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CASE REPORT

ECG FINDINGS IN PULMONARY EMBOLISM OR THE PSEUDOINFARCTION PATTERN

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ABSTRACT

Introduction/Objective: A diagnosis of STEMI can be made for patients with persistent chest discomfort or other symptoms of ischemia and an ST-segment elevation in at least two adjoining ECG leads. The differential diagnosis of ST-segment elevation consists of 4 major conditions: myocardial infarction, pericarditis, early repolarization syndrome, secondary changes and other diagnoses, such as pulmonary embolism (PE). The dilemma is whether an ST-elevation always requires the PPCI protocol to be activated? The objective of this article is to acquaint medical doctors with other similar clinical presentations and the importance of making the right decision that directly affects the course of treatment and the outcome for the patient.

Case report: An 84-year-old woman called the EMS hotline complaining of chest pain and breathing difficulty. She was found in aprone position on a flat surface, pale and covered in sweat, but without chest pain at the ti. She had had hip replacement surgery and was discharged from the hospital two days earlier. She was currently receiving 0.4ml of Fraxiparine daily. Her blood pressure was 90/60mmHg, heart rate 115 per minute, breathing frequency 20 breaths per minute, blood oxygen saturation 70%. Upon auscultation, her heart sounds were softer, breathing was normal with no wheezing or crackles. ECG: ST-segment elevation in leads II, III and avF and reciprocal ST-segment depression in leads I, avL and V2-V6 and an S1Q3T3 pattern.

Conclusion: There are many articles describing cases where the PPCI protocol had been activated for patients with similar ECG findings and who had been subjected to emergency coronary angiography. Since the coronary angiography findings had been normal and they were transferred for further diagnostics. Coronary angiography per se will not harm these patients, but delaying adequate treatment for pulmonary embolism could have a lethal effect. This is why it is very important to record serial ECG tracings. Rapid regression of ECG findings and Q-wave regression favorises pulmonary embolism.

Keywords: ECG findings, pulmonary embolism, pseudoinfarction pattern

Introduction

It is an established practice to diagnose patients suffering from persistent chest discomfort or other symptoms of ischemia coupled with an ST-segment elevation in at least two adjoining ECG leads with STEMI [1]. The differential diagnosis of ST-segment elevation consists of 4 major conditions: myocardial infarction, pericarditis, early repolarization syndrome and secondary changes. Other possible diagnoses are hyperkalemia, pulmonary embolism (PE) and Brugada syndrome [2]. ECG findings in pulmonary embolism tend to vary extensively, from sinus tachycardia, which is the most common finding, through right axis deviation, complete or incomplete right bundle branch block, T-wave inversion, S1Q3T3 pattern and ST-elevation as the least common ECG finding [3].

The pseudoinfarction pattern is defined as an abnormality of the ST-segment and T-wave coupled with pronounced Q-waves, simulating an inferior myocardial infarction [4] with a newly developed Q-wave, usually in leads III and avF. Between 16% and 24% of the patients develop an ST-segment elevation, but not as pronounced as it is in inferior myocardial infarction. Although the T-wave is more commonly negative in lead III than in avF, patients who have a history of inferior myocardial infarction are a differential diagnostic problem [4]. Approximately 11% of the patients with massive or submassive pulmonary embolism have this pattern with no history of heart disease. Most commonly, in cases of PE, ST-segment elevation is present in inferior leads, but it can also be found in anteroseptal leads [4].

Objective

To present differential diagnostic dilemmas in ST-elevation pattern interpretation and how to make the right decisions about prehospital treatment.

Case report

The Emergency Medical Service (EMS) intervened in the case of an 84-year-old woman who had a sudden onset of chest pain lasting longer than 30 minutes and difficulty breathing. She was found in a prone position on a flat surface, extremely pale and covered in sweat, without chest pain at the time, denying difficulty in breathing and nausea. She declared that she had had hip replacement surgery nine days ago and had

been discharged from the hospital two days ago. She was receiving 0.4ml of Fraxiparine daily in her home. She denied having hereditary diseases and has not been prescribed therapy for any form of heart disease. Upon examination, she was conscious, oriented, afebrile, tachycardic (120 beats per minute), hypotensive (90/60mmHg), mildly tachypnoeic (20 breaths per minute) with blood oxygen saturation of 70% on room air. Upon auscultation, her heart was in rhythm with softer sounds and no murmurs. Her breathing was normal with no wheezing or crackles. Her abdomen was soft with no tenderness or organomegaly. There was no pretibial oedema on her legs.

ECG findings: sinus tachycardia up to 120 beats per minute, pronounced ST-segment elevation in leads II, III and avF with reciprocal ST-segment depression in leads I, avL and V2-V6, as well as the S1Q3T3 pattern (**Figure 1**).

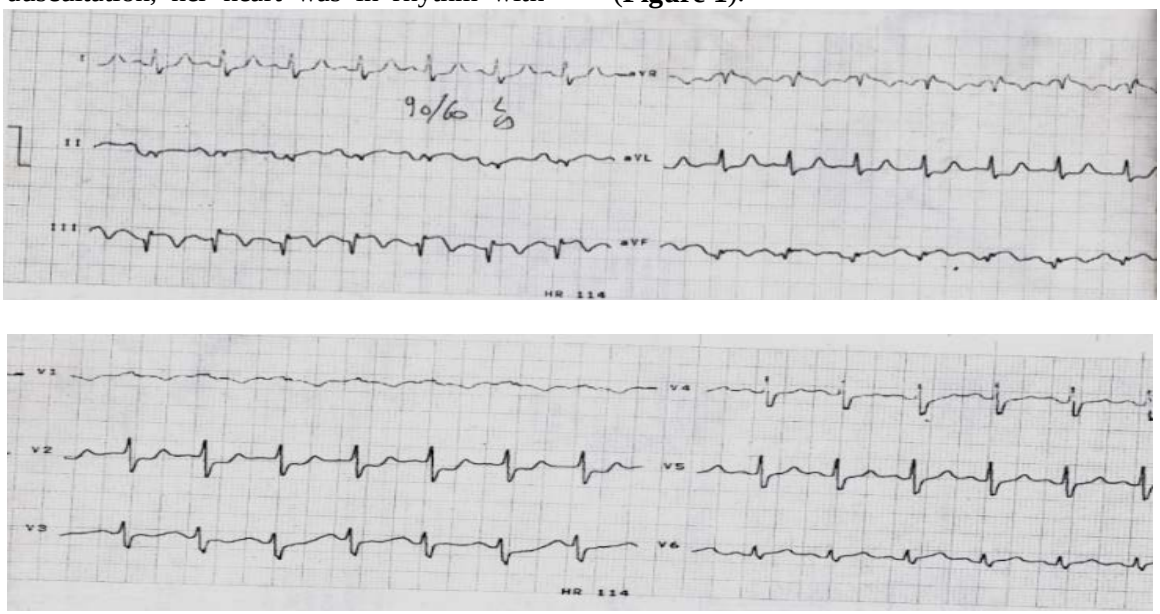

Figure 1. ECG tracing

The patient was immediately asked to chew up a 300mg tablet of Aspirin, an IV line was inserted and a sodium chloride 0.9% IV solution was administered as she was being prepared for transport. The Wells score was towards the top of moderate risk for developing pulmonary embolism, while ST-segment elevations in inferior leads larger than 3mm suggested a myocardial infarction. Under the diagnosis of a suspected inferior myocardial infarction and/or pulmonary embolism, she was transported to the University Hospital Medical Centre „Bezanijska Kosa“ for further evaluation. Her vital signs and ECG were continually monitored during transport and she was receiving oxygen by mask (2 litres per minute). Additional diagnostic procedures were performed at the hospital: chest X-ray, echocardiogram, Doppler ultrasound of the lower extremities, abdominal ultrasound, MDCT pulmonary angiography and lab work.

Chest X-ray: in a prone position, AP view – normal findings.

Echocardiogram: a poor echocardiography window due to low mobility as a result of the hip replacement surgery, she was unable to assume the left lateral decubitus position. The aortic root measured 3,5 cm, the right ventricle was marginally dilated and measured 3.7cm in apical view. The tricuspid function was normal with moderate tricuspid regurgitation, estimated end-diastolic pressure in the right ventricle was 47mmHg. The left ventricle was of normal size. The septum, the posterior and lateral walls of the left ventricle were visible and had no regional wall motion abnormalities, but the anterior and inferior walls were not visible and their wall motion could not be estimated.

Lower extremities Doppler ultrasound: no signs of acute thrombosis of the veins.

Abdominal ultrasound: normal findings with no free fluid in the abdomen.

Lab work: **sedimentation rate** 16.6 (women: 4-24 mm per hour, > 50 years of age); **WBC 13,6** (3,9-10x10^{9}); **Hgb 113...,78...(**110-180g/l); **RBC 3,58** (3,86-5,08x10^{12} for women); **Hct 0,336** (0,356-0,470 l/l); MCV 93,9 (81-99 fl); MCH 31,6 (29-32,9pg); MCHC 336 (310-350 g/l), **Glucose 14,5mmol/l** (3,5-6,1 mmol/l); **d-dimer 8.822 ng/ml** (normal range below 500) and **troponin HS TNT 554 ng/l** (normal range below 14); **LDH 565 (**220 - 460 U/L**);** CK 72 **(for women 26-192 U/L)**.

MDCT pulmonary angiography: the examination was performed according to the protocol for angiography as a life-saving measure before urea and creatinine results were obtained. The results showed massive pulmonary embolism, with a thrombus located in the left pulmonary artery as well as lobar, segmental and subsegmental arteries on both sides. Once the pulmonary embolism diagnosis had been confirmed, the patient was treated with thrombolytic therapy. The anaemia diagnosed in the lab work was corrected with one unit of packed red blood cells. She was discharged 10 days later, having achieved the expected recovery.

Discussion

Pulmonary embolism is a common and potentially fatal form of a disease that occurs when a thrombus originating from the venous system occludes one or more branches of the pulmonary artery [5]. Venous thromboembolism (VTE), with a clinical manifestation of either deep vein thrombosis or pulmonary embolism, is globally the third most common acute cardiovascular syndrome, behind myocardial infarction and stroke. The yearly incidence rate for pulmonary embolism is between 39-115 per 100 000 people. The incidence of VTE is almost eight times higher in patients 80 years or older than in patients in their forties [6]. Pulmonary embolism with signs of ST-segment elevation is a clinical rarity, described only in case reports. [7]. In a paper published in 2019, 34 case reports have been identified, most of which were men with a mean age of 56.5 ± 15.5 years. Most of the patients presented with dyspnoea (76,4%), chest pain (63,6%) and tachycardia (71,4%) [7]. The patient we presented is a rarity based both on sex and age. She complained of chest pain and difficulty in breathing when making the call to the EMS only to deny both those symptoms once the medical team arrived. Tachycardia, which is one of the most common pulmonary embolism clinical signs, was present both on the original ECG tracing done in the field, on the ECG monitor during transport and on the ECG tracing recorded at the hospital, upon arrival [7]. An interesting article by Mitic and associates states that syncope is a dominant symptom of pulmonary embolism [8].

According to Acute Coronary Syndrome Guidelines published in 2017 [9], treating patients with ST-segment elevation, due to its enormous epidemiological impact, is to begin immediately upon making the first medical contact and a working diagnosis of STEMI based on persistent chest pain and a 12-lead ECG. Reperfusion strategy guidelines promote primary percutaneous coronary intervention (PPCI) as the treatment of choice during the first 120 minutes from the onset of symptoms. In Serbia, as in most European countries, a STEMI network has been established to enable quick and adequate treatment of these patients [1]. The Belgrade Emergency Medical Service is a part of the STEMI network. Our patient satisfied the criteria for emergency PPCI: chest pain lasting longer than 20 minutes, ST-segment elevation in leads II, III and avF, reciprocal ST-segment depression in leads I, avL and V2-V6. In addition to that, the ECG tracing showed the S1Q3T3 pattern, which is a presentation of an acute right heart failure regardless of its cause (acute bronchospasm, pulmonary embolism, pneumothorax, acute lung disease etc.) [10]. The incidence of the S1Q3T3 pattern in pulmonary embolism varies between 12-50%. Therefore, even though it may not be sensitive or specific enough, it is still considered to be one of the most important ECG signs for making the diagnosis of pulmonary embolism [10]. The diagnostic dilemma of „what to do?“ is encountered when the patient has clinical signs and ECG patterns of both pulmonary embolism and acute coronary syndrome, as in our patient's case. According to STEMI protocols, such patients should be transported directly into the closest available cath lab [1]. Authors from Slovenia have described back in 2010 a case of a patient with the pseudoinfarction pattern, who had with no additional diagnostic procedures (transoesophageal /transthoracic ultrasound) been transported from a local hospital to a university medical centre with a cath lab, which was standard treatment at the time. Coronography results came back normal and the subsequently performed transoesophageal ultrasound showed signs of PE, which was then treated. It is important to add that the presented patient had been at all times hemodynamically stable [11]. However, if pulmonary embolism is not considered as a differential diagnostic possibility, patients could end up having needless PPCI based solely on ST-segment elevation. This delays the correct diagnosis and adequate treatment, which could lead to a less favourable outcome, especially in patients who are not stable [12]. As a result of diagnostic uncertainties, our patient was transported to the Emergency Room at the University Hospital Medical Centre „Bezanijska Kosa“, which does have a cath lab. Upon arrival, basic lab work (d-dimer and troponin) and a transthoracic ultrasound were performed. In the case report by Mistry and associates, a patient similar to ours with ST-segment elevation in inferior leads and the S1Q3T3 pattern also had an ultrasound performed and troponin analyzed at the hospital. This patient had a high troponin level, but due to the onset of sudden hemodynamic instability, had an emergency transthoracic ultrasound which uncovered acute pulmonary thromboembolism [12]. Similarly, in our case, lab work did not paint a clear picture because both the d-dimer (8.822 ng/ml, normal value below 500) and troponin HS TNT (554 ng/l, normal value below 14) values came back very high. Since the patient could not assume the left lateral decubitus position because of the hip replacement surgery and therefore present the inferior heart wall, the heart echo could not exclude the possibility of an inferior wall myocardial infarction. Based on anatomic characteristics, it is expected that a patient with an inferior wall myocardial infarction presents with bradycardia, rather than tachycardia. The affected blood vessel, the posterior descending artery (the culprit lesion) is in the majority of the patients a branch of the right coronary artery (right-dominant circulation) and in only a minority the branch of the circumflex artery, which originates from the left coronary artery (left-dominant circulation). The right coronary artery also supplies blood to the AV node, which is why inferior wall myocardial infarction is often associated with bradycardia, heart block and arrhythmia [13]. Due to pronounced anaemia (Hgb 113, RBC 78, 14,5; Hct 0,336; MCV 93,9; MCH 31,6; MCHC 336) in our patient, regardless of the statistically significant predominance of

right-dominant circulation, it is expected to have tachycardia as a compensatory mechanism to protect the body from hypoxia [14]. However, none of this can neither point to the diagnosis of pulmonary embolism nor rule out myocardial infarction. Therefore, because of the patient's deteriorating condition, an emergency MDCT pulmonary angiography was performed, which came back positive for massive pulmonary embolism affecting the left main branch of the pulmonary artery, as well as lobar, segmental and subsegmental branches on both sides. Once the pulmonary embolism diagnosis had been confirmed, the patient was treated with thrombolytic therapy. The anaemia diagnosed in the lab work was corrected with one unit of packed red blood cells. She was discharged 10 days later, having achieved the expected recovery.

Conclusion

The „heart team" guidelines for activating the PPCI protocol are clear. Problems occur when clinical presentations get mixed up, as in our case. There are many articles describing cases where the PPCI protocol had been activated for patients with similar ECG findings and who had been subjected to emergency coronary angiography. Since the coronary angiography findings had been normal and they were transferred for further diagnostics.

Coronary angiography per se will not harm hemodynamically stable patients with pseudoinfarction patterns, but delaying adequate treatment for hemodynamically unstable patients could have a lethal effect. Therefore, even with existing local „heart team" protocols, a type of revascularisation decision should be made independently for each patient. All patients should be transported attached to continuous ECG monitoring devices. Rapid regression of ECG findings and Q-wave regression favorises the diagnosis of PE.

Conflict of interest: None declared.

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PRIKAZ BOLESNIKA

EKG SLIKA PLUĆNE EMBOLIJE ILI PSEUDO INFARKTNOG OBRASCA

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SAŽETAK

Uvod/cilj: Dijagnozu STEMI postavljamo kod pacijenata sa perzistentnom nelagodnošću u grudima, ili drugim ishemijskim simptomima, i sa elevacijom ST segmenta, u najmanje dva susedna EKG odvoda. Diferencijalna dijagnoza ST elevacije obuhvata uz 4 velika stanja: IM, perikarditis, sindrom rane repolarizacije, sekundarne promene, i druge dijagnoze od kojih je jedna i plućna embolija (PE). Dilema je da li ST elevacija, uvek zahteva aktiviranje pPCI protokola? Cilj rada je upoznavanje lekara na postojanje udruženih kliničkih slika, i važnost pravilne odluke koja direktno utiče na lečenje i ishod.

Prikaz bolesnika: 84.-godišnja pacijentkinja, pozvala je hitnu medicinsku pomoć zbog bola u grudima i otežanog disanja. Zatečena da leži na ravnom, bleda i preznojena, ali trenutno bez bola u grudima. Operisala kuk, i pre dva dana otpuštena iz bolnice. Na terapiji fraksiparinom 0,4 ml/24h. TA 90/60mmHg, SF 115/min, RF je 20/min, SpO2 70%, srčana akcija ritmična, tonovi tmuliji, disanje vezikularno bez vizinga i pukota. EKG: ST elevacija u D2, D3, i AVF, sa recipročnom depresijom ST segmenta u D1, AVL, V2-6 odvodima, kao i S1Q3T3 obrazac.

Zaključak: U literaturi su pronađeni radovi, gde je kod pacijenta sa sličnim EKG zapisom aktiviran protokol za pPCI, i koji su završili u sali za kateterizaciju. Nalaz na koronarografiji je bio uredan, a pacijenti bi bili upućivani na dalju dijagnostiku. Koronarografija per se, neće nauditi ovim pacijentima, ali odlaganje terapije PE, može biti i kobno za pacijenta. Zato se izuzetan značaj pridaje serijskim EKG nalazima, u kojima nalaz rapidne regresije EKG promena, kao i gubitak Q zubca favorizuje PE.

Ključne reči: EKG nalaz, plućna embolija, pseudoinfarktni obrazac