

and exposure to cold (1). In the heart, NaV channels are essential for the orderly progression of action potentials throughout the myocardium to stimulate rhythmic contraction. NaV 1.4 channels are expressed principally in the skeletal muscle cells, but there are some demonstrations that the SCN4A a-subunit gene is expressed in normal human heart too (3).

As a result of increased duration of the action potential and refractory period, patients with hypokalemia are at increased risk for certain dysrhythmias like ventricular tachycardia. Extreme syncopal bradycardia and sinus arrest are rare findings in hypokalemia (4). In the literature, there was no association of hypokalemic periodic paralysis with supraventricular arrhythmias. Although it was a hypothesis, we thought that NaV 1.4 channels could play role in development of supraventricular tachycardias in such a case. So, this case is the first report regarding the occurrence of supraventricular tachycardias and hypokalemic periodic paralysis together.

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## References

1. Lin SH, Hsu YD, Cheng NL, Kao MC. Skeletal muscle dihydropyridine-sensitive calcium channel (CACNA1S) gene mutations in Chinese patients with hypokalemic periodic paralysis. *Am J Med Sci* 2005; 329: 66-70. [CrossRef]
2. Pereon Y, Lande G, Demolombe S, Nguyen The Tich S, Sternberg D, Le Marec H, et al. Paramyotonia congenita with an SCN4A mutation affecting cardiac repolarization. *Neurology* 2003; 60: 340-2. [CrossRef]
3. Kantola IM, Tarssanen LT. Familial hypokalemic periodic paralysis in Finland. *J Neurol Neurosurg Psychiatry* 1992; 55: 322-4. [CrossRef]
4. Maffè S, Signorotti F, Perucca A, Bielli M, Hladnik U, Ragazzoni E, et al. Atypical arrhythmic complications in familial hypokalemic periodic paralysis. *J Cardiovasc Med* 2009; 10: 68-71. [CrossRef]

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

*Aşık ar diyabet olmaksızın metabolik sendrom uzamış proaritmik elektrokardiyografik 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).

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 pathophysiologic association between prolonged duration of QRS, QT and increased resting heart rate (RHR), 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 (QTcd) 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  $\geq 7.0$  mmol/L or random plasma glucose level  $\geq 11.1$  mmol/L. The results revealed that MS patients had a higher increased RHR ( $86.7 \pm 11.2$  vs  $74.2 \pm 9.9$  beats/min,  $p < 0.001$ ), prolonged QRS duration ( $103.4 \pm 9.7$  vs  $98.3 \pm 10.3$  msec,  $p < 0.001$ ), QTc duration ( $434.6 \pm 36.0$  vs  $409.0 \pm 20.4$  msec,  $p < 0.001$ ) and increased QTcd ( $67.7 \pm 13.7$  vs  $47.1 \pm 7.2$  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 (4), insulin resistance, sympathetic over activation or parasympathetic under activation (5).

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

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## References

1. Empana J-P, Ducimetiere P, Balkau B, Jouven X. Contribution of the metabolic syndrome to sudden death risk in asymptomatic men: the Paris Prospective Study I. *Eur Heart J* 2007; 28: 1149-54. [CrossRef]
2. Cuddy TE, Halli PS, Tate RB. QT dispersion and heart rate predict the risk of sudden unexpected cardiac death in men: the Manitoba Follow-Up Study. *Prev Cardiol* 2009; 12: 27-33. [CrossRef]
3. Colantonio D, Casale R, Abuzzo BP, Lorenzetti G, Pasqualetti P. Circadian distribution in fatal pulmonary thromboembolism. *Am J Cardiol* 1989; 64: 403-4. [CrossRef]
4. Kumar G, Klarich KW, Collett ND, Lerman A. Electrocardiographic changes in coronary endothelial dysfunction. *Coron Artery Dis* 2008; 19: 395-8. [CrossRef]
5. Dekker JM, Feskens EJ, Schouten EG, Klootwijk P, Pool J, Kromhout D. QTc duration is associated with levels of insulin and glucose intolerance. The Zutphen Elderly Study. *Diabetes* 1996; 45: 376-80. [CrossRef]

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