ATYPICAL LOCATION OF ST ELEVATION ACUTE MYOCARDIAL INFARCTION IN RELATION TO THE ELECTROCARDIOGRAM

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Summary
Introduction. Electrocardiography is an initial non-invasive diagnostic algorithm for ST elevation acute myocardial infarction. Specific electrocardiographic phenomenon is described, when the occlusion of the proximal segment of the right coronary artery or the isolated occlusion of its ventricular branch is presented with ST elevation in the precordial leads. Case Report. A 78-year-old woman was admitted as an emergency due to chest pain and electrocardiographically recorded concave elevation in leads V1–V3. She was diagnosed with ST elevation myocardial infarction of the anterior region and sent to catheterization laboratory for emergency coronary angiography. It showed an occlusion of the proximal-medial right coronary artery. Behind the occlusion, the right coronary artery, posterior descending artery and posterior lateral artery, a hetero-collateral circulation was seen. Two drug-eluting stents were implanted into the proximal segment of the right coronary artery. Discussion. The phenomenon of acute myocardial infarction caused by occlusion of the proximal right coronary artery and/or ventricular branches of the right coronary artery, presenting with ST segment elevation in the precordial leads, is a consequence of several anatomical variations: occlusion of non-dominant right coronary artery, isolated occlusion of the ventricular branch of the right coronary artery; and the occlusion of the right coronary artery proximal to the ventricular branch with hetero-collateral circulation on the periphery of the right coronary artery, like in our case. Electrocardiographic characteristic pointing to the occlusion of the proximal right coronary artery and/or ventricular braches of the right coronary artery is higher ST elevation in the lead V1 than in the other leads, followed by the absence of Q wave development. This ST elevation is concave. Conclusion. It is necessary to emphasize the significance of differential diagnosis of culprit lesion in patients with chest pain and elevation of the ST segment in the precordial leads having in mind further different theaapeutic algorithms. Patients with right ventricular myo cardial infarction need to maintain an adequate “preload” and avoid vasodilators in order to maintain the right ventricular stroke volume.

Key words: ST Elevation Myocardial Infarction; Electrocardiography; Coronary Angiography; Heart Ventricles; Stents; Diagnosis, Differential

Sažetak
Uvod. Elektrokardiografija predstavlja inicijalnu neinvazivnu dijagnostičku metodu u algoritmu postavljanja dijagnoze akutnog infarkta miokarda sa ST elevacijom. Opisan je i specifičan elektrokardiografski fenomen kada se okluzija proksimalnog segmenta desne koronarne arterije ili izolovana okluzija ventrikularne grane desne koronarne arterije prezentuje sa ST elevacijom u odvodima V1-V3. Pod radnom dijagnozom infarkt miokarda sa ST elevacijom anterijorne regije, upućena je u kateterizacionu laboratoriju radi urgentne koronarografije. Koronarografijom se registruje okludiran proksimalnolateralni segment desne koronarne arterije. U istom aktu je urađena percutana koronarna intervencija sa implantacijom dva lekom obložena stenta u proksimalno-medijalni segment desne koronarne arterije. Diskusija. Fenomen da se akutni infarkt miokarda uzrokovao okluzijom proksimalnog segmenta desne koronarne arterije i/ili njene ventrikularne grane, manifestuje elevacijom ST segmenta u prekordialnim odvodima, nastaje kao posledica nekoliko anatomskih varijacija: okluzije nedominantne desne koronarne arterije, izolovane okluzije njene komorove grane i okluzije desne koronarne arterije proksimalnije od odvajanja ventrikularne grane sa koralateralnim prikazom distalnog segmenta i periferije kao u našem slučaju. Elektrokardiografske specifičnosti koje ukazuju na ovaj fenomen su veća ST elevacija u odvodu V1 nego u preostalim odvodima praćena odsustvom Q-zubca. Ovakva ST elevacija ima kupolast oblik. Zaključak. Neophodno je naglasiti značaj diferencijalne dijagnoze infarktna arterija kod pacijenata sa angiognznom tegobama i elevacijom ST segmenta u prekordialnim odvodima imajući u vidu različit terapijski algoritam. Kod pacijenata sa izolovanim infarktom miokarda desne komore neophodno je održati adekvatan preload i izbegavati vazodilatatatore kako bi se održao adekvatan udarni volumen desne komore.

Ključne reči: STEMI; elektrokardiografija; koronarna angiografija; srčane komore; stentovi; diferencijalna dijagnoza

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Ivanović V, et al. Atypical Acute Myocardial Infarction and ECG
Electrocardiography (ECG) is an initial non-invasive diagnostic algorithm for acute coronary syndrome. Acute coronary syndrome may be classified into three categories: acute ST elevation myocardial infarction (STEMI), acute non-ST elevation myocardial infarction (NSTEMI) and unstable angina pectoris (UAP). Electrocardiographic manifestations of NSTEMI and UAP are ST depression and/or negative T waves. The difference between these two entities is in the markers for myocardial necrosis. The NSTEMI is associated with elevated markers of myocardial necrosis. All three entities have the same pathoanatomic substrate - a complicated atherosclerotic plaque. Acute STEMI is the most severe manifestation of acute coronary syndrome.

The ST segment elevation on the ECG must be interpreted in the context of clinical findings. False-positive causes of ST segment elevation are early repolarization, left bundle branch block (LBBB), pre-excitation syndrome, Brugada syndrome, peri/myocarditis, pulmonary embolism, subarachnoid hemorrhage, metabolic disorders (hyperkalemia in the first place), various forms of cardiomyopathy, ECG lead transposition, cholecystitis, persistent juvenile ECG, use of tricyclic antidepressants and phenothiazines [1]. False-negative causes of ST segment elevation are prior myocardial infarction with Q waves and/or persistent ST elevation in paced rhythm and LBBB [1].

The ST segment elevation (measured at the J point) in two or more leads with ST elevation ≥ 2.5 mm in men < 40 years, ≥ 2 mm in men ≥ 40 years and ≥ 1.5 mm in women in V2 - V3 and/or ≥ 1 mm in other leads, indicates acute occlusion of the coronary artery [2].

The ST segment elevation in the precordial leads usually indicates occlusion of the left anterior descending artery (LAD) or one of its branches. There is a description in the literature of a specific electrocardiographic phenomenon where the ST segment elevation in the leads V1 - V3 occurs due to the occlusion of the proximal segment of the right coronary artery (RCA) and/or isolated occlusion of the ventricular RCA branch [3–5].

Several algorithms for electrocardiographic identification of infarct related artery have been established with high accuracy. However, their significance is limited in patients with coronary artery anomalies, LBBB, paced rhythm and patients who had prior myocardial infarction or surgical myocardial revascularization.

We present a patient with acute myocardial infarction caused by occlusion of the proximal-medial segment of RCA, but manifesting with ST segment elevation in the leads V1 - V3, instead in D2, D3, and augmented voltage foot (AVF), which is usual.

**Case Report**

A 78-year-old woman with arterial hypertension and type 2 diabetes mellitus was admitted as an emergency to the Clinic of Cardiology of the Institute of Cardiovascular Diseases of Vojvodina. The working diagnosis was STEMI in the anterior wall. Chest pain occurred about 3 hours prior to admission. The ECG showed concave ST segment elevations in leads V1 through V3. ST segment changes in the inferior leads were not registered (Figure 1) only significant Q waves in derivations D2, D3 and AVF, with negative T wave in D3 and AVF.

Ten years earlier, the patient had an inferior wall STEMI. At that time, she was treated with fibrinolytic therapy (Alteplase). Due to persistent chest pain and ST segment elevation, a rescue percutaneous coronary intervention (PCI) was performed along with implantation of two drug-eluting stents 24 x 3.0 mm and 23 x 3.5 mm in the proximal-medial RCA (overlap technique). At that time, angiography of the left coronary system was without significant stenosis. After the intervention and during the follow-up period, ECG showed: sinus rhythm, Q wave and negative T wave in D2, D3, AVF, V5, and V6 without rhythm disorders.

On admission to our emergency department, the initial blood pressure was 130/80 mmHg and heart rate 60 beats per minute. During the physical examination, the patient presented without cardiac murmur, abnormal breath sounds, swollen jugular vein or edema of lower extremities. Dual antiplatelet therapy with aspirin and ticagrelor was administered along with the analgesic therapy in the emergency room. The patient was immediately transferred to the catheterization laboratory for emergency coronary angiography. As usual, the non infarction artery was performed first, and according to ECG it should have been RCA. We expected LAD to be the infarction artery. However, coronary angiography showed an occlusion of the proximal-medial RCA which was the dominant artery (Figure 2A). Behind the occlusion of the RCA, posterior descending artery (PDA) and posterior lateral artery (PLA), a hetero-collateral circulation was seen (Figure 2B). Angiography of the left coronary system showed a non-significant stenosis.

One drug-eluting stent 38 x 3.00 mm (Resolute Onyx, Medtronic, USA) was implanted into the medial segment of RCA and an “overlap” drug-eluting stent 28 x 3.5 mm (Coroflex-Isar-Neo, Braun, Germany) into the proximal segment of RCA. After implantation of stents and establishment of thrombolysis in myocardial infarction (TIMI) 3 Flow
through the occluded blood vessel, two ventricular branches were observed (Figures 2C and 2D). The first branch had a small area of vascularization, while the other branch, with a diameter around 1.5 mm, was significantly longer with the significantly larger area of vascularization; the Blush flow was III. Successful PCI resulted in the resolution of the chest pain and ST segment elevation. The post PCI echocardiography showed normal left ventricular ejection fraction of 58. The medial-base segment of the inferior wall was hyperechogenic, non-homogeneous, and fibrotic, which was considered to be the consequence of the prior myocardial infarction. Also, the right ventricle was slightly dilated (tricuspid annular plane systolic excursion (TAPSE) 17 mm, RAVS 81 ml.

The patient was discharged from the hospital 5 days after the intervention without any subjective problems and with residual ST elevation up to +1 mm in V1 through V3, but without development of Q wave.

**Discussion**

Occlusion of the RCA commonly manifests with ST segment elevation in the inferior leads. However, an electrocardiographic phenomenon has been described in the literature where the occlusion of the proximal RCA and/or ventricular branches of RCA is associated with the elevation of the precordial ST segment in the V1 - V3 leads [3–5]. Kim et al. reported that the occlusion of the proximal segment of RCA with the collateral circulation in the distal segment and periphery of the RCA manifesting with the elevation of the ST segment in the precordial leads [3]. The same finding was seen at coronary angiography in our patient. An abundant hetero-collateral circulation was found in the inferior wall, while the RCA occlusion proximal to the ventricular branches caused myocardial infarction of the right ventricle, and was considered to be the cause of ST elevation in the precordial leads. Geft et al. published an experiment in which the isolated right ventricular myocardial infarction, caused by the infarct model in canine, manifested with the elevation of the ST segment in the precordial leads [6]. They came to the conclusion that when the myocardial infarction of the inferior wall and right ventricle coexist, the ST elevation dominates in the inferior leads, and the precordial ST elevation is mostly not registered. Inferior wall ischemia generates dominant electrical forces, while the changes caused by right ventricular ischemia are suppressed. This phenomenon happens because the inferior wall involves a large mass of myocardium compared to the right ventricle.

Acikel et al. reported that the cause of ST segment elevations in leads V1 through V3, while performing primary PCI in patients with acute inferior myocardial infarction, was the occlusion of the ventricular branch of the right ventricle of the RCA [5]. Kida et al. studied 57 patients undergoing PCI for RCA. All patients had angina pectoris, without evidence of myocardial infarction. In 8 patients, ST elevation was registered in the precordial leads when the proximal segment of the RCA was occluded by the
PTCA balloon. These patients either had a functionally dominant left coronary artery, or divided domination between the left and right coronary arteries [7]. The ST elevation was explained as a consequence of the right ventricular ischemia, considering the fact that the inferior wall was vascularized by the left coronary artery.

It can be concluded that isolated right ventricular myocardial infarction, manifesting with the elevation of the ST segment in the precordial leads, occurs in the following anatomical variations: occlusion of non-dominant RCA, isolated occlusion of the ventricular branch of RCA and the occlusion of the RCA proximal to the ventricular branch with hetero-collateral circulation in the periphery of the RCA (as in our case).

The literature describes ECG characteristics that indicate the isolated right ventricular myocardial infarction. First of all, it is a higher ST segment elevation in lead V1 and than in the precordial leads V2 and V3, followed by the absence of Q wave development [8, 9]. Also, this ST segment elevation is concave or dome-like [10]. These characteristics are found in our patient as well. However, these criteria do not have high sensitivity and specificity, because they are derived from a study with a small number of patients. RCA disease is the most common cause of sinus node dysfunction or, in other words, myocardial infarction caused by the occlusion of proximal RCA and/or ventricular branches is often followed by bradycardia [11]. Rhythm disorders were not registered in our patient. Echocardiography can register the slight dilatation of the right ventricle with or without local abnormal wall motion, as in our patient. A slight dilatation of the right ventricle is a predisposing factor for cardiac clockwise rotation, which can explain the appearance of ST segment elevation in the precordial leads. On the other hand, the position of leads V1, V2 and V3 is in the region of the right ventricle. In cases where, for some reason, the posteroinferior wall of the left ventricle is protected from ischemia, electrocardiogram shows ST segment elevation in these leads.

**Conclusion**

Finally, it is necessary to emphasize the significance of differential diagnosis of culprit lesion in patients with chest pain and elevation of the ST segment in the precordial leads having in mind further different therapeutic algorithms. Patients with right ventricular myocardial infarction need to maintain an adequate “preload” and avoid vasodilators in order to maintain the right ventricular stroke volume.

In the future, along with the progress of primary percutaneous coronary intervention, detection of an isolated myocardial infarction of the right ventricle will also increase. Furthermore, due to the percutaneous coronary intervention of right coronary artery complex lesions with the consequent occlusion of ventricular branches, a higher incidence of isolated right myocardial infarction may be expected.

**References**

1. Wang K, Asinger RW, Marriott HJ. ST-segment elevation in conditions other than acute myocardial infarction. N Engl J Med. 2003;349(22):2128-35.

2. Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, White HD, et al. Third universal definition of myocardial infarction. Eur Heart J. 2012;33(20):2551-67.

3. Kim SE, Lee JH, Park DG, Han KR, Oh DJ. Acute myocardial infarction by right coronary artery occlusion presenting as precordial ST elevation on electrocardiography. Korean Circ J. 2010;40(10):536-8.

4. Celik T, Yuksel UC, Kursaklioglu I, Iysiy A, Kose S, Isik E. Precordial ST-segment elevation in acute occlusion of the proximal right coronary artery. J Electrocardiol. 2006;39(3):301-4.

5. Ackel M, Yilmaz M, Bozkurt E, Gurlertop Y, Kose N. ST segment elevation in leads V1 to V3 due to isolated right ventricular branch occlusion during primary right coronary angioplasty. Catheter Cardiovasc Interv. 2003;60(1):32-5.

6. Gelf I, Shah P, Rodriguez L, Hulse S, Maddahi J, Berman D, et al. ST elevation in leads V1 to V5 may be caused by right coronary artery occlusion and acute right ventricular infarction. Am J Cardiol. 1984;53(8):991-6.

7. Kida M, Morishita H, Yokoi H, Yoshinaga M, Yasumoto H, Kimura T, et al. Precordial ST-segment elevation caused by right coronary artery occlusion. J Cardiol. 1987;17(3):455-64.

8. Golovchiner G, Matz I, Iakobishvili Z, Porter A, Strasberg B, Solodky A, et al. Correlation between the electrocardiogram and regional wall motion abnormalities as detected by echocardiography in first inferior acute myocardial infarction. Cardiology. 2002;98(1-2):81-91.

9. Alzand BS, Gorgels AP. Combined anterior and inferior ST-segment elevation electrocardiographic differentiation between right coronary artery occlusion with predominant right ventricular infarction and distal left anterior descending branch occlusion. J Electrocardiol. 2011;44(3):383-8.

10. Grollier G, Scanu P, Gofard M, Lognoné T, Valette B, Bureau G, et al. ST segment elevation in anterior precordial leads and right ventricular infarction. Apropos of 6 cases. Arch Mal Coeur Vaiss. 1992;85(1):67-75.

11. Ando G, Gaspardone A, Priotti I. Acute thrombosis of the sinus node artery: arrhythmological implications. Heart. 2003;89(2):E5.