Acute Coronary Syndrome (ACS) — Causes and Treatment

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The acute coronary syndrome is defined as acute anginal pain because of partial or total occlusion of one or more coronary arteries due to advanced coronary heart disease. Three clinical entities can be identified: Unstable angina (UA), Non-ST-segment elevation MI (NSTEMI) and ST-segment elevation MI. They can be differentiated based on the ECG changes and the cardiac markers. ACS is initially managed medically with a well-known algorithm. Revascularization of the coronaries either by cardiac catheterization or surgery should be planned as soon as possible.

Definition of Acute Coronary Syndrome

The three entities of acute coronary syndrome

Acute coronary syndrome (ACS) is composed of three entities:

- **Unstable angina pectoris**
- **Non-ST segment elevation myocardial infarction (NSTEMI)**
- **ST-segment elevation myocardial infarction (STEMI)**

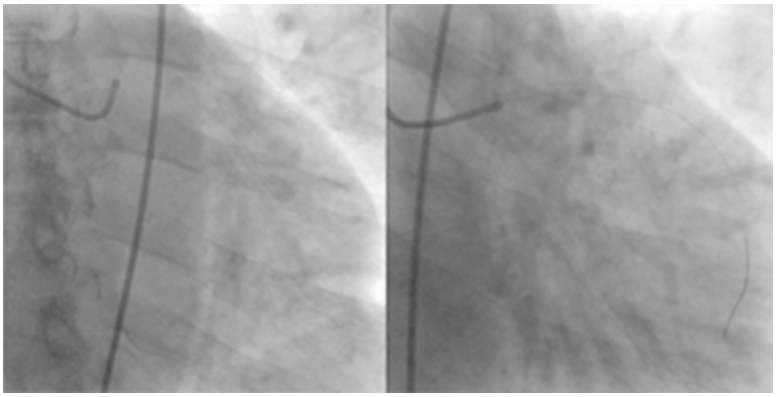

Image: “Acute coronary syndrome (ACS),” by Jer5150. License: CC BY-SA 3.0

Diagnosis	Clinical features	ECG findings	Laboratory findings
Unstable angina	Ischemic chest pain that occurs at rest or with previously tolerated levels of exertion	None, or ST-segment depressions	***None***
Non-ST-elevation Myocardial infarction (NSTEMI)	Ischemic chest pain in any setting	None or ST-segment depressions	***Elevated troponin***
ST-elevation Myocardial infarction (STEMI)	Ischemic chest pain in any setting	***ST-segment elevations***	(Elevated troponin)

Epidemiology of Acute Coronary Syndrome

The acute coronary syndrome as the most frequent cause of death in industrialized countries

Coronary heart disease, which, among others, can be symptomatic as an ACS, is the most common cause of death in industrialized countries. Every year about 8 million people experience mortality related to coronary heart disease.

- US: 780,000 cases/year
- Mean age: 68 (IQR 56—79)
- M: F 3:2
- 70 % non-ST-elevation

Etiology of Acute Coronary Syndrome

Causes for the acute coronary syndrome

In about 95% of the cases, a ruptured plaque, which has formed inside the coronary arteries within the context of atherosclerosis, is responsible for ACS symptoms. Arthritides, endocarditis, cocaine use, emboli of heart valve prostheses, or other paradoxical embolisms are of less significance etiologically.

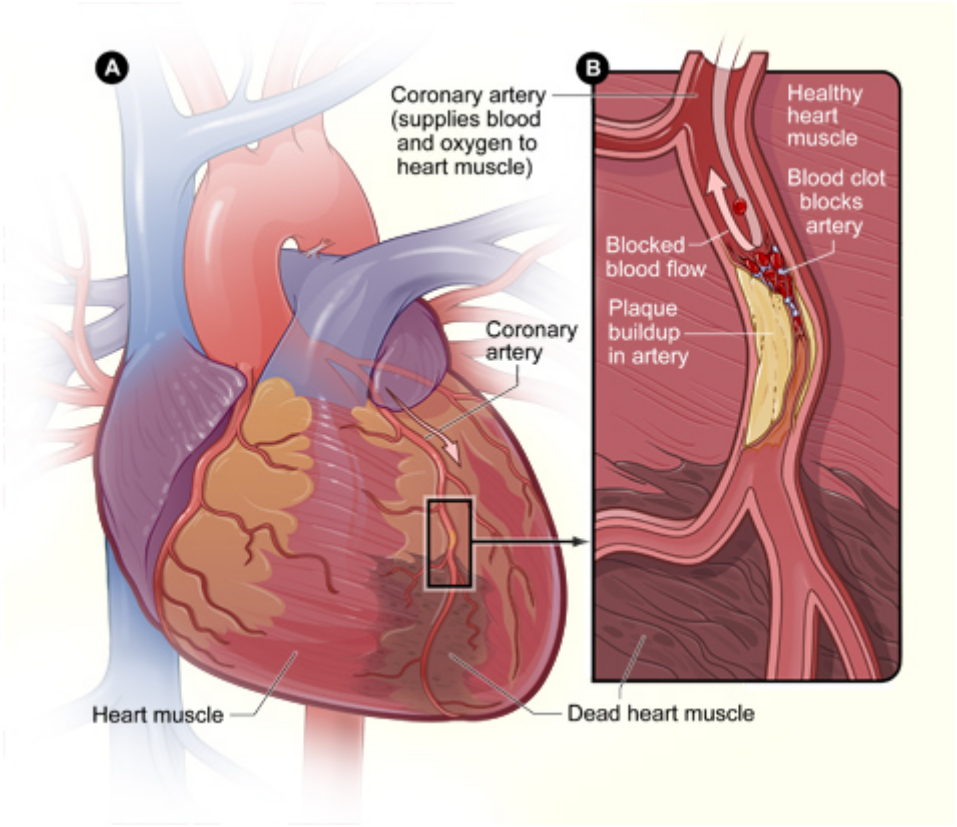

Image: “Figure A is an overview of a heart and coronary artery showing damage (dead heart muscle) caused by a heart attack. Figure B is a cross-section of the coronary artery with plaque buildup and a blood clot resulting from plaque rupture.” by National Heart Lung and Blood Institute (NIH). License: Gemeinfrei

Classification of Acute Coronary Syndrome

Classification of acute coronary syndrome

ACS classification can be carried out using the GRACE-Risk-Scores, which allows risk stratification. This score includes parameters such as age or heart rate and must be calculated using the GRACE-Risk-Scores-Software.

The risk can be determined both for the time of hospital stay and for the post-stationary mortality rate. The risk of dying in the hospital, for example, with a score of < 108 points, is less than 1%. With a score of > 140 points, however, the risk is 3%.

Pathophysiology of Acute Coronary Syndrome

Acute coronary syndrome as occlusion of a coronary artery

A plaque caused by manifest atherosclerosis in the coronary arteries ruptures and sets free plaque components. As a result, thrombocyte aggregation is activated in the blood vessels.

The repeated embolization can lead to the symptoms of unstable angina pectoris or NSTEMI. A thrombus causing complete occlusion of the coronary artery results in STEMI.

Symptoms of Acute Coronary Syndrome

Symptoms of acute coronary syndrome

There is often a sudden, severe pain that typically radiates to the left shoulder and left arm. (Caution: radiation to the right side of the body is also possible!) It often occurs without previous exertion, improves only slightly, or not at all, by the administration of nitroglycerin or rest and lasts longer than 15 minutes.

The pain can be described as an unbearable, cramp-like tightness in the chest. In addition, dyspnea and fear of death, as well as accompanying weakness, nausea, and sweating, are typical. Radiation to the upper abdomen, spine, or neck can also occur. A drop in blood pressure and tachycardia, accompanied by cold sweating, can be signs of a begin

Diagnostics of Acute Coronary Syndrome

ECG and assessment of cardiac enzymes in acute coronary syndrome

In addition to medical history and physical examination, which may indicate the typical pain characteristics and reveal blood pressure changes, a 12-lead ECG and the evaluation of cardiac enzymes CK, CK-MB (specific for myocardial infarction), and especially troponins I and T, are crucial.

Image: “ECG paper 12 leads,” by Madhero88. License: CC BY-SA 3.0

Further, assessments for myoglobin, LDH, GOT, CRP, and creatinine can be useful. However, myoglobin levels can also rise under strong physical effort, and GOT can also occur in liver cells and skeletal muscles, apart from the heart. LDH can only serve as a long-time marker as it only begins to rise after 6–12 hours and reaches its maximum after a few days.

Monophasic ST-segment elevation – Stages of infarction

Unstable angina pectoris is characterized by a lack of increase in troponin levels. For both NSTEMI and STEMI, the cardiac enzymes are altered. STEMI, according to its name, is characterized by a monophasic ST-segment elevation, which directly passes into the T-wave. Depending on the stage of the infarction, this alteration can vary:

- Stage 0 (initial stage): up to 6 hours after infarction, typically marked by a

peak increase of the T-wave, labeled as suffocation-T
- Stage 1 (acute infarct): a few hours to days after infarction, characterized by the typical monophasic ST elevation
- Stage 2 (intermediate stage): the ST elevation, as well as the R-wave, deteriorate, the so-called R-loss and a magnification of the Q-wave occur, which, as Pardée-Q, signals the sinking of the myocardium. A peak negative modulation of the T-wave is created
- Stage 3 (final stage): more than 6 months after infarction, the changes of the QRS-complex remain, the Pardée-Q typically even lasts for a lifetime, while the ST segment and T-wave normalize

With NSTEMI, this ST elevation is missing in the ECG; instead, ST segment depression or deep-negative T waves can be observed. Even if there is no direct increase in troponin values (70% after 3 hours, 90% after 6 hours), an infarct cannot immediately be excluded. The measurements must be repeated every 3 hours.

From the 3rd day onwards, the troponin values can provide information about the extent of the infarct. They are not suitable for the diagnosis of a re-infarct because they remain elevated for about 2 weeks before their levels return to normal. Therefore, it is better to use CK-MB instead of troponin to diagnose post-MI re-infarction. Cardiac markers can be influenced by other diseases, such as a pulmonary embolism, an acute cardiac degeneration, or even kidney insufficiency.

Echocardiography in acute coronary syndrome

In echocardiography, wall motion defects can be immediately displayed. The gold standard, however, is a heart catheter examination carried out as quickly as possible. In a cardiac MRI, infarct scars can be visualized.

Pathology of Acute Coronary Syndrome

Yellowish spots, whitish scar tissue

The pathology can be traced back to the lack of blood flow to the heart tissue. With perfusion of less than 25% of the normal flow, the tissue is irreversibly damaged. After 6–12 hours, the damage can be seen by light microscope in the form of yellowish spots.

It results in full myocytolysis, beginning with the cell nuclei fading and the cross-striation being lost. After a few days, granulation tissue forms, containing a hemorrhagic edge and many marginal leukocytes. Only after 2 weeks does whitish scar tissue occur.

Differential Diagnosis of Acute Coronary Syndrome

Diseases similar to acute coronary syndrome

Changes in ECG can also be ascribed to other diseases. For example, ST elevations may also occur during pericarditis or left heart hypertrophy, and deep Q waves may occur during pulmonary embolism, Wolff-Parkinson-White syndrome, or hypertrophic cardiomyopathy.

Furthermore, all other causes of chest pain have to be clearly distinguished from ACS. The most important causes are pulmonary embolism, aortic dissection, tension

pneumothorax, and spontaneous rupture of the esophagus (Boerhaave-Syndrome). Moreover, myocarditis, pericarditis, and musculoskeletal chest pain are important differential diagnoses that come up during academic tests.

Therapy of Acute Coronary Syndrome

Continuous administration of oxygen in acute coronary syndrome

From initial treatment up to the final diagnosis, oxygen can be administered. Keeping the upper body in an elevated position to relieve the shortness of breath or heart failure is also recommended. Nitroglycerin (1 capsule or 2 squirts = 0.8 mg, 1–5 mg intravenously, caution: contraindicated for SBP < 90 mm Hg and administration of PDE-5 inhibitors!) can be administered, and morphine (3–5 mg intravenously every 5–10 minutes) can be given during strong pain which is not relieved by nitroglycerine.

Acetylsalicylic acid and heparin administration, stress ECG in acute coronary syndrome

Furthermore, all patients receive acetylsalicylic acid (250–500 mg); in case of an infarct, they should also receive heparin (5000 IU). Intramuscular injections should not be performed because the CK values increase and make lysis therapy impossible. During unstable angina pectoris, which can only be safely distinguished from an infarct after 6 hours, more diagnostic tests, such as a stress ECG, are required. Subsequently, a decision on further therapy is made.

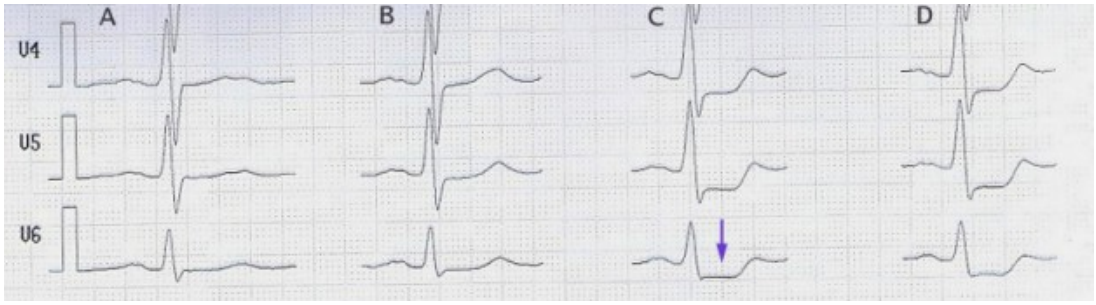

Image: “Stress test with ST-Depression (Arrow) from 100 W (column C),” by JHeuser. License: CC BY-SA 3.0

Cardiac catheterization for NSTEMI

In case of NSTEMI, cardiac catheterization is mandatory, with the time frame for implementation ranging from 2–72 hours, depending on the risk profile of the patient. For STEMI, the obstructed coronary vessel should be revascularized as quickly as possible by means of PCI.

Risk Assessment for Acute Coronary Syndrome

This takes priority in all patients with suspected ACS. There are many risk stratification tools available. Their goal is to predict major adverse cardiac events (MACE).

HEART score for chest pain patients

History	Highly suspicious	2
	Moderately suspicious	1
	Slightly suspicious	0
ECG	Significant ST depression	2
	Non-specific repolarization disturbance	1
	Normal	0
Age	≥ 65 years	2
	45—65 years	1
	≤ 45 years	0
Risk factors	≥ 3 risk factors or history of atherosclerotic disease	2
	1 or 2 risk factors	1
	No risk factors are known	0
Tropin	≥ 3x normal limit	2
	1—3x normal limit	1
	≤ normal limit	0

Risk Factors

- Hypertension
- Diabetes
- Hypercholesterolemia
- Obesity (BMI > 30)
- Positive family history of ACS < 65 years of age
- Known cardiovascular disease (CAD, TIA/stroke, PAD)
- Smoking

Using the HEART score

HEART score	% of patients	% MACE	Recommended management
0—3	32	1—2	Discharge
4—6	51	12—17	Observe, risk reduction, noninvasive testing
7—10	17	50—65	Admit, medical management, consider early invasive testing

Complications of Acute Coronary Syndrome

The complications of ACS are complex; their severity depends on the extent of myocardial injury. Depending on the damaged area, right or left ventricular failure or, in case of a damaged papillary muscle, insufficiency of the atrioventricular valves, can occur.

Heart rhythm disorders can result from the deprivation of an area, which can even lead to a sudden cardiac arrest. In addition to cardiogenic shock, there is the threat of myocardial aneurysm rupture. Moreover, a myocardial rupture is possible. As a result of infarction, pericarditis might still occur over the following 6 weeks.

Prevention of Acute Coronary Syndrome

As 95% of ACS cases are manifestations of coronary heart disease, the goal is to avoid the latter condition. For the prevention of coronary heart disease, it is advised that risk factors be eliminated, especially major risk factors like cholesterol, arterial hypertension,

diabetes mellitus, and smoking. The approaches are predominantly targeted at behavioral modifications such as nutrition and exercise. These approaches can also be assisted by medication.

References

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Notes