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ATYPICAL ELECTROCARDIOGRAPHIC PRESENTATIONS OF MYOCARDIAL INFARCTION WITH ST ELEVATION – ST ELEVATION MYOCARDIAL INFARCTION EQUIVALENTS
ATIPIČNI ELEKTROKARDIOGRAFSKI PRIKAZI INFARKTA MIOKARDA SA ST ELEVACIJOM – EKVIVALENTI INFARKTA MIOKARDA SA ELEVACIJOM ST SEGMENATA

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Summary
Introduction. Some patients with clinical symptoms and signs of acute myocardial and coronary artery occlusion have atypical electrocardiographic presentations – ST elevation myocardial infarction equivalents. Rapid recognition of these patterns is imperative, because the condition requires prompt reperfusion therapy following actual guidelines.

De Winter pattern. Diagnostic criteria are: tall, prominent, symmetrical T-waves in the precordial leads, upsloping ST segment depression > 1 mm at the J-point in the precordial leads, absence of ST elevation in the precordial leads, ST segment elevation (0.5 mm – 1 mm) in aVR.

ST Elevation in aVR. Electrocardiographic criteria include ST segment elevation in aVR ≥ 1 mm, ST segment elevation in aVR ≥ V1, and diffuse ST segment depression in lateral leads. Wellens syndrome. Wellens syndrome describes deeply inverted or biphasic T-waves in leads V2 – V3, highly specific for significant stenosis of the left anterior descending artery. Posterior infarction. Posterior infarction is confirmed with ST segment depression ≥ 0.5 mm in leads V1 – 3 and ST segment elevation ≥ 0.5 mm in posterior leads (V7 – V9). Conclusion. There are many electrocardiographic patterns that physicians should promptly recognize as clinical myocardial infarction with ST segment elevation equivalents in order to perform urgent reperfusion therapy for better prognosis and survival in these patients.

Key words: Electrocardiography; ST Elevation Myocardial Infarction; Early Diagnosis; Signs and Symptoms; Reperfusion; Coronary Occlusion; Acute Coronary Syndrome; Bundle-Branch Block; Coronary Stenosis

Introduction
Acute myocardial infarction (MI) is a clinical manifestation of coronary artery disease which develops when a blood vessel is narrowed or occluded leading to irreversible myocardial ischemia [1]. The leading symptom that initiates the diagnostic and treatment cascade in patients with acute coronary

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In 2008, in their study, Robert J. de Winter and Wellens pointed out the importance of recognizing the equivalents of acute MI with ST elevation and need for emergency reperfusion therapy. After analyzing data obtained from the database of PCIs, ECG on the first contact with the patient, ECG before treatment, findings of coronary angiography, they described ECG pattern found in approximately 2% of patients with angiography proven anterior MI with occlusion of the anterior descending coronary artery [8]. Diagnostic criteria for de Winter pattern are: tall, prominent, symmetrically peaked T-waves in the precordial leads, upsloping ST segment depression > 1 mm at the J-point in the precordial leads, absence of ST elevation in the precordial leads, ST segment elevation (0.5 mm – 1 mm) in aVR. Normal STEMI morphology may precede or follow the de Winter pattern. Unlike the hyper-acute T-waves which occur within minutes of coronary artery occlusion and progress rapidly to classical ST elevation MI, STEMI pattern, de Winter T-waves are not transient findings and remain present in subsequent ECGs (Figure 1).

Verouden et al. found de Winter pattern in 35 out of 1890 patients who needed PCI on the proximal left descending artery (in about 2% of patients). Patients with de Winter pattern were younger, more often male and with a higher incidence of hypercholesterolemia compared to classic MI with ST elevation [9].

De Winter pattern

The available data suggest that this pattern has a high positive predictive value for acute coronary occlusion [10]. Many case reports confirmed the importance of early recognition of this STEMI equivalent and early reperfusion therapy [11–13].

ST elevation in aVR

Occlusion of the left main coronary artery is a clinical entity that results in the development of acute MI in the anterior or anterolateral region with cardiogenic shock and high mortality rate. Because of the high risk of potential complications and high mortality, early detection of this type of STEMI equivalent and urgent reperfusion is imperative and provides better outcomes [14, 15].
ECG criteria are: ST segment elevation in aVR ≥ 1 mm, ST segment elevation in aVR ≥ V1, and diffuse ST segment depression in lateral leads (sub-endocardial ischemia) [16] (Figure 2).

ST segment elevation in aVR is not always specific for the occlusion of the left main coronary artery. It can be indicative for: left main equivalent disease (significant disease of the left anterior descending and left circumflex artery), occlusion of the anterior descending artery, severe three vessel disease and diffuse transmural ischemia [17, 18]. The absence of ST segment elevation in aVR excludes a significant lesion in the left main coronary artery. American Heart Association/American College of Cardiology recommended this pattern to be read as “ischemia due to multivessel or left main coronary artery obstruction”. When the resting ECG reveals ST segment depression greater than 0.1 mV (1 mm) in 8 or more body surface leads coupled with ST segment elevation in aVR and/or V1, but is otherwise unremarkable, the automated interpretation should suggest ischemia due to multivessel or left main coronary artery obstruction [3].

Some authors argue that the term “occlusion of the left main coronary artery” is incorrect, firstly because most patients with criteria for this clinical entity have at least slight flow through the main trunk of the left coronary artery, and secondly, occlusion quickly leads to acute MI with ST segment elevation, cardiogenic shock and death [19]. The lack of specificity of this pattern is documented by Knotts et al., because only 43% patients with sub-endocardial ischemia (ST elevation in aVR and diffuse ST depression) who underwent angiography had significant left main stenosis or a triple vessel disease [20]. Nevertheless, current guidelines recommend early reperfusion strategy for patients presenting with this pattern [3, 6].

Wellens syndrome

Wellens syndrome was first described in the 1980s by de Zwaan, Wellens, et al, who identified specific T-wave changes in precordial leads in 14% to 18% of patients with unstable angina who, subsequently, developed a large anterior wall MI [21]. Some of risk factors for this syndrome are diabetes mellitus, family history of premature heart disease, hypertension, hypercholesterolemia and metabolic syndrome. Wellens syndrome describes ECG changes in T-wave, particularly deeply inverted or biphasic T-waves in leads V2 – V3 that is highly specific for significant proximal stenosis of the left anterior descending artery. Patients with these ECG patterns can be without subjective symptoms at the time of recording ECG and have normal or slightly elevated cardiac enzymes. They are at high risk for developing acute large anterior MI in the next few days or weeks if they are on medical treatment only. As soon as the diagnosis is made or suspected, due to possible critical stenosis of the anterior descending artery, definitive treatment is cardiac catheterization with PCI [22].

Reinhardt et al. [23] described the following diagnostic criteria for Wellens syndrome:

1. ECG: deeply inverted T-waves in leads V2 – V3 (may also be seen in leads V1, V4 – V6) or biphasic T-waves (with initial positivity and terminal negativity) in V2 and V3; isoelectric or minimally elevated ST segment < 1 mm; preservation of precordial R wave progression and no precordial Q waves;

2. Clinical: recent history of angina, ECG pattern present in a pain-free state, normal or slightly elevated cardiac markers.

Wellens syndrome has two patterns of T-waves. In type A, T-waves are biphasic, with initial positivity and terminal negativity, present in approximately 25% of cases. Type B T-waves are deeply and symmetrically negative and are present in approximately 75% of cases. The T-wave abnormalities may be persistent for hours to weeks, even when the patient is pain-free [24] (Figures 3 and 4).

The mechanism of Wellens syndrome remains unclear. It is believed that the changes in T-wave are caused by ischemic changes of anterior myocardial wall after sudden occlusion or subocclusion of the anterior descending artery. Reperfusion of the anterior descending artery relieves the pain and iden-

Figure 2. ECG presentation of left main coronary artery stenosis

Slika 2. Elektrokardiogram stenoze glavnog stabla leve koronarne arterije

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Wellens syndrome has two patterns of T-waves. In type A, T-waves are biphasic, with initial positivity and terminal negativity, present in approximately 25% of cases. Type B T-waves are deeply and symmetrically negative and are present in approximately 75% of cases. The T-wave abnormalities may be persistent for hours to weeks, even when the patient is pain-free [24] (Figures 3 and 4).

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Figure 3. ECG pattern of Wellens syndrome - type A

Slika 3. Elektrokardiogram Velensovog sindroma - Tip A

tified T-wave changes as a reflection of reperfusion, form biphasic or negative T-waves. If the coronary artery remains occluded, acute anterior wall MI with ST elevation develops [25]. These ECG changes are not characteristic and limited to anterior MI, but
Isolated posterior MI

Posterior MI is a clinical entity that occurs in 15 – 20% of cases of acute MI with ST segment elevation, usually in combination with inferior or lateral MI, while isolated posterior infarction is only found in about 5% of all ST elevation MIs [28], and it is an indication for urgent coronary reperfusion. The ECG findings of a posterior wall MI are different than the typical ST segment elevation seen in other MIs [29]. However, the absence of ST elevation in the standard ECG can lead to missed diagnosis and these patients do not receive reperfusion therapy, the only proper treatment in the acute phase of infarction. About 90% of these patients have critical stenosis or occlusion of the right coronary artery, and 10% of the left coronary artery [30].

There is a high degree of suspicion for posterior MI when the following findings are persisting in the standard ECG: ST segment depression (horizontal, upsloping, downsloping) and prominent upright R wave (> 30 ms) in V1 – V3 leads (can be equal voltage of R and S wave in V1), the R/S ratio > 1.0 in V2 lead, combination of horizontal ST segment depression with a prominent R-waves in leads V1 – V3. Usually, coexistence of acute inferior and/or lateral MI can be seen [31]. To confirm the diagnosis, extra posterior leads can be used (V7 – V9) [32]. Lead V7 is on the left posterior axillary line in the fifth intercostal space, lead V8 on the left side of the back at the tip of the scapula and V9 on the left paraspinal line in the fifth intercostal space) [33]. The use of additional posterior chest wall leads (V7 – V9) in patients with high suspicion of posterior MI (with circumflex occlusion) should be considered, in order to increase the number of diagnosed MIs, leading to better risk assessment, prognosis and survival, due to reperfusion therapy.

From the last ESC guide for acute MI with ST elevation, posterior infarction is confirmed with the presence of ST segment depression ≥ 0.5 mm in leads V1 – V3 and ST segment elevation ≥ 0.5 mm in posterior leads (V7 – V9), and prompt primary percutaneous coronary intervention strategy is recommended in patients with ongoing symptoms consistent with myocardial ischemia [6] (Figure 5).

New or presumed new left bundle branch block

ST segment criteria for the diagnosis of acute ischemia are affected by the presence of the left bundle branch block, because of the presented secondary ST changes that occur in this pattern. Left bundle branch block presents a dilemma in the evaluation of chest pain [34]. ECG diagnosis of MI in this pattern is difficult, but possible. The third universal definition of MI [35] gives diagnostic criteria for MI: rise and/or fall of cardiac biomarker values plus symptoms of ischemia, or new or presumed new significant ST segment T-wave changes, or new left bundle branch block. So, new or presumed new left bundle branch block in symptomatic patients is recognized as a STEMI equivalent. Neeland et al. [36] found that only about 40% of patients with presumed new left bundle branch block had a culprit lesion on angiography, and new or presumed new left bundle branch block cannot be a diagnostic criteria “per se”, especially not for prompt percutaneous coronary intervention strategy.

The American Heart Association/American College of Cardiology/Heart Rhythm Society Guide [3] recommends to think about the possibility of acute infarction in patients with left bundle branch block who have some of ST segment changes: ST segment elevation ≥ 0.1 mV (1 mm) in leads with a positive ventricular depolarization (QRS), or ST depression ≥ 0.1 mV (1 mm) in leads V1 – V3 (concordant ST segment changes), ST segment elevation ≥ 0.5 mV (5 mm) in leads with negative QRS complex (discordant ST segment changes). The same criteria are used in ESC guidelines for the management of acute MI in patients presenting with ST segment elevation, with a suggestion for patients with a clinical suspicion of ongoing myocardial ischemia and left bundle branch block to be managed in a way similar to STEMI patients [6] (Figure 6).

Sgarbossa criteria [37] are a set of ECG findings generally used to identify MI in the presence of a left bundle branch block or a ventricular paced
rhythm, in order to increase the number of early diagnosed MIs and perform prompt reperfusion. Three included criteria are: ST elevation ≥ 1 mm in a lead with positive QRS complex (concordance) – 5 points, concordant ST depression ≥ 1 mm in leads V1, V2, or V3 – 3 points, and ST elevation ≥ 5 mm in a lead with negative (discordant) QRS complex – 2 points: ≥ 3 points = 90% specificity of STEMI. In the original Sgarbossa criteria, a score < 3 is typically not considered diagnostic of acute MI, but it also does not rule it out. In a review of ventricular-paced ECGs, the most clinically useful Sgarbossa criterion in identifying acute MI was ST segment elevation > 5 mm discordant with the QRS complex and it is helpful in identifying patients who may ultimately benefit from early aggressive treatment strategies [38].

Smith et al. modified Sgarbossa original criteria replacing the ST elevation measurement ≥ 5 mm in the third component with a ST/S ratio less than –0.25, greatly improving the diagnostic utility of the rule. Using these criteria resulted in good prediction for acute coronary occlusion: at least one lead with concordant ST elevation ≥ 1 (Sgarbossa criterion 1) or at least one lead of V1 – V3 with concordant ST depression ≥ 1 (Sgarbossa criterion 2) or ST/S ratio ≤ –0.25 and at least 2 mm of ST elevation (replaces Sgarbossa criterion 3) [39].

Conclusion

In everyday clinical practice, especially in emergency room settings, it is very important to think about the possibility of having patients with chest pain and atypical electrocardiographic presentations of myocardial infarction with ST elevation and with coronary artery occlusion. In these patients, with clinical presentation of ongoing myocardial ischemia, a primary percutaneous coronary intervention strategy should be indicated to prevent complications and mortality, because these patients are at higher risk than others with uncomplicated myocardial infarction with ST elevation.

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