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ACUTE CORONARY SYNDROMES IN THE PRACTICE OF THE EMERGENCY MEDICAL TEAM

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ABSTRACT

An acute coronary syndrome (ACS) is described when myocardial ischemia occurs due to impeding blood flow through the coronary vessels, which in turn results in insufficient supply of oxygen. As a disease entity, it causes almost half of all deaths in Poland. The high mortality of ACS is primarily in the pre-hospital setting. The classification of acute coronary syndromes is based on criteria such as clinical symptoms, the nature and type of changes in the electrocardiogram, and indicators of myocardial necrosis. In the conditions of the emergency medical team, the division is based on the image of the ECG test. On this basis, ACS with elevation of the ST segment (STEMI) and without ST segment elevation (NSTEMI) are defined. Pre-hospital diagnosis of ACS includes an interview with the patient and a 12-lead ECG performed preferably in no more than 10 minutes since the first medical contact. The task of the emergency medical team is to quickly recognize, perform teletransmission to the interventional cardiology centre and implement pharmacological treatment, and then provide transport to the nearest centre of invasive cardiology, where the procedure should be performed to clear the blocked coronary artery. Therefore, it is important that the staff of medical emergency teams have appropriate knowledge and proper equipment for ambulances.

KEY WORDS: acute coronary syndrome, ischemic heart disease, myocardial infarction, emergency medical teams, pre-hospital treatment, STEMI, NSTEMI

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INTRODUCTION

Ischemic heart disease (IHD) is the single most common cause of death in the world, and its prevalence increases regularly, while in Europe, over the past three decades, there has been a general trend towards reducing mortality caused by this disease [1]. Statistics show that there are almost 1.8 million deaths annually caused by IHD, which is 20% of all deaths in Europe, but there is considerable variation in various European countries [2, 3]. Acute coronary syndromes are a group of clinical syndromes in which sudden myocardial ischemia occurs. These syndromes as part of cardiovascular disease (CVD), in recent years, according to the Central Statistical Office, were reported as the cause of almost half of all deaths in Poland. According to the sources of the Central Statistical Office, in 2015, coronary artery disease caused the death of 10.2/10,000 people, including acute myocardial infarction 3.6/10,000 inhabitants [4]. Despite the observed downward trend in the number of deaths as a result of CVD, mortality remains high and it should be noted that in contrast to a significant reduction in in-hospital mortality, out-of-hospital mortality remains relatively high [5]. Therefore, early diagnosis and implementing the appropriate treatment as soon as possible is extremely important. Emergency medical teams, as the first in contact with the patient, can start to perform

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the appropriate tasks, starting from the interview and performing the ECG, interpreting the record, recognizing ACS, teletransmission and giving appropriate pharmacology.

GENERAL DEFINITION AND CLASSIFICATION OF ACUTE CORONARY SYNDROMES

An acute coronary syndrome is recognized when myocardial ischemia occurs due to impeding blood flow through the coronary vessels, which in turn results in insufficient supply of oxygen [6–8]. When cardiomyocyte necrosis occurs as a result of myocardial ischemia, it is recognized as acute myocardial infarction (AMI). The concept of acute myocardial infarction should only be used in situations where evidence of myocardial injury has been obtained (defined as an increase in cardiac troponin concentrations ≥ 1 above the upper limit of normal values at the 99th percentile) with necrosis in a clinical situation corresponding to myocardial ischemia [9]. Taking into account the clinical symptoms, the nature and type of changes in the electrocardiogram and indicators of muscle necrosis, it can be distinguished:

- Elevation of the ST segment, where the clot completely and suddenly closes the lumen of the coronary artery. Myocardial necrosis begins to develop within 15–30 minutes after the blood flow stops and progresses from the subendocardium to the epicardium. The time of necrosis depends on the diameter of the closed vessel and collateral circulation.
- No ST segment elevation (NSTEMI, NSTE-ACS, non-ST elevation acute coronary syndrome). It often occurs as a consequence of unstable angina. The infarcted area usually has a fairly well developed collateral circulation or is small (i.e., provided by a distal segment of the coronary artery). The presence of Q waves [8, 10, 11] is also not observed.
- Unstable angina pectoris (UA), in which despite the characteristic clinical symptoms of myocardial ischemia, there are no electrocardiographic changes or an increase in the index of myocardial necrosis.
- Indefinite myocardial infarction, i.e. one in which the ECG was performed after 24 hours from the beginning of the symptoms and in the ECG itself there are such changes that cause difficulties in the diagnosis of ST segment elevation.
- Sudden cardiac death [6, 8].

In addition to the previously mentioned classifications, the differentiation of myocardial infarctions is also considered taking into account differences in pathogenesis, clinical picture and prognosis, as well as in various therapeutic procedures (Tab. 1).

Describing individual classifications of acute coronary syndromes with ST segment elevation the division of infarcts caused by the myocardial injury zone cannot be omitted. This is how the sharp failures located in the bottom wall, front wall, side wall and back wall can be distinguished (Tab. 2).

In the case of ST segment elevation in leads II, III and aVF, it is necessary to perform a modified ECG recording the leads on the right side of the chest. They get the name V1R, V2R, V3R, V4R, V5R, V6R. One should look for ST segment elevation at the J point in V3R and V4R leads by min. 0.05 mV (i.e. 0.5 mm) (in the case of men before 30 years of age 0.1 mV, i.e. 1 mm), which signifies a right ventricular infarction.

Table 1. Classification of myocardial infarction. Own study based on [10–12]

Classification of myocardial infarction	
Type I	Spontaneous myocardial infarction — associated with atherosclerotic plaque rupture and ulceration; erosion or dissection of the arterial wall, leading to the formation of a thrombus in the light of a few or one coronary arteries, which results in decreased blood flow through the myocardium or embolism of blood platelets and consequences in the form of necrosis of cardiomyocytes
Type II	Infarction as a result of inequalities between the supply of oxygen and the need for myocardial oxygen. Such inequality may occur due to a different reason than coronary artery disease. For example, contraction of the coronary artery, brady — or tachyarrhythmia, anemia, respiratory failure
Type III	Myocardial infarction is not confirmed by biomarker concentration
Type IVa	PCI-related stroke (percutaneous coronary intervention)
Type IVb	A heart attack associated with stent thrombosis
Type V	Myocardial infarction associated with CABG (coronary artery bypass graft)

Table 2. Infract location. Own study based on [13]			
I Side or front wall	**aVR**	**V1** Front wall	**V4** Front wall
II Lower wall	**aVL** Side or front wall	**V2** Front wall	**V5** Front wall
III Lower wall	**aVF** Lower wall	**V3** Front wall	**V6** Side or front wall

CAUSES OF ACS

There may be many reasons for the appearance of ACS, however the most frequent reason contributing to the occurrence of myocardial ischemia is the separation of an atherosclerotic plaque in the coronary arteries. Other causes include: coronary artery spasm, coronary artery embolism, coronary artery inflammation and changes in them resulting from disturbances in the course of metabolism, coronary vascular defects, coronary artery injury, etc. [8]

As a result of coronary risk factors, including hypercholesterolemia, diabetes, hypertension and smoking, large and medium vessels are damaged by progressive atherosclerotic processes. It involves the formation of internal vessels in the inner membrane, the so-called unstable atherosclerotic plaques. Their formation leads to the development of inflammatory states, and this initiates changes at the cellular level and predisposes to the formation of thrombi [7, 8].

Symptoms of ACS

The main symptom of ACS is increased chest pain, which lasts > 20 minutes. The pain in the course of a coronary episode has the character of a choking, pressure, strangling pain, most often located in substernal area and with radiation of pain to the left upper limb, neck and mandible. It can be a continuous pain or occur intermittently. In addition, you can observe non-specific symptoms such as localized pain in the upper abdomen instead of the chest, nausea, vomiting, fainting, paleness, sweating, tachycardia, cardiac arrest or an asymptomatic course. The group of patients at risk of non-specific symptoms include women, the elderly, diabetics or people after surgical procedures [6, 7, 9, 14–16].

Diagnostic criteria for ACS

Diagnosis of ACS is a multi-stage process. You should not confirm or exclude ACS based on just one symptom. The interview plays a key role in the proper assessment of the patient's condition. A correctly collected history can properly target and help you choose the right diagnostic and therapeutic path [7].

The next stage in the diagnosis of ACS is the fastest (preferably within 10 min of the first medical contact) performance and interpretation of a 12-lead resting ECG. To recognize ACS in the form of STEMI, the following criteria should be adopted [12, 14, 18]:

- ST segment elevation at point J in any lead except V2–V3 by a minimum of 0.1 mV (i.e.1 mm)
- ST segment elevation in V2-V3 leads with a minimum of:

1) 0.25 mV (i.e. 2.5 mm) for men before 40 years of age
2) 0.2 mV (i.e. 2 mm) for men over 40 years of age
3) 0.15 mV (i.e. 1.5 mm) for women regardless of age.

The criteria described above, however, do not apply to patients who may have features of left ventricular hypertrophy or left bundle branch block (LBBB). However, if you suspect acute myocardial ischemia and simultaneous presence of LBBB or RBBB (right bundle branch block), it is recommended to implement such procedures as in patients with ST segment elevation [14, 15].

In ACS NSTEMI, ECG changes can be very subtle or even completely absent. However, if they occur, they present as:

- ST segment depression
- Transient ST elevation and T-wave changes.

It should be remembered that a correct ECG recording is not a basis for exclusion of ACS. In a situation where symptoms continue or recur, it is useful to repeat the ECG recording or compare the current one with the previous ones [7, 10, 11, 15].

In pre-hospital situations, ACS diagnosis is based on the above diagnostic criteria. Therefore, it is important that the personnel of emergency medical teams are well trained, and ambulances are equipped with the appropriate equipment enabling rapid diagnostics, ECG and, if necessary, teletransmission and pharmacology [18]. The contemporary

role of the members of the emergency medical team should not only be limited to a quick determination of the initial diagnosis or ensuring an efficient patient transport with ACS to the hospital. Physicians and paramedics in their positions also play an extremely important role in the initiation of therapy for patients with ACS, especially antiplatelet therapy [6].

Pre-hospital treatment of ACS

The medical rescue team, after recognizing the symptoms of ACS and confirming them with the above diagnostic criteria, should transport the patient (semi-recumbent) to the nearest centre of invasive cardiology in the shortest possible time. The affected artery should be unblocked within 60 minutes from the first medical contact with the patient (this applies to ACS patients with STEMI and NSTEMI with high-risk symptoms such as hemodynamic instability, recurrent or refractory coronary pain, or life-threatening cardiac arrhythmias). In the case of patients with NSTEMI, the time that can elapse from the first medical contact to the implementation of invasive procedures in the hemodynamic laboratory is: > 24 hours in patients at high risk and > 72 hours in patients with intermediate risks e.g. diabetes, renal insufficiency [4, 7, 18]

The tasks of the emergency medical team include pain management, administration of nitrates, administration of antiplatelet drugs and appropriate oxygen therapy [4, 6, 13]:

- In the analgesic treatment of ACS, intravenous morphine is most often used, however, special attention should be drawn to patients with consciousness impairments or bradycardia. The administration of NSAIDs (non-steroidal anti-inflammatory drugs) should be discontinued in the case of ACS as they have a pro-thrombotic effect.
- Nitrates. The routine use of nitrates in STEMI in a randomized controlled clinical trial compared to placebo did not show benefit and is therefore not currently recommended [19, 20]. Intravenous nitrates may be useful in the acute phase in patients with hypertension or heart failure, however, under conditions of no hypotension, right ventricular infarction and absence of type 5 phosphodiesterase inhibitors in the previous 48 hours. Nitrates remain valuable drugs for long-term treatment in order to reduce residual angina symptoms.
- Antiplatelet therapy is an extremely important element of pre-hospital therapy and should be implemented almost immediately after confirmation of STEMI in the ECG. Antiplatelet therapy should include:

1) Administration of saturated doses of ASA (acetylsalicylic acid). The first dose should be 150–300 mg and be given as an easily absorbed uncoated tablet, then 75–100 mg/day
2) The second drug is the P2Y12 inhibitor. The recommended preparation is ticagrelor (at a dose of 180 mg) or clopidogrel (at a dose of 300–600 mg) [5, 19].

- Oxygen therapy in ACS is currently not recommended as a standard treatment for any patient with ACS because it has been shown that excess oxygen can be detrimental to the patient and can increase the area of myocardial injury. It is recommended to use oxygen therapy in patients with significant hypoxemia (SpO2 < 90%) [5, 15, 19, 20].

In addition, it is important to constantly monitor the patient's heart rhythm, and if ventricular fibrillation occurs — initiate a resuscitation algorithm [8].

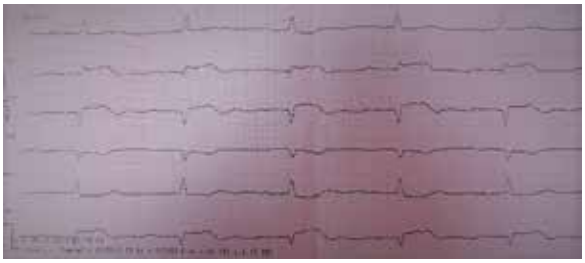

FIGURE 1. STEMI lower wall. Limb leads. Author's own material

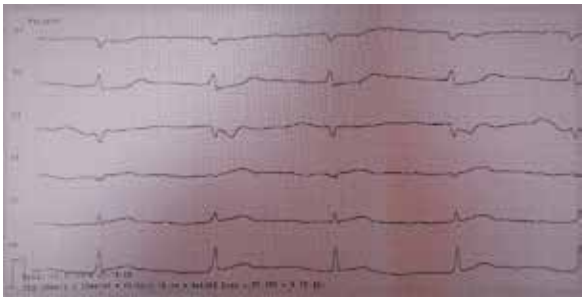

FIGURE 2. STEMI lower wall. Precordial leads. Author's own material

SUMMARY

A well-developed emergency notification system as well as trained and well-equipped emergency medical team are crucial in the emergency response to a patient with chest pain. It is therefore reasonable

that all members of emergency medical teams are trained in the diagnosis of AMI symptoms, administration of oxygen if necessary, pain control as well as the use of advanced resuscitation activities [21]. Rapid pre-hospital diagnosis and diagnosis of STEMI as well as appropriate treatment and prompt transport for rescue PCI have undoubtedly a significant impact on the reduction of patients' mortality [22].

Diagnostics and therapy of patients with suspected ACS are a challenge at every stage of the procedure. It is believed that properly trained medical staff and the use of diagnostic and therapeutic algorithms can contribute to therapeutic success, and hence, reduction of mortality in patients with ACS during the first contact of the patient with a medical professional. The use of the ECG teletransmission system and teleconsultation by emergency medical teams should be standard treatment in all patients with suspected ACS [23], because the use of consultation capabilities facilitates the rapid diagnosis of STEMI and NSTEMI-ACS, allowing early implementation of DAPT in all patients who require it. In addition, it improves logistics and reduces the delay of revascularization treatment [5]. The education of the society and appropriate prevention are also important.

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