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Case report

Cerebral T wave on ECG in a patient with stroke

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Abstract Cerebral T wave, represents transient, deep, symmetrical, negative T wave on the electrocardiogram (ECG), in patients with stroke. It is important to recognize the cerebral T wave, differentiate it from other disorders presenting with negative T waves, and thus avoid the pitfall of acute coronary syndrome misdiagnosis, and wrong therapy selection (i.e. fibrinolytic, anticoagulant and antithrombotic), which would be very dangerous for a patient with cerebral hemorrhage. The female patient, 84 years old, was hospitalized due to a hip fracture to the orthopedic department of the Health center Zajecar hospital. Her past medical history was remarkable for treated arterial hypertension and paroxysmal atrial fibrillation. She was hemodynamically stable and asymptomatic. As part of the preoperative management, an internist consultation was sought due to newly developed ECG abnormalities: sinus rhythm, HR of 65/min, left bundle branch block (LBBB) and negative T waves of 5mm in the precordial leads. There were echocardiographic wall motion abnormalities of the left ventricle. Hs troponin level was 278 ng/ml and acute coronary syndrome was suspected. The patient was started on ACE inhibitor, beta-blocker, statin and dual antiplatelet therapy. The patient develops right-sided muscle weakness. Brain CT was done and the zone of left low temporoccipital acute ischemia was described. The patient gave the informed consent for coronary angiography this time and after stabilization it was performed. The angiographic findings were normal. Consequently, with optimized medical therapy, she was returned to orthopedics for further orthopedic care.

Key words cerebral T wave, ischemic stroke

Introduction

Cerebral T wave, represents transient, deep, symmetrical, negative T wave on the electrocardiogram (ECG), in patients with stroke [1]. It was first described by George Burche in 1954[2]. It is important to recognize the cerebral T wave, differentiate it from other disorders presenting with negative T waves, and thus avoid the pitfall of acute coronary syndrome misdiagnosis, and wrong therapy selection (i.e. fibrinolytic, anticoagulant and antithrombotic), which would be very dangerous for a patient with cerebral hemorrhage.The pathophysiology is incompletely understood[3]. In various articles numerous potential explanations exist. *Sakamoto et al* believe that focal myocytolysis occurs due to excessive stimulation of sympathetic centers in the hypothalamus which leads to the release of catecholamines that can lead to myocardial damage, either by direct toxic effects or by causing constriction of myocardial microcirculation leading to focal ischemia[4]. *Baroldi et al* think that focal myocardial lesions are caused by the release of catecholamines from the intramyocardial nerve endings and not from the circulation[5]. Some of the authors believe that the ECG changes that occur in an acute cerebral event are actually a consequence of injury or stimulation of the islet cortex, which has been proven to have cardiovascular effects on stimulation[6,7]. The belief that cerebral T waves are neuron-induced is supported by the observation that negative T waves can be normalized if brain death occurs.
ECG abnormalities seen in patients with elevated intracranial pressure are: cerebral T wave, QT interval prolongation and bradycardia, indicating brainstem herniation - Cushing's reflex. Far less often abnormalities on the ECG are: increased U wave amplitude, arrhythmias, as well as ST segment elevations or depressions that can mimic pericarditis or myocardial ischemia. ECG changes, in case of increased intracranial pressure, most often occur in large intracranial hemorrhages, namely: in subarachnoid hemorrhage, intraparenchymal hemorrhage. Also it occurs in large ischemic stroke which leads to cerebral edema. Then there are traumas as well as tumors and metastases in the brain as etiologies[8]. In the case of our patient, a negative T wave is the result of temporooccipital ischemia. According to a study by *Goldstein et al* in 150 consecutive patients with acute stroke, cerebral T wave occurs in as many as 29%[9].

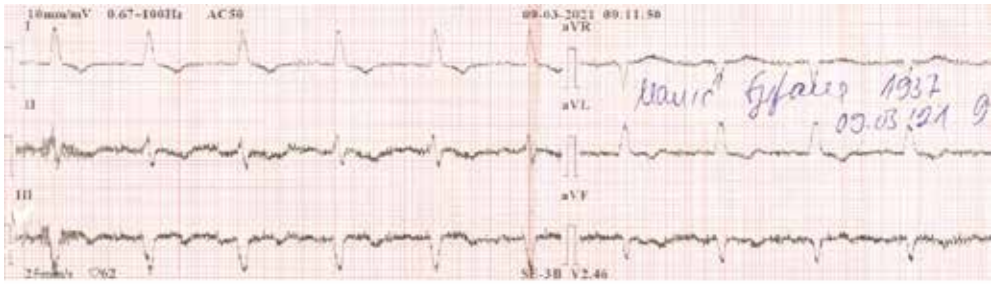

Figure 1a. ECG tracing of the patient during the hospital stay - standard leads

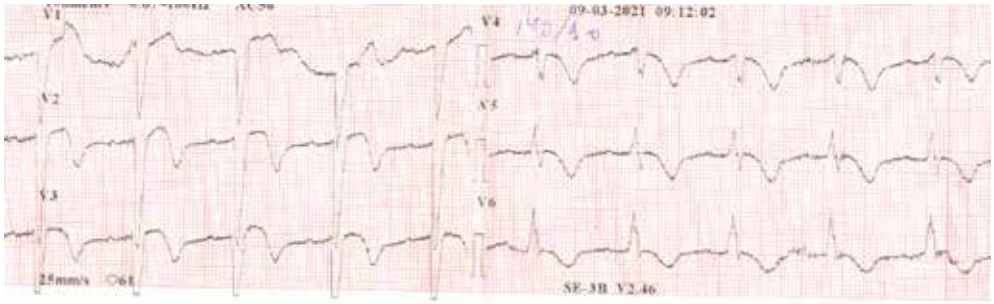

Figure 1b. ECG tracing of the patient during the hospital stay - precordial leads

Case report

Female patient, 84 years old, was admitted to the orthopedics department on March 8, 2021 due to a left hip fracture. Surgical treatment with the partial hip arthroplasty was indicated. At the presentation, in addition to left leg and hip pain, she doesn't have other complaints. Past medical history is remarkable for arterial hypertension and paroxismal atrial fibrillation. She takes an ACE inhibitor for her hypertension.

On physical exam the patient is conscious, oriented, afebrile, eupneic. Preoperatively an internist consultation was requested on March 9, 2021. The patient is without any complaints. Lung and heart examination is unremarkable. TA is 130/80 mmHg. On ECG there was the sinus rhythm, normal axis, HR 75 bpm, LBBB, negative T waves precordially (not described on previous ECG tracing and figured as novel finding - figures 1a, 1b). As part of further assessment, the cardiospecific enzymes and echocardiographic examination were requested.

On March 10, 2021, an ultrasound examination of the heart was performed, where the LV ejection fraction was estimated at 44% with apicoseptal and distal anterior akinesia. Diastolic dysfunction by type of delayed relaxation was observed. Aortic root dimension was 2.9 cm, left ventricle was dilated 5.6 cm diastolic and 4.3 cm systolic dimension. Laboratory results were as follows: hs troponin 278 ng/mL and BNP 179 pg/mL. A working diagnosis of acute coronary syndrome is made and dual antiplatelet therapy, low molecular weight heparin, beta blocker and statin are introduced. Coronary angiography was indicated, but the patient refused invasive diagnostic workup.

On the next day, March 11th 2021. weakness of the right extremities develops, a neurologist is consulted, and a brain CT scan (left low temporooccipital zone of acute ischemia) was performed, and she was transferred to the neurology department, where treatment of ischemic stroke was continued (Figure 2).

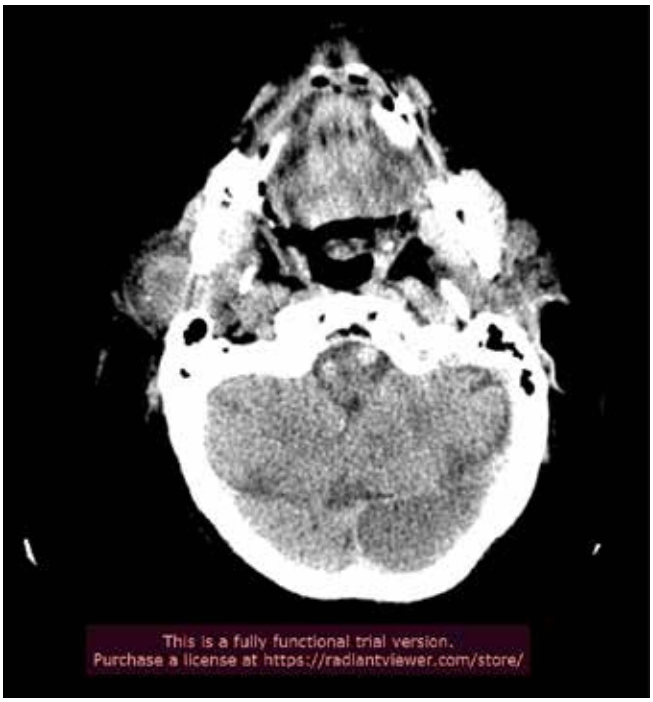

Figure 2. CT scan of the brain: left low temporooccipital zone acute ischemia (ischemic stroke)

The same day ECG (March 11th 2021) showed: sinus rhythm, LBBB, HR 65 bpm, -T to 5mm in D1, aVL, V4-V6 (figure 3).

After clinical stabilization, a decision is made to transfer her to the internal ward for the purpose of performing coronary angiography,for which this time the patient gave the informed consent. This was done in order to later perform surgical treatment of the hip fracture in the orthopedics. Patient is without any complaints, hemodynamically stable, transferred to the internal ward. ECG on admission: sinus rhythm, LBBB, HR 90 bpm, -T of 5mm in D1,aVL.

Coronary angiography was performed on March 18th 2021. There are no angiographically significant stenoses

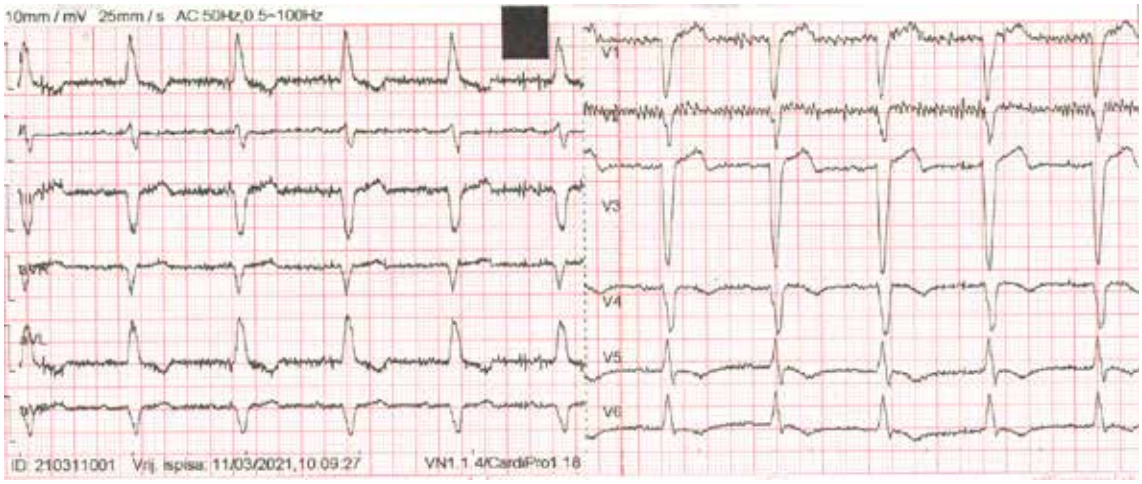

Figure 3. ECG during admission to the neurology department (March 11th 2021)

Figure 4. Coronary angiography was performed on March 18th 2021.

on the epicardial coronary arteries that would be a contraindication for the planned orthopedic procedure - left hip arthroplasty (figure 4)

The patient was transferred to the orthopedic department for planned left hip arthroplasty. Therapy on internal ward discharge: bisoprolol 1x5mg, aspirin 1x75mg, pantoprazol 1x20mg, atorvastatin 1x40mg. An anesthesiologist was consulted at the orthopedic department, who concludes that she is currently inoperative. The procedure is contraindicated and the patient is discharged and referred for further treatment to the Internal Department of OB Knjaževac. Therapy on discharge is as follows: bisoprolol 1x5mg, aspirin 1x75mg, pantoprazol 1x20mg, atorvastatin 1x40mg, enoxaparine 40mg o.d. s.c.

Discussion

Cerebral T wave, represents transient, deep, symmetrical, negative T wave on the electrocardiogram (ECG), in patients with stroke[1]. The differential diagnosis of a negative T wave on a 12-lead ECG includes: ischemia and myocardial infarction, ventricular hypertrophy, pulmonary embolism, hypertrophic cardiomyopathy, bundle branch block, and increased intracranial pressure.Its frequency seems to be much higher than it is usually thought. And thus is the importance of indicating it and proper diagnosis. According to the early study, in 150 consecutive patients with acute stroke, cerebral T wave occurs in as many as 29%[9].

It is very important to emphasize that ECG changes during acute stroke can mimic those in acute coronary syndrome. This can pose a risk of misdiagnosis of STEMI in a patient with intracranial hemorrhage and to consequently lead to the wrong fibrinolytic therapy. Therefore, it is necessary to differentially think about the possibility of increased intracranial pressure.

Our patient was suspected of having the acute coronary syndrome, based on ECG changes of the newly formed block of the left branch of the His bundle, with deep negative T waves and wall motion abnormalities of the left ventricle on echocardiography with slightly elevated cardiac specific enzymes. It should be noted that there is literature which tested the hypothesis that cerebral T waves are associated with transient cardiac dysfunction. The study included patients diagnosed with hemorrhagic or ischemic brain strokes. The ECG was monitored for cerebral T which was defined as the inversion of a T wave depth ≥5 mm in ≥4 precordial leads. An echocardiographic examination was performed for left ventricular wall motion abnormality. Of all 800 patients, 17 had cerebral T wave (2,1%). Of all patient with cerebral T wave, 14 (82%) had normal wall motion, while only 3 had transient wall motion abnormalities (18%). Of these patients, two had cardiomyopathy similar to

Takotsubo cardiomyopathy, and one globally decreased left ventricular function. Coronary angiography was performed in these patients that showed no significant stenoses to explain left ventricular dysfunction[3].
Some authors believe that the changes on the ECG withdraw very quickly, in proportion to the normalization of intracranial pressure. So most ECG changes will withdraw in 3 days, although they can last up to 8 weeks from the withdrawal of intracranial pressure.
In our case, coronary angography was of great importance for the correct diagnosis and thus therapy, in addition to markers of cardiac necrosis and echocardiography, which is also emphasized in similar works[10].

Conclusion

It is important to emphasize that ECG changes, within a stroke, can mimic changes in acute coronary syndrome. It is important to recognize the cerebral T wave, to differentiate it from other negative T waves etiologies, and thus avoid the pitfall of misdiagnosis of acute coronary syndrome, and thus wrong therapy (ie fibrinolytic, anticoagulant and antithrombotic therapy) which would be dangerous for the patient with cerebral hemorrhage. In this case, the importance of coronary angiography as a diagnostic tool for making a correct diagnosis should be emphasized.

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Sažetak

Cerebralni T talas na EKG-u kod pacijenta sa moždanim udarom

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Cerebralni T talas predstavlja prolazni, duboki, simetrični, negativni T talas na elektrokardiogramu (EKG) kod pacijenata sa moždanim udarom. Važno je prepoznati cerebralni T talas, diferencijalno dijagnostički ga razlikovati od ostalih poremećaja praćenih negativnim T talasom, i na taj način izbeći zamku pogrešne dijagnoze akutnog koronarnog sindroma, a samim tim i primenu pogrešne terapije (npr. fibrinolitičke, antikoagulantne i antitrombocitne), što bi za bolesnika sa moždanom hemoragijom bilo jako opasno. Pacijentkinja starosti 84 godina, hospitalizovana je zbog preloma vrata butne kosti, na ortopedsko odeljenje Zdravstvenog centra u Zaječaru. Od ranije boluje od arteriske hipertenzije i povremenih epizoda atrijalne fibrilacije. Hemodinamski je stabilna, bez subjektivnih tegoba. U okviru preoperativne pripreme, pregledana je od interniste zbog novonastalih EKG promena: sinusni ritam, normogram, sf 65/min, blok leve grane Hissovog snopa (BLG), negativan T talas preko 5mm u prekordijalnim odvodima. Ehokardiografski su viđeni segmentni ispadi u kinetici zidova leve komore.hs troponin je bio 278ng/mL pa je postavljena radna dijagnoza akutnog koronarnog sindroma. Uvedena je terapija ACE inhibitorom, beta blokatorom, statinom i dvojna antitrombocitna terapija. Kod pacijentkinje dolazi do razvoja desnostrane mišićne slabosti. Urađen je CT endokranijuma na kome je opisana zona akutne ishemije nisko temporokcipitalno levo. Nakon kliničke stabilizacije, urađena je koronarografija, sa kojom je ovog puta pacijentkinja saglasna. Koronarni angiogram bio je uredan. Sledstveno, pacijentkinja je, uz optimizaciju medikamentne terapije, vraćena na ortopediju radi daljeg ortopedskog zbrinjavanja.

Ključne reči: cerebralni T talas; ishemijski moždani udar