The Relationship Between Heart Rate Variability Parameters and Atrioventricular Nodal Reentrant Tachycardia

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ABSTRACT

Introduction: Heart rate variability (HRV) is associated with sympathetic tone. Studies have disputed the interpretation of the low frequency (LF) and low/high frequency (LF/HF) ratio for the indication of sympathetic cardiac control and autonomic balance. This study aims to investigate the association between HRV parameters and atrioventricular nodal reentrant tachycardia (AVNRT) and observe the effect of autonomic nervous system on AVNRT.

Patients and Methods: In total, 354 subjects with palpitations underwent the electrophysiological study (EPS). Most (71%; 253/354) patients had at least an ambulatory Holter ECG recording of 24 h. As a consequence of the exclusion criteria, 160 individuals were classified into two groups: control group (no arrhythmia induced, n= 90) and AVNRT group (n= 70).

Results: Daytime and nighttime LF and LF/HF ratio were significantly higher in the AVNRT group ($p< 0.05$) than in the control group. Interestingly, daytime ultra-LF, very LF, and total power were significantly lower in the AVNRT group.

Conclusion: AVNRT is associated with altered sympathovagal balance. Furthermore, increased LF and LF/HF may be indicative of enhanced sympathetic activity in patients with AVNRT because of inhomogeneous ventricular activation via the slow accessory pathway.

Key Words: Atrioventricular nodal reentrant tachycardia; heart rate variability; low frequency; low/high frequency

Kalp Hızı Değişkenliği Parametreleri ile Atriyoventriküler Nodal Reentrant Taşikardi Arasındaki İlişki

ÖZET

Giriş: Kalp hızı değişkenliği (HRV) sempatik ton ile ilişkilidir. Çalışmalar sempatik kardiyak kontrol ve otonom denge endikasyonu için düşük frekans (LF) ve düşük/yüksek frekans (LF/HF) oranının yorumlamasını tartışmışlardır. Bu çalışma, HRV parametreleri ile atriyoventriküler nodal reentrant taşikardi (AVNRT) arasındaki ilişkiyi araştırmayı ve otonomik sinir sisteminin AVNRT üzerindeki etkisini gözlemeyi amaçlamaktadır.

Hastalar ve Yöntem: Çarpıntısı olan 354 olguya elektrofizyolojik çalışma yapıldı (EPS). Hastaların %71 (253/354)'i en az bir 24 saatlik ambulatuvar Holter elektrokardiyografi (EKG) kaydına sahipti. Dışlama kriterlerinin bir sonucu olarak, 160 birey iki gruba ayrıldı; kontrol grubu (aritmi indüklenmedi, n= 90) ve AVNRT grubu (n= 70).

Bulgular: Gündüz ve gece LF ve LF/HF oranı AVNRT'de kontrollerden anlamlı derecede yüksekti ($p< 0.05$). İlginç olarak, gündüz ultra düşük frekans, çok düşük frekans ve toplam güç AVNRT'de anlamlı olarak düşüktü.

Sonuç: AVNRT değişmiş sempatovagal denge ile ilişkilidir. Dahası, artmış LF ve LF/HF, AVNRT hastalarında yavaş aksesuar yolla homojen olmayan ventriküler aktivasyonun bir sonucu olarak artmış sempatik aktivitenin bir göstergesi olabilir.

Anahtar Kelimeler: Atriyoventriküler nodal reentrant taşikardi; kalp hızı değişkenliği; düşük frekans; düşük/yüksek frekans

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INTRODUCTION

Atrioventricular nodal reentrant tachycardia (AVNRT) is a common type of supraventricular arrhythmia and mostly affects older women and men[1,2]. AVNRT occurs because of a reentrant circuit located in or near the atrioventricular (AV) node causing the heart to beat prematurely[3]. The anatomical structure of the AV node is normal in AVNRT. The functional duality of the AV node plays a role in the discontinuous AV conduction[4,5].

Although electrophysiological differences are found, the influence of the autonomic nervous control on the AV node is important in the reentrant circuit and arrhythmias[6]. Because heart rate variability (HRV) is different in AVNRT, it is a noninvasive marker for evaluation of cardiac autonomic modulation, quantification of cardiac adaptation mechanisms, and analysis of cardiac autonomic status[7,8]. In this study, we compared the cardiac autonomic status between patients with AVNRT and healthy control subjects.

PATIENTS and METHODS

This cross-sectional study was approved by the Adiyaman University ethics committee. In total, 354 consecutive patients who were admitted to our clinic between 1 November 2015 and 1 December 2016 with a complaint of palpitation were enrolled in the study.

Study Population

Epicrises, transaction reports, intracardiac records, and baseline clinical and demographic characteristics were obtained from all the enrolled subjects. Electrophysiological study was conducted in all the included patients. More than half (71%; 253/354) of the patients had at least 24 hours of ambulatory Holter ECG recording. Exclusion criteria were as follows: hypertension (n= 5), diabetes mellitus (n= 5), coronary artery disease (n= 5), use of beta-blockers (n= 8), use of calcium channel blockers (n= 2), and non-AVNRT (n= 63). After evaluation of exclusion criteria, a total of 160 patients were enrolled in the study. The control group (group 1) comprised 90 healthy control subjects with no arrhythmia and the AVNRT group (Group 2) comprised 70 patients. Subjects with a positive smoking history during the past year were classified as smokers. The Simpson's method was used to calculate the left ventricular ejection fraction.

Ambulatory Electrocardiography

A two-channel bipolar recording was performed during a working day. All patients had a similar daily routine. The ambulatory electrocardiographies (ECGs) were recorded continuously for 24 hours using a DR-512 VX3 ECG recorder system (Biomedical Systems, Century series Holter analysis systems, Maryland. Heights, MO, USA). The electrodes were positioned to obtain leads CM2 and CM5. The period from 08.00 to 24.00 was considered day and from 00.00 to 08.00 as night. All tapes were analyzed by two experienced cardiologists. HRV time and frequency domain indices were evaluated. The 24-h average heart rate (HR) was recorded during 24-h electrocardiographic monitoring.

Heart Rate Variability

Power spectral analysis was split into short recording and long recording frequency domain indices. Short recording frequency domain was divided into ultra-low frequency (ULF: < 0.003 kHz), very low frequency (VLF: 0.003-0.04 kHz), low frequency (LF: 0.04-0.15Hz), and high frequency (HF: 0.15-0.4Hz) and recorded between 2 and 5 min. LF and HF powers were reported as normalized units. Long recording frequency domain of ULF, VLF, LF and HF were obtained by 24-h recordings. The measurements were analyzed day/night. Signal powers of LF and HF bands were used due to the restriction duration of recordings. Logarithmic transformation of the integrals under the respective power spectral density function (expressed in ms^2) was used. LF is an indicator of sympathetic activity. LF/HF ratio defines which nervous system is dominant, with a high ratio indicating dominance of the sympathetic system a low ratio indicating the dominance of the parasympathetic system.

Electrophysiologic Evaluation

All subjects were informed of the possible risks and provided their consent through signing the consent forms. Patients underwent EPS under local anesthesia after a 12-h fasting and a 3-day period of no antiarrhythmic drugs, including beta and ca-channel blockers. Multipolar electrode catheters were introduced into the right atrium, the coronary sinus, His bundle, and the right ventricular apex via the femoral and left subclavian veins. The standard 12-lLead ECG (30-500 Hz) with digital system and optical disk storage (EP Tracer, Holland) was used. To prohibit an accessory pathway, a set of well-described maneuvers, i.e., the activation pattern in the coronary sinus (CS) during ventricular stimulation and decremental retrograde VA conduction, was used. After this procedure was completed, we performed programmed atrial stimulation. It is common practice to induce AVNRT in the electrophysiology lab. If tachycardia could not be induced, isoproterenol or atropine infusion was administered such that the HR increased by at least 25%. This procedure was repeated during medication infusion and washout phase. If AVNRT could not be induced, the presence of an A-H jump (a prolongation of the AH interval > 50 ms) and an atrial echo with a documented history of supraventricular tachycardia compatible with AVNRT were sufficient to start the ablation procedure. Dual pathways were not demonstrated with atrial extrastimulus techniques in healthy control subjects.

Radiofrequency Ablation

Radiofrequency energy was delivered to the presumed selected ablation site in the posterior part of Koch's triangle located in the superficial paraseptal endocardium of the right atrium. A standard deflectable 7F catheter with a 4-mm distal electrode was used for mapping and ablation (St. Jude Medical Inc., St. Paul, MN, USA). The ablation generator (IBI-1500T11,

St. Jude Medical Inc., St. Paul, MN, USA) delivered 20 to 35 W of energy by a continuous unmodulated sine wave output at a frequency of 500 kHz. Ablation was performed during sinus rhythm. The ablation methods were similar for all patients with AVNRT, as previously described[9-12].

Statistical Analysis

All statistical analyses were performed using SPSS software (version 21.0 for Windows; SPSS Inc., Chicago, IL, USA). Normality of the data distribution was analyzed using the Kolmogorov-Smirnov test. Continuous data are presented as mean ± SD or median and interquartile ranges based on normality of variables. Differences among groups were compared by independent samples t-test or Mann-Whitney U test according to normality of variables. Categorical variables were summarized as percentages and compared using chi-square test. The correlation between baseline biomarkers was assessed with Spearman's and Pearson's correlation coefficients. Multivariate logistic regression analysis was used to determine the independent predictors for AVNRT. Statistical significance of the findings was interpreted based on p values, and $p< 0.05$ was set as the level of statistical significance.

RESULTS

Table 1 summarizes the baseline characteristics of the study patients. Group 1 consisted of 90 patients (mean age, 46.1 ± 18.1) and group 2 consisted of 70 patients (mean age, 43.9 ± 16.5). The demographic characteristics, including age, smoking, and gender, were similar between the two groups (all, $p> 0.05$). The 24-h average HR and LVEF were not different between the groups ($p> 0.05$). There were no significant differences in serum biochemistry between the two groups (all $p> 0.05$). The hematological parameters were similar in both groups (all, $p> 0.05$) (Table 1).

Daytime HRV time-domain indices were similar between the groups (all, $p> 0.05$). In addition, daytime HRV frequency domain indices (HF, LF NUs, HF NUs) were also similar in both groups (all, $p> 0.05$). Daytime LF and LF/HF ratio were significantly higher in the AVNRT group than in the control group ($p= 0.001$ and $p< 0.001$, respectively). By contrast, daytime ULF, VLF, and total power were significantly higher in the control group ($p= 0.026$, $p= 0.040$, and $p= 0.025$, respectively). Nighttime HRV time-domain indices were similar between the two groups ($p> 0.05$). Nighttime HRV frequency domain indices were similar in both groups (all $p> 0.05$), except the LF and LF/HF ratio. Nighttime LF and LF/HF were significantly higher in the AVNRT group than in the control group (all, $p< 0.001$) (Table 2).

AVNRT positively correlated with daytime total power, ultra-low frequency (ULF) and very low frequency (VLF) power ($r= 0.177$, $p= 0.025$; $r= 0.177$, $p= 0.025$ and $r= 0.163$, $p= 0.040$). Daytime LF, LF/HF ratio, and nighttime LF and LF/HF ratio were inversely correlated with AVNRT ($r= -0.256$, $p= 0.001$; $r= -0.348$, $p< 0.001$ and $r= -0.463$, $p< 0.001$; $r= -0.385$, $p< 0.001$) (Table 3). Multivariate analysis was performed to determine independent predictors of AVNRT. Daytime LF, VLF, and nighttime LF were determined as independent predictors of AVNRT (OR= 1.002, 95% CI: 1.001-1.004, $p= 0.002$; OR= 0.999, 95% CI: 0.998-1.000, $p= 0.006$; and OR= 1.003, 95% CI: 1.001-1.004, $p< 0.001$) (Table 4).

Table 1. Baseline clinical and laboratory characteristics of study population

Variables	Control group (n= 90)	AVNRT group (n= 70)	p
Age (years)	46.1 ± 18.1	43.9 ± 16.5	0.418
Gender (male, %)	41 (45.6)	26 (37.1)	0.285
Smoking (%)	39 (43)	43 (61)	0.319
Heart rate 24-h (bpm)	75.5 (58-89)	76.0 (56-98)	0.953
LV-EF (%)	59.2 ± 3.7	58.8 ± 3.9	0.494
Glu (mg/dL)	89.0 (61-175)	90.0 (66-182)	0.865
Cre (mg/dL)	0.75 (0.36-1.5)	0.74 (0.1-1.4)	0.603
TC (mg/dL)	153.3 ± 42.2	156.9 ± 48.0	0.617
TG (mg/dL)	151.0 (43-277)	150.0 (48-242)	0.395
HDL (mg/dL)	34.2 ± 8.2	33.3 ± 9.0	0.520
LDL (mg/dL)	33.0 (22-178)	33.5 (18-142)	0.915
WBC ($10^3 \times \mu L$)	12.6 ± 3.5	11.4 ± 3.8	**0.037**
HGB (g/dL)	13.3 ± 1.9	13.1 ± 2.1	0.527
Plt ($10^3 \times \mu L$)	241.6 ± 91.7	239.1 ± 78.9	0.855

AVNRT: Atrioventricular nodal reentrant tachycardia, Cre: Creatinine, Glu: Glucose, HDL: High-density lipoprotein, HGB: Hemoglobin, LDL: Low-density lipoprotein, LV-EF: Left ventricular ejection fraction, Plt: Platelet, TC: Total cholestrol, TG: Triglyceride, WBC: White blood cell.

Table 2. Comparison of HRV parameters between groups

Daytime HRV time domain indices	Control group (n= 90)	AVNRT group (n= 70)	p
SDNN	114.6 (60.2-298.4)	102.3 (51.2-207.2)	0.052
SDANN	96.5 ± 31.7	92.0 ± 34.4	0.386
PNN50	30.8 (10.0-455.0)	35.4 (20.3v455.0)	0.228
rMSSD	8.5 (1.0-48.0)	10.0 (1.0-55.0)	0.554
Daytime HRV frequency domain indices			
Total power (ms^2)	8712.0 (2494.0-46264.0)	7694.5 (1764.0-36268.0)	**0.025**
ULF power (ms^2)	4581.0 (1188.0-17225.0)	3698.5 (658.0-17225.0)	**0.026**
VLF power (ms^2)	2443.0 (634.0-9539.0)	1984.0 (340.0-9010.0)	**0.040**
LF power (ms^2)	1122.0 (424.0-9621.0)	1718.0 (166.0-6616.0)	**0.001**
HF power (ms^2)	640.0 (198.0-4498.0)	567.5 (59.0-2617.0)	0.350
LF Nus	40.1 ± 10.5	42.1 ± 12.0	0.269
HF Nus	56.0 ± 11.7	54.7 ± 13.7	0.516
LF/HF ratio	1.87 ± 0.85	2.90 ± 1.9	**< 0.001**
Nighttime HRV time domain indices			
SDNN	102.1 (45.9-446.2)	94.1 (45.9-205.5)	0.269
SDANN	68.0 (11.4-425.7)	67.0 (26.4-212.9)	0.866
PNN50	44.8 (13.0-143.3)	42.6 (11.0-143.3)	0.957
rMSSD	15.5 (1.0-61.0)	14.0 (1.0-63.0)	0.629
Nighttime HRV frequency domain indices			
Total power (ms^2)	7805.0 (2661.0-99334.0)	7303.5 (2661.0-28447.0)	0.510
ULF power (ms^2)	2397.5 (689.0-54251.0)	2224.0 (564.0-8135.0)	0.835
VLF power (ms^2)	2551.0 (675.0-17361.0)	2231.0 (703.0-13204.0)	0.179
LF power (ms^2)	1086.0 (284.0-10702.0)	1761.5 (488.0-10287.0)	**< 0.001**
HF power (ms^2)	851.0 (235.0-31419.0)	855.5 (163.0-5998.0)	0.916
LF NUs	45.6 ± 11.3	44.6 ± 13.8	0.636
HF NUs	52.9 ± 11.0	89.2 ± 17.6	0.090
LF/HF ratio	1.43 ± 0.8	2.4 ± 1.4	**< 0.001**

AVNRT: Atrioventricular nodal reentrant tachycardia, HF: High frequency, LF: Low frequency, LF NUs: Low frequnecy normalized units, ULF: Ultra-low frequency, VLF: Very low frequency.

DISCUSSION

Our study demonstrates that increased HRV is associated with AVNRT. Patients with a history of AVNRT and overt ventricular preexcitation in the presence of a slow accessory pathway had higher LF of HRV and higher LF/HF ratios compared with control subjects. Based on these findings, we suggest that patients with AVNRT have higher sympathetic modulation at baseline and during daily routine life, leading to alterations in their autonomic status.

Catheter ablation for AVNRT has a success rate of 95%, but it is associated with 0.5%-1% risk in AV block and 4% recurrence rate[13]. Katritsis et al. suggested catheter ablation to be the most common treatment modality in patients with symptomatic AVNRT[14]. Catheter ablation was also the first choice for the

Table 3. Correlation between AVNRT and variables

Variables	Correlation coefficient (r)	Significance (p)
Heart rate 24-h (bpm)	-0.05	0.954
LV-EF	-0.054	0.501
Daytime HRV parameters		
Total power (ms^2)	0.177	**0.025**
ULF power (ms^2)	0.177	**0.025**
VLF power (ms^2)	0.163	**0.040**
LF power (ms^2)	-0.256	**0.001**
HF power (ms^2)	0.074	0.352
LF Nus	-0.083	0.295
HF Nus	0.070	0.378
LF/HF	-0.348	**< 0.001**
Nighttime HRV parameters		
Total power (ms^2)	0.052	0.512
ULF power (ms^2)	0.017	0.836
VLF power (ms^2)	0.107	0.179
LF power (ms^2)	-0.463	**< 0.001**
HF power (ms^2)	0.008	0.917
LF NUs	-0.002	0.982
HF NUs	-0.004	0.962
LF/HF ratio	-0.385	**< 0.001**

ULF: Ultra-low frequency, VLF: Very low frequency, LF: Low frequency, HF: High frequency, LF/HF: LF/HF ratio, NUs: Normalized units.

Table 4. Independent predictors of AVNRT

Variables	Multivariate OR, 95% CI	Multivariate p
Heart rate 24-h (bpm)	0.989 (0.945-1.035)	0.634
LV-EF	0.992 (0.879-1.121)	0.903
Daytime total power (ms^2)	1.000 (1.000-1.000)	0.999
Daytime ULF power (ms^2)	1.000 (1.000-1.001)	0.423
Daytime VLF power (ms^2)	0.999 (0.998-1.000)	**0.006**
Daytime LF power (ms^2)	1.002 (1.001-1.004)	**0.002**
Daytime HF power (ms^2)	0.999 (0.997-1.001)	0.213
Daytime LF/HF ratio	0.990 (0.607-1.616)	0.969
Nighttime LF power (ms^2)	1.003 (1.001-1.004)	**< 0.001**
Nighttime HF power (ms^2)	0.999 (0.998-1.000)	0.080
Nighttime LF/HF ratio	1.541 (0.875-2.717)	**0.135**

LV-EF: Left ventricular ejection fraction, HF: High frequency, LF: Low frequency, ULF: Ultra-low frequency, VLF: Very low frequency.

treatment of patients with symptomatic AVNRT in our study. HE et al. reported that empirical slow accessory pathway ablation is a safe and effective method[15]. Patients without tachycardia detection on ECG before ablation have a better outcome. Previous studies have shown that the application of RF current in the slow accessory pathway area can make AVNRT non-inducible despite persistent slow accessory pathway conduction[15,16].

The R-R intervals prior to ablation in children were longer than after ablation of the slow accessory AV pathway[1]. Furthermore, the 24-h Holter monitoring demonstrated that the mean and maximal HR were increased, whereas the HRV parameters were decreased after ablation of AV slow accessory pathway in children[17]. Interestingly, administration of isoproterenol after ablation to evaluate the slow accessory pathway and generate AV nodal echo beats led to no significant differences in the recurrence rates between patients with complete elimination of the slow pathway and patients with residual jump and/or single echo beat[18]. Nigro et al. showed that the HRV significantly changed before the onset of AVNRT[19]. They believed that the increased LF components during the prior hour before the onset of AVNRT was because of adrenergic predominance, and the decreased HF components suggested a parasympathetic drive. The pattern of the LF/HF ratio was compatible with the dynamic changes in autonomic tone. Our study also suggests that sustained typical AVNRT episodes are preceded by increase in adrenergic drive.

The fluctuations of autonomic tone before the onset of AVNRT were emphasized by the results of time-domain HRV analysis: SDNN and SDANN were decreased, implying an increase in the sympathetic tone with a decrease RMSSD and pNN50, which reflects vagal modulation[19]. In our study, the time-domain indices were not related to AVNRT. Cardiac autonomic conditions play a significant role in supraventricular tachyarrhythmia. The regularity of atrial and ventricular ectopic beats plays a vital role in tachycardia, antegrade and retrograde conduction, refractory periods of the AV node and accessory pathways. The association of neurohumoral activation and supraventricular tachycardia has been well documented[7,8]. It has been proposed that LF NUs is a sensitive marker of sympathetic modulation in control subjects, whereas the LF/HF ratio is a good marker for the sympathovagal balance[20-24]. According to our findings, the increase in LF components during the hour preceding the onset of AVNRT suggests an adrenergic predominance. The fluctuations of autonomic tone occurring before the onset of AVNRT may be underlined by the results of increased LF/HF ratio.

HRV is reduced in patients with diabetic neuropathy, but not in uncomplicated diabetes[25]. Multiple lesions in the atrium (including slow AV nodal pathway and fast AV nodal pathway) primarily affect sympathetic termination and lead to a decrease

in HRV[26]. Acute myocardial infarction changes the cardiac geometry, leading to an enhanced activity of sympathetic afferent activity and inhibition of vagal efferent activity[27]. HR increases in case of low hemoglobin levels in the blood. The increase in HR causes tachycardia and can trigger AVNRT via atrial ectopic beats[28]. Yo et al. showed that aging affects the LF/HF ratio in healthy and hypertensive subjects [29]. We excluded patients with diabetes, uncontrolled hypertension, documented cardiovascular disease, and hematological (including anemia) disorders from our study because these comorbidities may influence the autonomic nervous system.

CONCLUSION

HRV analysis has become an important cardiology tool because of its noninvasive and easy-to-implement features. Moreover, its measurements have relatively good reproducibility and provide useful prognostic information on patients with heart disease. This study showed that AVNRT was associated with altered sympathovagal balance. We considered that increased LF and LF/HF might be an indicative feature of enhanced sympathetic activity in patients with AVNRT as a result of inhomogeneous ventricular activation via the slow accessory pathway.

Limitations

In this single-center study, non-pharmacological evaluation of the autonomic tone was performed. Preprocedural hypovolemia, pain, and emotional stress can affect the autonomic tone and interfere with the HRV. Lack of postablation HRV domain indices was another limitation of the study. Future studies are warranted for these findings to be applied in clinical practice.

CONFLICT of INTEREST

The author reported no conflict of interest related to this article.

AUTHORSHIP CONTRIBUTIONS

Concept/Design: LA

Analysis/Interpretation: LA

Data Acquisition: LA

Writting: LA

Critical Revision: LA, ST

Final Approval: All of authors

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