Paroxysmal supraventricular arrhythmias during hypokalemic episodes in a patient with hypokalemic periodic paralysis

Hipokalemik periyodik paralizili bir hastada hipokalemik epizodlar sırasında gelişen paroksismal supraventriküler aritmiler

Dear Editor,

A 21-year-old female patient was admitted to our hospital with severe muscle weakness, fatigue, unable to move all extremities and palpitation following a high carbohydrate meal. The patient described similar symptoms a week ago, which she recovered spontaneously in 48 hours. Her past and family history was unremarkable. Physical examination was notable for flaccid tetraparesis, decreased deep tendon reflexes, with sparing of the facial, oropharyngeal and respiratory muscles. Sensory testing was intact. Thyroid and other system examinations were unremarkable. Electrocardiography (ECG) on admission revealed supraventricular tachycardia (180 bpm) (Fig. 1A). Initial laboratory tests showed a potassium level of 2.67 mEq/L (normal range 3.5-5.1 mEq/L); all other routine examinations and thyroid hormone levels were normal. She presented sinus rhythm after intravenous potassium replacement and diltiazem (12.5 mg). Control potassium level showed 3.78 mEq/L. Electrophysiological study revealed dual AV nodal physiology, inability to induce any tachycardia and no ablation therapy. She discharged uneventfully. While she was asymptomatic for 2 months, the patient admitted to emergency room with palpitation again. ECG on admission showed atrial tachycardia (186 bpm) (Fig. 1B). In addition, biochemistry tests showed potassium level of 2.78 mEq/L. Her palpitation was resolved after intravenous potassium replacement and diltiazem (12.5 mg). Control potassium level showed 3.78 mEq/L. Electrocardiography (ECG) on admission showed atrial tachycardia (186 bpm) (Fig. 1B). In addition, biochemistry tests showed potassium level of 2.78 mEq/L. Her palpitation was resolved after intravenous potassium replacement and diltiazem (12.5 mg). Control potassium level showed 3.78 mEq/L. Electrocardiography (ECG) on admission showed atrial tachycardia (186 bpm) (Fig. 1B). In addition, biochemistry tests showed potassium level of 2.78 mEq/L. Her palpitation was resolved after intravenous potassium replacement and diltiazem (12.5 mg). Control potassium level showed 3.78 mEq/L. Electrocardiography (ECG) on admission showed atrial tachycardia (186 bpm) (Fig. 1B). In addition, biochemistry tests showed potassium level of 2.78 mEq/L. Her palpitation was resolved after intravenous potassium replacement and diltiazem (12.5 mg). Control potassium level showed 3.78 mEq/L. Electrocardiography (ECG) on admission showed atrial tachycardia (186 bpm) (Fig. 1B). In addition, biochemistry tests showed potassium level of 2.78 mEq/L. Her palpitation was resolved after intravenous potassium replacement and diltiazem (12.5 mg). Control potassium level showed 3.78 mEq/L. Electrocardiography (ECG) on admission showed atrial tachycardia (186 bpm) (Fig. 1B). In addition, biochemistry tests showed potassium level of 2.78 mEq/L. Her palpitation was resolved after intravenous potassium replacement and diltiazem (12.5 mg). Control potassium level showed 3.78 mEq/L. Electrocardiography (ECG) on admission showed atrial tachycardia (186 bpm) (Fig. 1B). In addition, biochemistry tests showed potassium level of 2.78 mEq/L. Her palpitation was resolved after intravenous potassium replacement and diltiazem (12.5 mg). Control potassium level showed 3.78 mEq/L. Electrocardiography (ECG) on admission showed atrial tachycardia (186 bpm) (Fig. 1B). In addition, biochemistry tests showed potassium level of 2.78 mEq/L. Her palpitation was resolved after intravenous potassium replacement and diltiazem (12.5 mg).

Hypokalemic periodic paralysis is an autosomal dominant disorder which is accompanied by muscle weakness/paralysis and hypokalemia. Attacks can be induced by exercise, carbohydrate-rich meals

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Address for Correspondence/Yazışma Adresi: Enes Elvin Gül, MD
Selçuk Üniversitesi, Meram Tıp Fakültesi, Kardiyoji Sekreterliği, Meram, 42090 Konya-Türkiye
Phone: +90 332 223 60 72 Fax: +90 332 223 71 21
E-mail: elvin_salamov@yahoo.com
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Metabolic syndrome without overt diabetes is associated with prolonged pro-arrhythmogenic electrocardiographic parameters

Asikar diyabet olmaksızın metabolik sendrom uzamış proarritmik elektrokaridiyografik parametreler ile ilişkilidir

Dear Editor,

It is shown in many studies that both metabolic syndrome (MS) and the risk factors related to MS [such as diabetes mellitus (DM)] were independently associated with sudden cardiac death (SCD) (1). Moreover, in a study, a follow-up of asymptomatic MS patients for 21 years showed that SCD was more frequently encountered than non-SCD (1).

References


Address for Correspondence/Yazışma Adresi: Dr. Uğur Canpolat Hacettepe Üniversitesi Tip Fakültesi, Kardiyoloji Anabilim Dalı, Ankara-Turkey

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Metabolic syndrome without overt diabetes is associated with prolonged pro-arrhythmogenic electrocardiographic parameters

Asikar diyabet olmaksızın metabolik sendrom uzamış proarritmik elektrokaridiyografik parametreleri ile ilişkilidir

It has been well established that most cases of SCD are related to severe ventricular arrhythmias. Several electrocardiographic (ECG) pro-arrhythmogenic parameters are risk factors for sudden death and therefore might be used in risk stratification (2). There is a pathophysiological association between prolonged duration of QRS, QT and increased resting heart rate (HRR), QT dispersion (QTD) with SCD.

While previous studies mentioned an increased risk of arrhythmias in MS patients, such a tendency could well be caused by DM, which frequently appears as co-morbidity in these patients. Nevertheless for the first time our study results indicate that pro-arrhythmogenic parameters such as prolonged QRS, corrected QT (QTc) duration and increased RHR, QTc dispersion (QTD) could be useful in evaluating arrhythmic risk and provide new insights to the relationship of SCD and MS in patients without overt diabetes (3).

We conducted a case-control study, which consisted of 142 MS patients, age- and gender-matched, and 170 control subjects. Patients were also excluded if they received any anti-diabetic drug treatment, had a fasting blood glucose level ≥7.0 mmol/L or random plasma glucose level ≥11.1 mmol/L. The results revealed that MS patients had a higher increased RHR (86.7±11.2 vs 74.2±9.3 beats/min, p<0.001), prolonged QRS duration (103.4±9.7 vs 98.3±10.3 msec, p<0.001), QTc duration (434.6±36.0 vs 409.0±20.4 msec, p<0.001) and increased QTD (67.7±13.7 vs 47.1±7.2 msec, p<0.001). In addition, we showed that pro-arrhythmogenic parameters, other than QRS duration, change as the MS score increases. We showed MS criteria (such as increased waist circumference) as an independent predictors of increased RHR, QTD and prolonged QRS, QTc. Increased duration of repolarization parameters in patients with MS can be explained as follows: endothelial and myocardial dysfunction, sympathetic over activation or parasympathetic under activation.

As a result, we proposed that pro-arrhythmogenic parameters such as QRS, QTc durations, RHR and QTD might be used in the development of risk stratification schemes for SCD in MS patients.