Acute Coronary Syndrome (ACS) — Causes and Treatment

The acute coronary syndrome is one of the most common causes of death in the industrialized countries. Since with every minute that passes, more heart muscle tissue is irreversibly damaged, it requires a rapid diagnosis and therapy. Every physician must, therefore, recognize the symptoms and immediately respond in an adequate fashion. Reading this article will prepare you to recognize the symptoms of acute coronary syndrome both in your future practice and on your exams.

Definition of Acute Coronary Syndrome

The three entities of the acute coronary syndrome

- **Unstable angina pectoris**
- **Myocardial infarction**: Non-ST segment elevation (NSTEMI) and ST segment elevation (STEMI).
- **Acute coronary syndrome (ACS)**

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![Diagram](image-url)
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 the acute coronary syndrome, is the most common cause of death in industrialized countries. Every year about eight million people worldwide die of its consequences.

Etiology of Acute Coronary Syndrome

Causes for 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 the symptoms of the acute coronary syndrome. Arthritides, endocarditis, the consumption of cocaine, emboli of heart valve prostheses or other paradoxical embolisms are of less significance for etiology.
Classification of Acute Coronary Syndrome

Classification of the acute coronary syndrome

A classification can be carried out using the GRACE-Risk-Scores, whereby risk stratification can be conducted. 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 the hospital stay and for the post-stationary mortality rate. The risk of dying in the hospital, for example, with a score of fewer than 108 points, is less than 1 %. With a score of over 140 points, however, the risk is already at 3 %.

Pathophysiology of Acute Coronary Syndrome

Acute coronary syndrome as occlusion of a coronary artery

A plaque caused by manifested 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 the
Non-ST segment elevation myocardial infarction. A thrombus causing a complete occlusion of a coronary artery results in an ST-segment elevation myocardial infarction.

Symptoms of Acute Coronary Syndrome

Symptoms of acute coronary syndrome

There is often a sudden, severe pain, which typically radiates into 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 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 into 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 beginning cardiogenic shock. Infarcts often occur during the early hours of the morning.

Diagnostics of Acute Coronary Syndrome

ECG and determination of the cardiac enzymes in acute coronary syndrome

In addition to the medical history and physical examination that hint to the typical pain characteristics and, for example, can reveal blood pressure changes, a 12-lead ECG and the determination of the cardiac enzymes CK, CK-MB (specific for myocardial infarction) and especially the troponins I and T is of crucial importance.

Also, the determination of myoglobin, LDH, GOT, CRP and creatinine can be useful. However, it is to be noted that myoglobin levels can also rise under the strong physical effort, and GOT, apart from the heart, can also occur in liver cells and skeletal muscles. LDH can only serve as a long-time marker as it only begins to rise after six to twelve hours and reaches its maximum after a few days.

Monophasic ST segment elevation – Stages of infarction

The unstable angina pectoris is characterized by a lack of increase in troponin levels. For
both NSTEMI and STEMI, the cardiac enzymes are altered. The ST segment elevation myocardial infarction, due 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 six 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 six 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 again.

With non-ST-segment elevation myocardial infarction, 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 third 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 two weeks before their levels return to normal. Therefore, it’s better to use CK-MB marker 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 a **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 made visible.

### 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 a **perfusion of less than 25 %** of the normal flow, the tissue is irreversibly damaged. After six to twelve hours, the damage can be seen by light microscope in the form of **yellowish spots**.

It results in a 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 two weeks, **whitish scar tissue** occurs.
Differential Diagnosis of Acute Coronary Syndrome

Similar disease patterns to acute coronary syndrome

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

Furthermore, all other causes of chest pain have to be clearly separated from acute coronary syndrome. The most important causes are a pulmonary embolism, aortic dissection, tension pneumothorax and spontaneous rupture of the esophageal (Boerhaave-Syndrome). Also, myocarditis, pericarditis, and musculoskeletal chest pain are often asked during tests.

Therapy of Acute Coronary Syndrome

Continuous administration of oxygen in acute coronary syndrome

For initial treatment up to the final diagnosis, oxygen can be administered. It is also recommended to keep the upper body in an elevated position to relieve the shortness of breath or heart failure. The administration of nitroglycerin (1 capsule or 2 squirts = 0.8 mg, 1—5 mg intravenously, pay attention: contraindicated under systolic blood pressure < 90 mmHg and administration of PDE-5 inhibitors!) and morphine (3—5 mg intravenously every 5—10 minutes) can be given during strong pain which is not relieved by nitroglycerine.

ASS and heparin administration, stress ECG in acute coronary syndrome

Furthermore, all patients receive ASS (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 a lysis therapy impossible. During unstable angina pectoris, which can only be safely distinguished from an infarct after six 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 non-ST-segment elevation myocardial infarction

In case of non-ST-segment elevation myocardial infarction, a cardiac catheterization must be conducted, with the time frame for the implementation ranging from 2 to 72 hours, depending on the risk profile of the patient. In case of ST-segment elevation myocardial infarction, the obstructed coronary vessel should be revascularized as quickly as possible by means of PCI.

Complications of Acute Coronary Syndrome

The complications of acute coronary syndrome are complex; their severity depends on the extent of the myocardial injury. Depending on the damaged area, right or left ventricular failure or, in case of a damaged papillary muscle, an 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 the cardiogenic shock, there is the threat of the rupture of a potential myocardial aneurysm. Also, a myocardial rupture is possible. As a result of infarction, a pericarditis might still occur over the following 6 weeks.

Prevention of Acute Coronary Syndrome

As 95 % of the cases of acute coronary syndrome are manifestations of coronary heart disease, the goal is to avoid the latter. For the prevention of coronary heart disease, it is advised to eliminate the risk factors, especially the main risk factors like cholesterol, arterial hypertension, diabetes mellitus, and smoking. The approaches are predominantly concerned with the patient’s behavior, for examples such as nutrition and sporty behavior. These approaches can also be assisted by medication.

Review Questions

The correct answers can be found below the references.

1. Which of the following is not a part of the pathological diagnosis in any stage of acute coronary syndrome?
   A. A hemorrhagic edge
   B. Leukocytes
   C. Yellowish Spots
   D. Whitish scar tissue
   E. Newly emerging myocytes

2. Which of the following symptoms is least typical of acute coronary syndrome?
   A. Pain radiating into the left arm.
   B. Pain radiating into the right arm.
   C. Pain that is relieved under administration of nitroglycerin.
   D. Pain that remains constant when resting.
   E. Pain radiating into the upper abdomen.

3. What is the Pardée-Q most likely associated with?
A. The initial stage of the infarct.
B. An acute infarct.
C. A non-ST segment elevation myocardial infarction.
D. The unstable angina pectoris.
E. An infarction dating back longer in time.

References
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Duale Reihe: Innere Medizin, 3. Auflage – Thieme 2013
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Correct answers: 1E, 2C, 3E

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