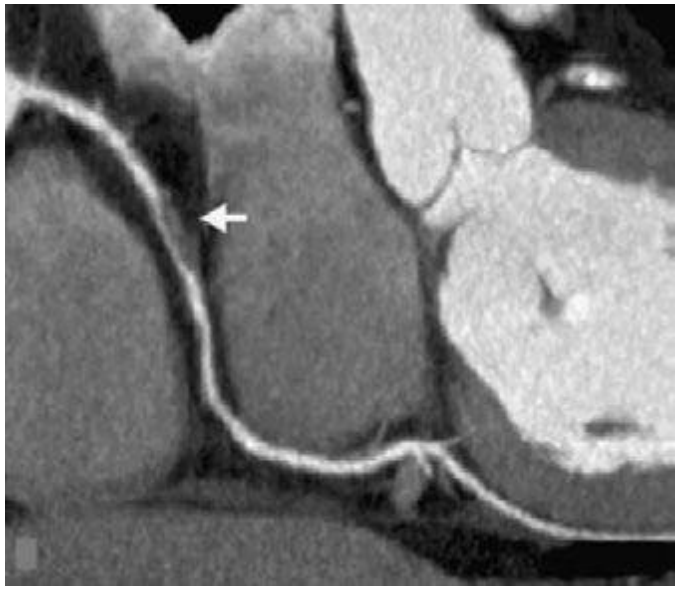

FIGURE 294-2 Coronary computed tomographic angiogram showing an obstructive plaque in the right coronary artery. *(From PJ de Feyter, K Nieman. Multislice computed tomography in acute coronary syndromes, in Theroux P [ed]: Acute Coronary Syndromes, 2nd ed. Philadelphia: Elsevier, 2011.)*

or on eroded coronary artery endothelium. Severe ischemia or myocardial necrosis may occur consequent to the reduction of coronary blood flow caused by the thrombus and by downstream embolization of platelet aggregates and/or atherosclerotic debris. Other causes of NSTE-ACS include: (1) dynamic obstruction (e.g., coronary spasm, as in Prinzmetal's variant angina [see "Prinzmetal's Variant Angina" later]); (2) severe mechanical obstruction due to progressive coronary atherosclerosis; and (3) increased myocardial oxygen demand produced by conditions such as fever, tachycardia, and thyrotoxicosis in the presence of fixed epicardial coronary obstruction. More than one of these processes may be involved.

Among patients with NSTE-ACS studied at angiography, approximately 10% have stenosis of the left main coronary artery, 35% have three-vessel CAD, 20% have two-vessel disease, 20% have single-vessel disease, and 15% have no apparent critical epicardial coronary artery stenosis; some of the latter may have obstruction of the coronary microcirculation and/or spasm. The "culprit lesion" responsible for ischemia may show an eccentric stenosis with scalloped or overhanging edges and a narrow neck on coronary angiography. Optical coherence tomography (an invasive technique) and contrast-enhanced coronary computed tomographic angiography (CCTA), a noninvasive technique (Fig. 294-2), have shown that culprit lesions are composed of a lipid-rich core with a thin fibrous cap. Patients with NSTE-ACS frequently have multiple such plaques that are at risk of disruption (vulnerable plaques).

CLINICAL PRESENTATION

Diagnosis The diagnosis of NSTE-ACS is based largely on the clinical presentation. Typically, chest discomfort is severe and has at least one of three features: (1) it occurs at rest (or with minimal exertion), lasting >10 minutes; (2) it is of relatively recent onset (i.e., within the prior 2 weeks); and/or (3) it occurs with a crescendo pattern (i.e., distinctly more severe, prolonged, or frequent than previous episodes). The diagnosis of NSTEMI is established if a patient with these clinical features develops evidence of myocardial necrosis, as reflected in abnormally elevated levels of biomarkers of cardiac necrosis (see below).

History and Physical Examination The chest discomfort, often severe enough to be described as frank pain, is typically located in the substernal region or sometimes in the epigastrium, and radiates to the left arm, left shoulder, and/or neck. Anginal "equivalents" such as dyspnea, epigastric discomfort, nausea, or weakness may occur instead of chest pain and appear to be more frequent in women, the elderly, and patients with diabetes mellitus. The physical examination resembles that in patients with stable angina (Chap. 293) and may be unremarkable. If the patient has a large area of myocardial ischemia or a large NSTEMI, the physical findings can include diaphoresis; pale, cool skin; sinus tachycardia; a third and/or fourth heart sound; basilar rales; and, sometimes, hypotension.

Electrocardiogram ST-segment depression occurs in 20 to 25% of patients; it may be transient in patients without biomarker evidence of myocardial necrosis, but may be persistent for several days in NSTEMI. T-wave changes are common but are less specific signs of ischemia, unless they are new and deep T-wave inversions (≥0.3 mV).

Cardiac Biomarkers Patients with NSTEMI have elevated biomarkers of necrosis, such as cardiac troponin I or T, which are specific, sensitive, and the preferred markers of myocardial necrosis. The MB isoform of creatine kinase (CK-MB) is a less sensitive alternative. Elevated levels of these markers distinguish patients with NSTEMI from those with UA. There is a characteristic temporal rise and fall of the plasma concentration of these markers and a direct relationship between the degree of elevation and mortality (see Fig. 294-4*B*). However, in patients *without* a clear clinical history of myocardial ischemia, minor cardiac troponin (cTn) elevations have been reported and can be caused by congestive heart failure, myocarditis, or pulmonary embolism, or using high-sensitivity assays, they may occur in ostensibly normal subjects. Thus, in patients with an *unclear* history, small elevations of cTn, especially if they are persistent, may not be diagnostic of an ACS.

With more widespread measurement of troponin, especially using high-sensitivity assays, an increasing fraction of patients with NSTE-ACS are found to have NSTEMI, whereas the fraction of patients with UA is dwindling.

DIAGNOSTIC EVALUATION

In addition to the clinical examination, three major noninvasive tools are used in the evaluation of NSTEMI-ACS: the electrocardiogram (ECG), cardiac biomarkers, and stress testing. CCTA is an additional emerging option (Fig. 294-2). The goals are to: (1) recognize or exclude myocardial infarction (MI) using cardiac biomarkers, preferably cTn;