Cardiovascular Magnetic Resonance Imaging

Textbook and Atlas

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7 Coronary Artery Disease – Acute Coronary Syndromes

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7.1 Definition and Spectrum

The term "acute coronary syndromes" (ACS) refers to any group of clinical symptoms compatible with acute myocardial ischemia and covers the spectrum of clinical conditions ranging from unstable angina (UA) to non-ST segment elevation myocardial infarction (NSTEMI or NSTE-ACS) and to ST segment elevation myocardial infarction (STEMI or STE-ACS). UA and NSTEMI are closely related conditions, but though their pathophysiological origin and clinical presentations are similar, they differ in severity. STEMI refers to prolonged myocardial ischemia resulting in myocyte cell death that may be diagnosed by biochemical, electrocardiographic, and imaging modalities. The differentiation between these three types of ACS depends on clinical presentation, electrocardiographic changes and biochemical cardiac markers (Figure 7-1).

Serum concentration of cardiac enzymes and markers of necrosis define the diagnostic categories of acute coronary syndrome, unstable angina and myocardial infarction (Thygesen 2007). Troponin T (cTnT) and troponin I (cTnI) are cardiac markers, which are extremely sensitive to myocardial injury and damage. Minimal myocardial damage can be detected by elevation in troponin concentration without significant rise in creatine kinase or other cardiac enzymes. The guidelines of the European Society of Cardiology and American College of Cardiology state that any elevation of troponin or creatine kinase MB (muscle, brain) isoenzyme is evidence of myocardial necrosis and should be classified as myocardial infarction (Van de Werf 2003, Antman 2004).

The **three types** of ACS include:

- **myocardial infarction (MI) with ST segment elevation** = STEMI = STE-ACS: severe refractory chest pain, persistent > 20 min of ≥ 0.2 mV ST elevation in at least two contiguous leads in men or ≥ 0.15 mV in women in leads V2–3 and/or ≥ 0.1 mV in other leads (Thygesen 2007) and elevated markers of myocardial necrosis;

- **myocardial infarction (MI) with no ST segment elevation** = NSTEMI = NSTE-ACS: severe chest pain, ST segment and/or T-wave alterations without ST-elevation, elevated markers of myocardial necrosis; and
- **unstable angina** = UA: intermittend chest pain, varying ECG alterations except ST segment elevations, e. g. ST segment depression and/or T-wave alterations, normal serum concentrations of markers of myocardial necrosis.

The differentiation between UA, NSTEMI and STEMI is made by ECG changes and biomarkers of myocardial necrosis (Figure 7-1).

7.2 Etiology and Pathophysiology

It is well established that ACS in their different clinical presentations share a widely common pathophysiological substrate. Pathological, biological, and angioscopic observations have demonstrated that atherosclerotic plaque erosion or rupture with differing degrees of superimposed thrombosis and distal embolization with consecutive regional myocardial ischemia represent the basic mechanism in most ACSs. After fissuring or rupture of these plaques the core constituents such as collagen, lipids, and smooth muscle and foam cells are exposed to flowing blood resulting in platelet aggregation and adhesion and the formation of intracoronary thrombus by concomitant generation of thrombin and deposition of fibrin. Such fresh thrombus – if persistent –

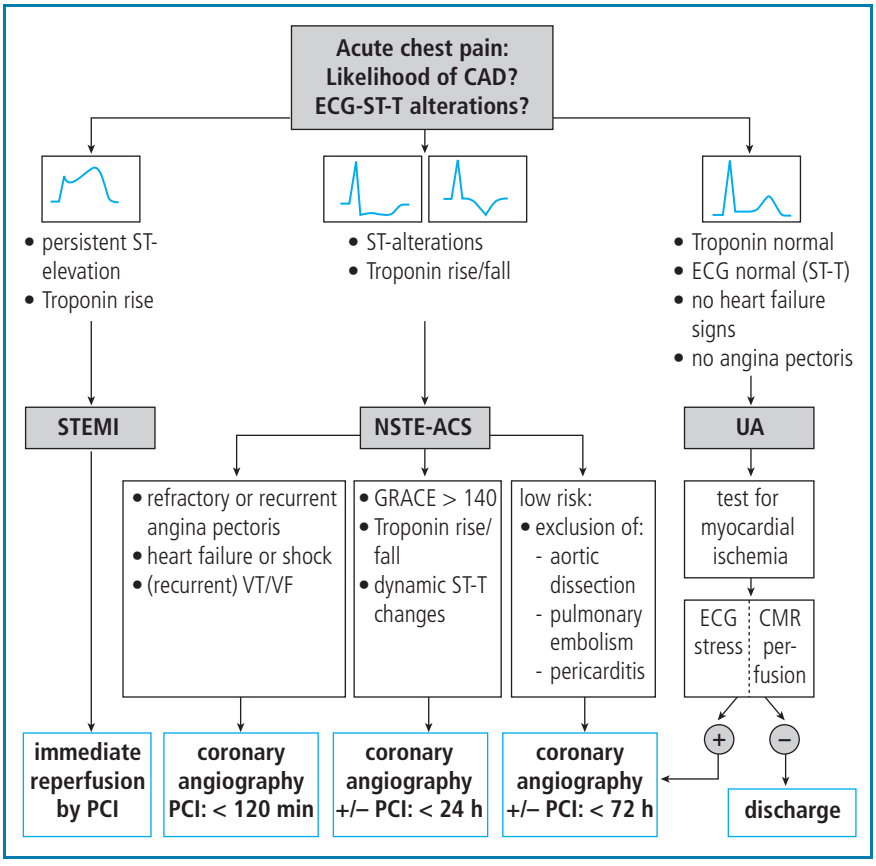

Figure 7-1 Stepwise diagnostic approach, risk stratification, and treatment in patients with acute coronary syndromes (ACS) (adapted and modified from the current Guidelines of the European Society of Cardiology; Hamm et al. 2011). Patients with obvious STEMI should immediately undergo reperfusion. Patients with NSTEMI and high risk (refractory angina, heart failure or shock, VT) should have coronary angiography within 12 h, those with intermediate risk (GRACE score > 140, troponin rise/fall, dynamic ST-T changes) within 24 h, and those with low risk within 72 h. Patients with unstable angina (UA) (no or insignificant ECG changes, troponin normal, no heart failure or angina pectoris) should undergo a stress test (treadmill stress test, stress echo, stress CMR) to detect myocardial ischemia. If the stress test is negative, patients can be treated medically and followed without any further intervention; in case of a positive test with demonstrable myocardial ischemia patients should undergo coronary angiography and revascularization, if appropriate and necessary. Note that using CMR imaging (for stress perfusion or stress regional function to detect myocardial ischemia with high accuracy, exact measurement of infarct size, and detection of microvascular obstruction) independent prognostic parameters can be obtained that may influence further diagnostic and therapeutic work-up of the patients.

may occlude the respective vessel resulting in STEMI or STE-ACS, or cause luminal narrowing resulting in NSTEMI or NSTE-ACS with subsequent myocardial ischemia of the corresponding myocardial perfusion territory. Plaque rupture and thrombus formation may also induce local vasoconstriction that may further reduce regional myocardial perfusion and exacerbate ischemia. Prolonged myocardial ischemia, e.g. in thrombotic occlusion, causes myocyte cell death within 2–4 h or longer, depending on the presence of collateral circulation, persistent or intermittent coronary artery occlusion, the sensitivity of myocytes to ischemia, and individual myocardial oxygen demand. In case of an acute myocardial infarction myocardial edema occurs prior to myocardial necrosis (Willerson 1977).

7.3 Clinical Presentation and Therapy

7.3.1 Relevance of Clinical Signs of Acute Coronary Syndromes (ACS)

A systematic review of 21 studies examined the usefulness of sixteen different clinical signs and symptoms for the diagnosis of acute coronary syndromes and found that no single sign or symptom taken in isolation was discriminatory (Mant 2004).

> The guidelines of the American Heart Association/American College of Cardiology (AHA/ACC) recommend that **five factors should be considered together** when assessing the likelihood of myocardial ischemia related to ACSs (Antman 2004). These are:
> - the nature of the symptoms,
> - the history of ischemic heart disease,
> - gender,
> - increasing age, and
> - the number of traditional cardiovascular risk factors present.

High risk features include worsening angina, prolonged pain, pulmonary edema, hypotension and arrhythmias.

7.3.2 ST-Elevation Myocardial Infarction (STEMI or STE-ACS)

Patients present with severe chest pain lasting for 10–20 min or more not responding fully to nitroglycerine. Often located in the substernal region the pain or pressure frequently radiates to the neck, jaw, left shoulder, or left arm. Other primary locations of pain my be epigastric or interscapular. Though there are no individual physical signs diagnostic of STEMI, many patients have evidence of autonomic nervous system activation (pallor, sweating) and either hypotension or narrow pulse pressure, as well as irregularities of the pulse including bradycardia or tachycardia. The 12-channel ECG shows persistent (> 20 min) ST segment elevation or presumed new left bundle branch block (LBBB). Blood sampling in the early hours for cardiac markers of necrosis (troponins, creatinine kinase) should be done, but immediate reperfusion therapy should not be deferred for getting the results of laboratory tests. Echocardiography may detect regional wall motion abnormalities that occur within seconds after coronary artery occlusion well before myocardial necrosis. After establishing the working diagnosis of STEMI patients should be treated for pain relief (i.v. opiods), oral O_2 (2–4 l/min), and anxiety (tranquilizer), if necessary. In addition, a β-blocker (i.v. low dose, contraindicated in patients presenting with hypotension) and a loading dose of a platelet aggregation inhibitor (clopidogrel, aspirin) has to be given in the acute setting, as well as anticoagulant agents (heparin). The patients should immediately undergo a reperfusion therapy, preferably catheter-based mechanical coronary reperfusion (PCI), because of the wavefront evolution of infarct size over time ("time is muscle"). In cases without PCI facility (PCI not manageable within 90 min from symptom onset) systemic thrombolysis should be performed followed later on by PCI ("facilitated PCI"). Longterm routine treatment includes oral platelet inhibitor, β-blocker, ACE-inhibitor, and a statin. Post-interventional follow-up includes regular clinical and echocardiographic examinations and – if available – serial CMR studies for early detection of infarct complications, adverse ventricular remodeling, and for determining prognosis.

7.3.3 Non-ST-Elevation Myocardial Infarction (NSTEMI or NSTE-ACS) and Unstable Angina (UA)

Patients present with chest or left arm pain or discomfort of varying duration that reproduces previously documented angina. A high likelihood of an ACS (NSTEMI or UA) exists, if patients with chest pain have a known history of CAD, including MI, have transient mitral regurgitation murmur, hypotension, diaphoresis, pulmonary edema, or rales on clinical examination, show new or presumably new transient ST segment deviation (≥ 1 mm) or T-wave inversion in multiple precordial leads of the ECG, and have elevated cTnT, cTnI, or CK-MB levels (Kumar and Cannon 2009). Patients with unstable angina (UA) present with chest pain of varying duration, ST segment depression and/or T-wave alterations, but normal levels of biomarkers of necrosis (cTnT, cTnI, CK-MB). Diagnosis is established by clinical

examination, serial ECG recordings (ST-T-abnormalities, often dynamic changing ECG patterns), blood sampling (cTnT, cTnI, CK-MB), and echocardiography (regional wall motion abnormalities). CMR imaging may be extremely helpful in clinically stable patients for detecting old infarcts (scars by LGE), assessing global or regional wall motion abnormalities or involvement of the right ventricle, and to detect and quantify the severity of myocardial ischemia on stress testing (dobutamine stress CMR or adenosine stress first-pass CMR).

The goal of immediate treatment of patients with NSTEMI/UA is to provide relief of ischemia and to prevent the recurrence of adverse ischemic events. Therefore, treatment with anti-ischemic (nitroglycerine, morphine, β-blocker, calcium channel blocker, ACE-inhibitor), antiplatelet (aspirin, clopidogrel, newer P2Y12 ADP inhibitors like Prasugrel, Ticagrelor), and anticoagulant agents (unfractionated heparin, low-molecular heparin, direct thrombin inhibitors, factor Xa inhibitors) is fundamental (ACC/AHA 2007 guidelines).

In addition to aggressive medical therapy, **two treatment pathways** have emerged for treating NSTEMI/UA patients:

- an **early invasive strategy**, i. e. routine cardiac catheterization within 4–12 h after admission followed by PCI or bypass grafting, and
- an **initial conservative strategy**, i. e. initial medical management, followed by catheterization and revascularization only if ischemia recurs despite vigorous medical therapy, according to a risk stratification protocol (Figure 7-1).

High risk patients with the need of immediate invasive strategy are those with recurrent angina or ischemia at rest or low level activities despite intensive medical therapy, elevated biomarkers (cTnT, cTnI), new or presumably new ST segment depression, signs or symptoms of heart failure or new or worsening mitral regurgitation, hemodynamic instability, high-risk findings from non-invasive testing, PCI within 6 mths or prior CABG, high risk score (TIMI, GRACE), and reduced LV ejection fraction. Low risk patients with initial conservative therapy are those with a low risk score (TIMI, GRACE), and patient or physicians preference in the absence of high risk features (Kumar and Cannon 2009). Long-term follow-up includes regular clinical examinations, echocardiographic controls, and stress tests, and CMR imaging may play a significant role in the long-term surveillance of patients with NSTEMI/UA.

7.4 CMR Sequences and Protocols

CMR is regarded the gold standard in the measurement of left ventricular volumes and mass, because it relies on the acquisition of a 3D stack of contiguous short-axis cines covering the entire left and right ventricles without any geometric assumptions. Left and right ventricular end-diastolic and end-systolic volumes are assessed by planimetry for each slice and consecutive summation. CMR offers higher diagnostic accuracy and reproducibility in comparison to other imaging techniques and may be used in longitudinal studies over time and for reducing sample size for drug studies. Steady-state free precession (SSFP) cine images in short-axis orientation with complete biventricular coverage, from the atrioventricular valve plane to the apex are recommended for functional analysis (Kramer 2008). For further details see Chapter 5.

In acute coronary syndromes (ACS) early myocardial edema occurs in many forms of acute myocardial injury. CMR can visualize myocardial areas of edema by T2-weighted sequences (Aletras 2006, Friedrich 2008). In ACS edematous regions represent areas of myocardium at risk. First-pass myocardial perfusion CMR visualizes myocardial passage of a bolus of contrast agent and may detect regional perfusion abnormalities in stable angina as well as in ACS (Schwitter 2001). CMR is superior to most other imaging

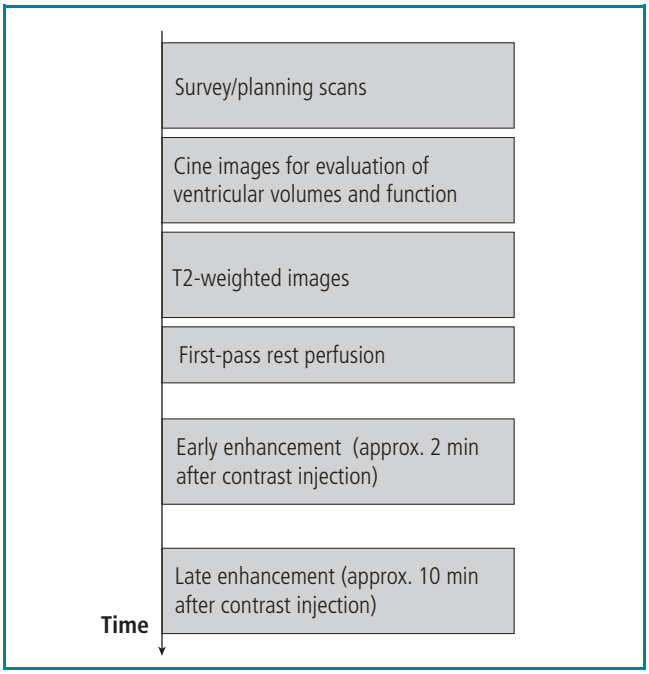

Figure 7-2 CMR protocol to investigate patients with an acute coronary syndrome (ACS).