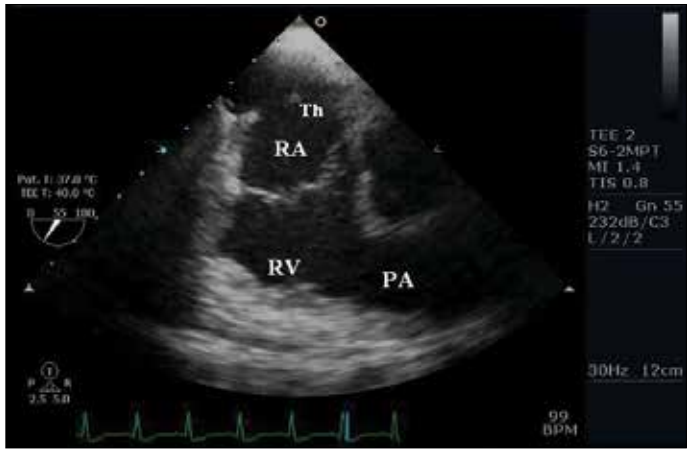

Figure 3. Post-anticoagulation therapy, transesophageal echocardiography showing markedly reduced thrombi in the right atrium
PA - pulmonary artery, RA - right atrium, RV - right ventricle, Th - thrombus

MTHFR mutation has not been reported before. In conclusion, we suggest analyzing genetic mutations in patients with VTE who had not any predisposing factors. Because diagnosis of genetic mutation associated with VTE will require long-term anticoagulation.

Enes Elvin Gül, Halil İbrahim Erdoğan, Ufuk Tan Bayram, Kurtuluş Özdemir
Department of Cardiology, Meram School of Medicine, Selçuk University, Konya-*Turkey*

Video 1. Pretreatment transesophageal echocardiography showing two mobile thrombi in the right atrium
PA - pulmonary artery, RA - right atrium, RV - right ventricle, Th - thrombus

Video 2. Post-anticoagulation therapy, transesophageal echocardiography showing markedly reduced thrombi in the right atrium
PA - pulmonary artery, RA - right atrium, RV - right ventricle, Th - thrombus

References

1. Casazza F, Bongarzoni A, Centonze F, Morpurgo M. Prevalence and prognostic significance of right-sided cardiac mobile thrombi in acute massive pulmonary embolism. Am J Cardiol 1997; 79: 1433-5. [CrossRef]
2. Nizankowska-Mogilnicka E, Adamek L, Grzanka P, Domagala TB, Sanak M, Krzanowski M, et al. Genetic polymorphisms associated with acute pulmonary embolism and deep venous thrombosis. Eur Respir J 2003; 21: 25-30. [CrossRef]
3. Isotalo PA, Donnelly JG. Prevalence of methylenetetrahydrofolate reductase mutations in patients with venous thrombosis. Mol Diagn 2000; 5: 59-66. [CrossRef]
4. Bezemer ID, Doggen CJ, Vos HL, Rosendaal FR. No association between the common MTHFR 677C->T polymorphism and venous thrombosis: results from the MEGA study. Arch Intern Med 2007; 167: 497-501. [CrossRef]
5. Auerbach AD, Sanders GD, Hambleton J. Cost-effectiveness of testing for hypercoagulability and effects on treatment strategies in patients with deep vein thrombosis. Am J Med 2004; 116: 816-28. [CrossRef]

Address for Correspondence/Yazışma Adresi: Enes Elvin Gül, MD
Selçuk Üniversitesi, Meram Tıp Fakültesi, Kardiyoloji Sekreterliği, Meram, 42090 Konya-*Türkiye*
Phone: +90 332 223 60 72 Fax: +90 332 323 71 21
E-mail: elvin_salamov@yahoo.com
Available Online Date/Çevrimiçi Yayın Tarihi: 22.06.2012

doi:10.5152/akd.2012.168

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 the 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 (166 bpm) (Fig. 1B). In addition, biochemistry tests showed potassium level of 2.78 mEq/L. Her palpitation was resolved after intravenous potassium replacement again. She was referred for investigation of the reasons of hypokalemia. Serum potassium levels were normal in between emergency admissions. Also serum magnesium, sodium, calcium levels and thyroid function tests were in normal limits. Urinary potassium level was decreased (24 mEq/L). Urinary potassium/creatinine ratio was 0.50. Transtubular potassium gradient was 6.8 (normal range: 7-9). Arterial blood gas analysis showed no metabolic alkalosis. Serum renin, aldosterone and ACTH levels were normal. Adrenal gland imaging with computed tomography revealed normal findings. So, hypokalemic periodic paralysis was considered in differential diagnosis, which was confirmed by genetic testing (mutation in SCN4A, Arg669H). The patient was discharged with oral potassium supplement (potassium citrate 2.17 gr/day plus potassium carbonate 2.0 gr/day) and avoidance of strenuous exercise and high carbohydrate diet. The 6-months follow-up was free of new paralysis and palpitation episodes.

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

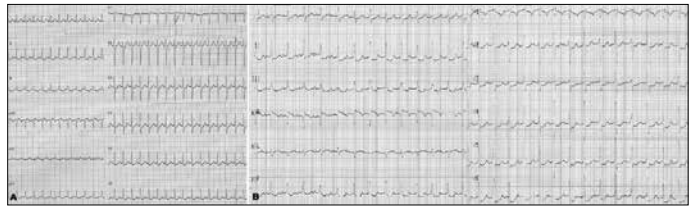

Figure 1. Initial electrocardiography showing supraventricular tachycardia (180 bpm) on first admission (A). Atrial tachycardia (166 bpm) was also seen on second admission (B)

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.

Uğur Canpolat, Hamza Sunman, Kudret Aytemir, Ali Oto
Department of Cardiology, Faculty of Medicine, Hacettepe University, Ankara-*Turkey*

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]

Address for Correspondence/Yazışma Adresi: Dr. Uğur Canpolat
Hacettepe Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, 06100 Sıhhiye, Ankara-*Türkiye*
Phone: +90 312 305 17 80 Fax: +90 312 305 41 37
E-mail: dru_canpolat@yahoo.com
Available Online Date/Çevrimiçi Yayın Tarihi: 22.06.2012

doi:10.5152/akd.2012.169

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

Aşikar 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 ≥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±9.9 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 QTcd (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 (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.

Turgay Işık, Mustafa Kurt[1], Hüseyin Uyarel
Department of Cardiology, Faculty of Medicine, Balıkesir University, Balıkesir-*Turkey*
[1]Clinic of Cardiology, Erzurum Education and Research Hospital, Erzurum-*Turkey*

References

1. Empana JP, Duciemetiere 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, Abruzzo 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]

Address for Correspondence/Yazışma Adresi: Dr. Turgay Işık
Balıkesir Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, Çağış Kampüsü, Balıkesir-*Türkiye*
Phone: +90 266 612 14 55 Fax: +90 266 612 14 59
E-mail: isikturgay@yahoo.com
Available Online Date/Çevrimiçi Yayın Tarihi: 22.06.2012

doi:10.5152/akd.2012.170