Myocardial infarction of interior wall: a case study

Zawał mięśnia sercowego ściany dolnej – opis przypadku

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Słowa kluczowe: zawał mięśnia sercowego, wywiad SAMPLE, schemat MONA.

Abstract

Cardiovascular diseases are the primary death factors of people in the world. Myocardial infarctions and strokes are the most predominant among them. Securing a patient with myocardial infarction requires a rapid pre-hospital procedure and a fast cardiac intervention at an invasive cardiology centre. The paper describes a case of a 55-year-old man diagnosed with acute coronary syndrome with ST-segment elevation myocardial infarction (STEMI), i.e. myocardial infarction of the bottom wall. The operative procedure requires following the MONA algorithm (M – morphine, O – oxygen, N – nitroglycerin, A – aspirin). The process of data tele-transmission is an important element of the pre-hospital proceedings at the level of Medical Emergency Team. It makes it possible to send quickly the ECG record from the ambulance or patient's home to a cardiology centre.

Streszczenie

Choroby układu krążenia stanowią główną przyczynę zgonów ludzi na świecie. Wśród nich najczęściej przeważają zawały mięśnia sercowego i udary mózgu. Pacjent z zawałem mięśnia sercowego wymaga szybkiego postępowania przedszpitalnego oraz szybkiej interwencji kardiologicznej w ośrodku kardiologii inwazyjnej. W artykule przedstawiono przypadek 55-letniego mężczyzny, u którego rozpoznano ostry zespół wieńcowy z uniesieniem odcinka ST, czyli zawał mięśnia sercowego, w tym przypadku ściany dolnej. Obowiązującym schematem jest postępowanie według algorytmu MONA (M – morfina, O – tlen, N – nitrogliceryna, A – kwas acetylosalicylowy). Ważnym elementem w postępowaniu przedszpitalnym na poziomie ZRM (Zespół Ratownictwa Medycznego) okazuje się proces teletransmisji danych, który umożliwia szybkie przesłanie zapisu EKG z ambulansu lub domu pacjenta do ośrodka kardiologicznego.

Introduction

The typical symptoms of heart attack include chest pain, usually retrosternal, dyspnoea, sweating, anxiety, and nausea. The pain may radiate to the lower jaw, back, shoulders, and epigastrium. An important criterion, suggesting cardiac infarction as the origin of pain, is its duration, which is usually not shorter than 20 min. It often lasts several hours. About 25% of heart attacks give unusual, minor ailments or proceed without any pain. Most frequently, painless infarction occurs in patients suffering from diabetes with diabetic neuropathy, which is responsible for their reduced perception of pain. It happens that a heart attack is manifested in the most dramatic way – cardiac arrest and sudden death [1].

The majority of people who survive the first 2 h of infarction have a chance of recovery. However, you have to account for complications. It is estimated that 7–12% of infarction patients die during the hospitalisation period [1].

Cardiogenic shock is the primary cause of death in patients hospitalised due to fresh myocardial infarction. At the same time, about 80% of cases include infarction that is not complicated by any additional factors, but it simply covers at least 40% of the left ventricle area. These additional, rare complications of myocardial infarction leading to cardiogenic shock include rupture of the heart muscle (free wall, interventricular septum, papillary muscle) or right ventricular infarction [2].

Despite the progress in the treatment of acute myocardial infarction, including advanced invasive cardiology and cardiac surgery, 40–60% of patients die within 1 month due to the complication of infarction, even when they are given invasive therapy including transdermal coronary intervention. Before the era of reperfusion treatment of infarction with ST-segment elevation (STEMI) cardiogenic shock occurred in approximately 20% of patients with STEMI, now it involves about 7–9% of patients [3].

As "time is muscle", patients with suspected myocardial infarction must be diagnosed immediately. The time from their admission to the emergency ward to the diagnosis and initiation of treatment should be minimised [4].

A preliminary evaluation of the patient and physical examination involves the implementation of the ABC scheme: A – unobstructed airways, B – breathing for 10 s – seeing, feeling, hearing, C – rating circulation on the carotid artery, evaluation of skin and capillary relapse. A SAMPLE interview is also carried out: S – symptoms, A – allergies, M – medications, P – previous illnesses, L – last meal, and E – other ailments. It is standard procedure to apply the MONA algorithm during the acute coronary syndrome (ACS) with the STEMI: M – morphine, O – oxygen, N – nitroglycerin, and A – aspirin.

At this stage, there occurs the concept of the golden hour in the pre-hospital care. It is the time from the appearance of the symptoms and the call for help to the moment when the patient reaches the hospital [5].

Case report

Dispatch

At 5.38 a.m. the unit of the Emergency Medical Service in Kielce received a notification about a 55-year-old man who reported severe pain in the chest. The Medical Rescue Team S (Specialised) was ordered to the incident. The team consisted of: a doctor-surgeon, a paramedic, and a paramedic-driver. The S team departed for the indicated address in the alarm mode K1 (with the siren on). The arrival time was 3 min.

Arrival at the site

At 5.43 a.m. the team reached the address, which was a detached house. The team members along with the equipment (life-saving kit, ampoule bag, oxygen therapy set, resuscitation kit, defibrillator) headed for the patient's house. Inside they found a man lying flat on his back on the bed. The doctor beg an the initial assessment of the patient and physical examination.

Subjective examination

The patient reported intensification of pain in the chest. The pain extended to the shoulders and throat, and he felt pain in his stomach. A detailed interview according to the SAMPLE scheme: S – pain in the chest giving a sensation of choking and pressure for about an hour; A – no allergies in the interview; M – no steady drugs – about an hour ago when the first pain symptoms appeared the man took one tablet of propranolol; P – no information about past diseases history; L – supper the previous night; and E – pain extending to the left arm, chronic smoking.

Objective examination

The patient was pale, skin cold, sweaty, sticky. His blood pressure was 90/70 mm Hg, pulse oximetry (sPO_2) 90%, standard glycaemia, respiratory rate 18/min, heart rate 50 beats/min, capillary relapse more than 2 s, and Glasgow Coma Scale 15 points. His pupils were equal and reacted properly to light, he had correct auscultatory murmur, and his heart sounded clean and loud.

Recommendations were as follows: instant oxygen therapy using a Venturi oxygen mask with a reservoir in the flow of 10 l/min of 100% oxygen, continuously checking pulse oximetry, bearing in mind that the saturation values with the application of oxygen should not exceed 94–95% (European Resuscitation Council Guidelines of 2010).

In ECG: in a rapid assessment after an analysis of the recording, the team noticed changes in leads II and III (Figure 1). After the 12-lead ECG the following record was obtained (Figure 2).

Diagnosis

On the basis of the total clinical picture, ACS with STEMI, i.e. inferior myocardial infarction, was diagnosed in this case. The patient was in the initial phase of cardiogenic shock. In order to exclude myocardial infarction of the right ventricle the electrodes were clasped on the right side of the chest. The record did

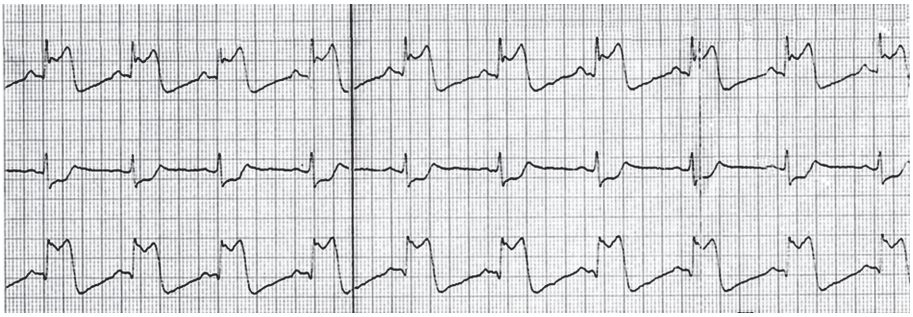

Figure 1. Record ECG. Changes in leads: II and III

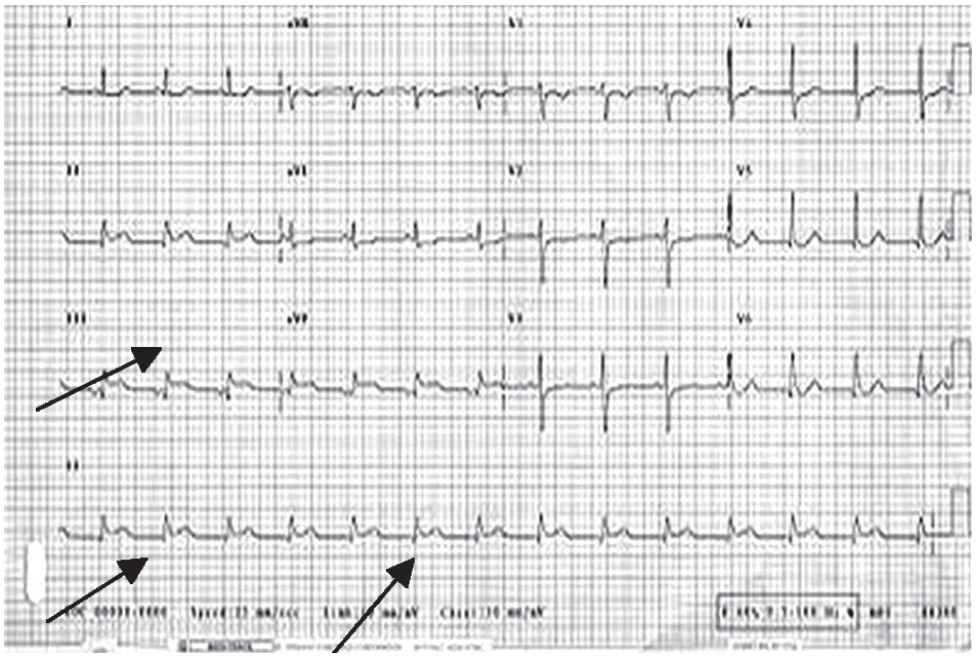

Figure 2. Record of the 12-lead ECG showing inferior myocardial infarction. ST- segment elevations of the Pardee wave type are present in leads II, III and aVF

not prove any changes corresponding to the right-ventricular myocardial infarction.

Procedure

Immediately the patient received initial protection. Two large intravenous approaches (*i.v.*) cannula 1.8 G were obtained, fluid therapy was started and transfusion of natrium chloratum (NaCl). Next, data was tele-transmitted to the nearest invasive cardiology centre, which was 30 km away. Air transport Helicopter Emergency Medical Service (HEMS) of the patient was not available because of the early hour and unfavourable weather conditions.

The patient was administered the following: 300 mg ASA (aspirin) per os crushed in the teeth, morphine in fractioned doses starting with 2 mg *i.v.* dissolved in 0.9% NaCl and 5000 units of low molecular weight heparin dissolved in 0.9% NaCl.

The patient was also evaluated on a numerical scale 1–10 in terms of pain, from the weakest to the strongest pain. The patient described his discomfort at level 8 before being given morphine.

Transport

The patient was secured for the time of carrying him in a cardiac chair into the ambulance with a continuous oxygen therapy. In the ambulance the patient was on a stretcher in a half-seated position. The doctor re-evaluated the patient's condition according to the ABCDE scheme: A – airway, B – breathing for 10 s – hearing, seeing and feeling – about 18–20 breaths/min, C – circulation – comparing central and peripheral pulse, auscultating again the sound of heart, and checking the monitor and pulse oximetry – 50 beats/min, assessing skin and capillary recurrence.

Continuous monitoring of the patient greatly highlighted changes in leads II, III, and aVF in the form of ST segment elevation. During the transport the patient's pain in the chest intensified. The total dose of morphine administered during the presence of the patient at home and during the whole transport was 10 mg. After reaching the cardiac centre an on-site cardiac team took over the patient and instantly, after initial laboratory and diagnostic investigation, began an urgent invasive procedure – coronarography. The condition of the patient was identified as severe.

Discussion

Most patients with ACS die before reaching the hospital. These are usually sudden deaths, meeting the criteria of sudden cardiac death, or death within an hour of the onset of the symptoms. The basic mechanism killing the patients is ventricular fibrillation; in 85% of cases it is the direct cause of sudden death.

It is not easy to estimate the exact magnitude of the phenomenon of death during pre-hospital care in patients with ACS. The confirmation of coronary atheromatosis as pathology causing sudden cardiac death requires a post-mortem examination or at least the possibility of obtaining a reliable interview as to the pre-existing coronary artery disease.

Nevertheless, such studies are performed. The results show that of all the people aged 35–64 years, who died of ACS within 28 days of the onset of symptoms, 2/3 of the patients died before reaching the hospital [6]. Direct treatment of sudden cardiac arrest requires immediate resuscitation by bystanders and calling for qualified emergency workers equipped with a defibrillator.

Minutes count here. With every minute of delay in performing defibrillation during ventricular fibrillation the chances of restoring circulation and saving the patient diminish. When the defibrillation takes place within 2 min of the cardiac arrest, it is possible to rescue almost 50% of patients – the term "rescue" should not be understood as a reversal of the arrhythmia and restoring haemodynamically effective heart beat, but as bringing the patient to hospital discharge and survival of at least 1 month. When defibrillation is delayed for more than 20 min only 2% of patients survive [7].

Both the diagnosis and treatment of fresh myocardial infarction starts with the moment of the first medical contact. The first step is to determine a preliminary diagnosis, usually on the basis of chest pain lasting for at least 20 min and not stopping after taking nitroglycerin. An important indication of myocardial infarction is coronary artery disease (CAD) in an interview, and radiation of pain to the neck, lower jaw, or left arm. The pain may not be strong. In some patients (up to 30%) with fresh STEMI the symptoms are less typical, such as nausea, vomiting, dyspnoea, fatigue, palpitation, and syncope [8].

The occurrence of bradycardia is particularly frequent in the case of inferior myocardial infarction. It is probably associated with an increase in vagus nerve tension and ischaemia of the sinus node. It occurs in 9–25% of all the patients within the first hour of acute myocardial infarction. It does not worsen the prognosis and does not require treatment if the frequency QRS is greater than 40/min.

Inferior wall myocardial infarction: first-degree AV block, second-degree AV block, Mobitz type 1 block.

It occurs more frequently in the case of patients with inferior myocardial infarction rather than anterior myocardial infarction. It is associated with the closure of the right coronary artery (RCA) and directly caused by ischemia atrioventricular node (AV node), transitory (abates within 72 h), sometimes it is transformed into a complete heart block. The patients do not require special treatment if the frequency of QRS does not fall below 50/min, there is no LBBB or cardiac insufficiency.

Inferior wall myocardial infarction: an advanced AV block (Mobitz type second-degree and third-degree AV block): It generally occurs together with inferior myocardial infarction with atrioventricular block, a post-mortem examination: acute necrosis of prenatal myocardium, in the absence of changes in the conducting system of the heart, often ischemia/infarction of the AV node secondary to hypoperfusion within the AV node artery. It is characterized by high mortality and high risk of complications: pericarditis, AF, VF, LK failure [9].

Pre-hospital procedure in the case of bradycardia. Evaluation of the patient according to the ABC scheme, oxygen therapy, providing intravenous access, monitoring ECG, blood pressure, SpO, performing 12-lead ECG.

The diagnosis and treatment of reversible causes (e.g. electrolyte disturbances). It should be assessed whether there are critical symptoms such as shock, syncope, myocardial ischaemia, or heart failure. If they are present, atropine 500 μg *i.v.* should be given. If there is no response, atropine 500 μg *i.v.* up to a maximum dose of 3 mg, isoprenaline 5 μg/min, and adrenaline 2–10 μg/min should be given.

Alternative medications (aminophylline, dopamine, glucagon when β-blockers and calcium channel blockers overdosed, and glycopyrolate) can be used instead of atropine [10]. Continuous bradycardia at 50 beats per minute may be the result of taking propranolol, which, as a medication from the group of β-blockers, will slow down the already failing cardiac muscle due to acute coronary syndrome and will decrease, as a consequence, the blood pressure fall, especially systolic.

Certain demographic and clinical features (advanced age, heart failure – II–IV Killip classes, three-vessel disease, anterior myocardial infarction, long-lasting ischaemia, and reduced TIMI flow) increase the risk of complications. It may be necessary to monitor more closely the condition of the patient if these features are present. Mechanical complications of myocardial infarction may occur suddenly during the first days after STEMI. They occur less frequently in the case of rapid and effective reperfusion. All of these conditions are life threatening, so they must be instantly diagnosed and treated [11].

Complications of STEMI: acute mitral insufficiency: the treatment is to reduce afterload using intravenous diuretics, vasodilators, and inotropic positive drugs. The symptoms include protosystolic murmur, refractory cardiogenic shock, and respiratory failure. This condition has very high mortality. The treatment involves urgent surgical intervention (mitral valve replacement) [12].

Ventricular septum rupture: intravenous diuretics and vasodilators should be applied with caution in the case of hypotension. Urgent surgical treatment is inevitable, but there is no consensus on the best time to perform the operation.

Right ventricular myocardial infarction: this can be isolated, but more frequently it accompanies inferior myocardial infarction. Despite the jugular vein distension, fluids should be transfused in order to

maintain right ventricular filling pressure. Diuretics and vasodilators should not be used as they intensify hypotension. Appropriate treatment should be applied in cases of atrial fibrillation or atrioventricular block.

Accompanying right ventricular infarction occurs in approximately 30–50% of inferior myocardial infarctions. There are a triad of clinical symptoms: low arterial blood pressure (ABP), clean lung fields, and raised pressure in the pulmonary veins in patients with inferior wall STEMI (high specificity, low sensitivity of symptoms), frequent conduction abnormalities (sinus bradycardia, atrioventricular blocks) and/or supraventricular and ventricular arrhythmia (often atrial fibrillation).

The diagnosis of right ventricular infarction is important as it can manifest as cardiogenic shock, and the correct treatment strategy is completely different from that applied in a shock caused by left ventricular dysfunction. In each case of diagnosing inferior myocardial infarction a routine record of right ventricular leads (at least V4R) should be conducted as soon as possible [13, 14].

Conclusions

It is standard procedure to follow the MONA scheme in ACS. In the presented case inferior wall myocardial infarction and cardiogenic shock were the contraindications to implement the full MONA scheme. The S team withdrew from administering nitroglycerin to the patient, which is a coronary vasodilator and thus lowers blood pressure.

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