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The Changes of von Willebrand Factor and Endocan Concentrations During and After Reversion of Paroxysmal Ventricular Tachycardia

*Wang Y[1], Feng XK[2], Ma GD[1], Jin H[1], Zhai Z[1] and Li C[3]**

[1]Department of Cardiovascular Medicine, Tsinghua University YuQuan Hospital, China

[2]Department of Respiratory and Critical Care Medicine, Beijing Chao-Yang Hospital, Capital Medical University, China

[3]Beijing Friendship Hospital, Capital Medical University, China

Abstract

Objective: To observe the risk factors of paroxysmal supraventricular tachycardia is important, in order to evaluate trigger conditions and their control. To observe the changes of von Willebrand Factor (vWF) and Endocan concentrations during and after reversion of Paroxysmal Ventricular Tachycardia (PSVT), and to explore the relationships between vWF Endocan and PSVT.

Methods: 100 adults with PSVT who were admitted to the rescue room of Beijing Chao-Yang Hospital, Capital Medical University from November 2016 to October 2019 were taken as a study group. The group was evaluated the risk factors of supraventricular tachycardia. It was further divided into three subgroups (32, 32 and 36 for each) based on the disease onset time ($T \leq 3$ h, 3 h $< T \leq 24$ h, and $T > 24$ h). In the meantime, 35 patients with sinus rhythm were also taken as study group for control analysis. All patients vWF and Endocan levels were measured by Enzyme-Linked Immunosorbent Assay (ELISA) for statistical analysis.

Results: 1. Alcohol intake, smoking, and stress were risk factors for paroxysmal supraventricular tachycardia. 2. The levels of vWF and Endocan in PSVT group were higher than those in control group ($P<0.01$). 3. The levels of vWF and Endocan in the three PSVT subgroups were increased gradually during the disease attack ($P<0.05$). 4. The levels of vWF and Endocan after PSVT were decreased significantly more than upon disease attack ($P<0.01$).

Conclusion: Alcohol intake, smoking, and stress were risk factors for paroxysmal supraventricular tachycardia. VWF and Endocan concentrations were significantly increased upon PSVT attack, but greatly decreased after the disease attack while still higher than those of the control group. The concentrations of vWF and Endocan in PSVT group were increased gradually in a time course manner, which showed that these two hormones involved in the pathophysiological process of PSVT. This phenomenon may be one of homeostasis regulation functions as the body's stress compensation mechanism.

Keywords: Paroxysmal ventricular tachycardia; Risk factors; von Willebrand Factor; Endocan

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Correspondence:
Chunsheng Li, Beijing Friendship Hospital, Capital Medical University, Beijing, China

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Introduction

Paroxysmal ventricular tachycardia is one of the most common cardiac arrhythmias in the emergency department. In 2015, the American College of Cardiology, the American Heart Association and the American Heart Association (ACC/AHA/HRS) syndicated that the prevalence of SVT in the general population was 2.25 per 1,000 persons [1]. When adjusted by age and sex in the U.S. population, the incidence of PSVT was estimated to be 36 per 100,000 persons per year. There were approximately 89,000 new cases per year and 570,000 persons with PSVT [1]. PSVT affected not only the life quality of the patients, but also did harm to the health of the patients. Current researches show that the pathogenesis of PSVT is mainly the presence of atrioventricular accessory pathway or differences in functional conductivity with nonstress of atrioventricular nodal function. The treatments haven't been changed in the last decade, but in recent years more emphasizes have been put on the choice of drug therapy and catheter ablation in patients with healthy problems. With the increasing number of the supraventricular tachycardia case, tachycardia induced-hemodynamic changed seriously impact the quality of patients' life, thus the study of patients' neurohormonal

became more and more important. Von Willebrand factor (vWF) is a large multimeric plasma glycoprotein produced by endothelial cells and megakaryocytes [2]. It is an important vital endothelial damage indicator [3]. Endocan is a newly identified human endothelial cell-specific molecule [4]. It is expressed in the vascular endothelium, and its secretion is regulated by cytokines and growth factors [5]. This study was the first time to investigate the changes of vWF and Endocan concentrations during and after reversion of PSVT, and to explore the relationships between vWF Endocan and PSVT.

Subject and Methods

Study design and patients

This study was approved by the Medical Ethics Committee of Beijing Chao-yang Hospital (2013 department 124). One hundred adults with PSVT who were admitted to the rescue room of Beijing Chao-Yang Hospital, Capital Medical University from November, 2016 to October, 2019 were chosen as a study group, in which 63 were males and 37 were females. This group was further divided into three subgroups (32, 32 and 36 for each) based on the disease onset time ($T \leq 3$ h, 3 h $< T \leq 24$ h, and $T > 24$ h). In the meantime, 35 patients with sinus rhythm were also taken as study group for contrast analysis, which included 25 males and 10 females. Diagnosis was confirmed by 12 lead electrocardiography which showed a regular tachycardia of 150 to 250 beats per minute with absent P waves and a narrow QRS complex (<0.12 ms) [2]. Exclusion criteria: (1) Left Ventricular Ejection Fraction (LVEF) <50%. (2) Hyperthyroidism or hypothyroidism patients. (3) Patients with cardiomyopathy. (4) Rheumatic heart disease. (5) Acute coronary syndrome. (6) Liver or kidney function insufficiency. (7) Electrolyte disorder patients. (8) Patients who cannot cooperate with the inspection of the patients.

Data acquisition

Patients with confirmed PSVT were taken into in the PSVT group. Regular monitoring were performed, including blood pressure, heart rate, respiratory rate, body mass index, alcohol intake, smoking, stress, glucose, cholesterol, triglyceride, lactate dehydrogenase, creatine kinase, creatinine, urea nitrogen, electrolyte, heart color ultrasound examination. Normal venous blood was extracted when the patients were enrolled in this study. Another 4 ml venous blood was extracted after reversion for 30 min and followed by centrifuging. The serum samples were stored at -80°C for preparing the detection of vWF and Endocan. During the same period, the control group was taken out of the venous blood four ml, and the serum samples were kept in -80°C for preparing the detection of vWF and Endocan.

Main reagents and instruments

Serum concentrations of vWF and Endocan were measured using available quantitative enzyme linked immunosorbent assay kits (vWF and Endocan ELISA kits, CEA833Hu, USA, SEC463Hu, USA).

Statistical analysis

All data was expressed as the mean ± S.E.M and analyzed using the SPSS 22.0 statistical analysis software (SPSS Inc, Chicago, IL, USA). T-test was used to analyze the differences between the data and the control group, A P-value of less than 0.05 or 0.01 was considered to be statistically.

Results

There were no significant differences of laboratory parameters, including body mass index, glucose, cholesterol, triglyceride, lactate dehydrogenase, sodium, potassium, creatinine, urea, PH values, arterial partial pressure of oxygen, partial pressure of carbon dioxide, and UCG did not differ significantly between the two groups (Table 1, 2). Alcohol intake, smoking, and stress were significant between the two groups (Table 1). Respiratory rate was similar during and after reversion of PSVT (Table 3). The systolic blood pressure diastolic blood pressure and mean arterial pressure were lower at the time of the attack than after reversion with difference (Table 3). Heart rate was higher than after reversion (Table 3). The levels of vWF and Endocan were higher during the whole PSVT period (Table 4 and Figure 1, 2). During the attack, significant increasing of both vWF and Endocan levels were observed in three subgroups (Table 5 and Figure 3, 4).

Table 1: Comparison of the clinical data between PSVT group and control group.

Group	PSVT group	Control group	P-Value
n	100	35	
Age (years)	53.15 ± 10.53	49.34 ± 8.90	0.058
Gender (man/female)	63/37	25-Oct	0.163
Body mass index (kg/m^2)	25.66 ± 1.51	25.31 ± 1.47	0.889
Alcohol intake	56/44	Oct-25	0.005
Smoking	33/67	Mar-32	0.005
Stress	42/58	0/35	0
K (mmol/L)	4.07 ± 0.19	4.02 ± 0.17	0.176
Na (mmol/L)	139.23 ± 1.86	139.94 ± 1.86	0.052
CREA (umol/L)	75.69 ± 8.66	75.53 ± 9.26	0.929
BUN (mmol/L)	4.67 ± 0.37	4.54 ± 0.36	0.064
Glu (mmol/L)	5.46 ± 1.25	5.22 ± 0.42	0.308
Tc (mmol/L)	4.41 ± 1.29	4.53 ± 0.71	0.656
TG (mmol/L)	1.11 ± 0.58	0.94 ± 0.34	0.135
LDH (u/L)	163.66 ± 30.73	161.82 ± 26.51	0.734
CK (u/L)	83.21 ± 26.66	73.33 ± 23.62	0.112
PH	7.40 ± 0.12	7.39 ± 0.01	0.085
PaO_2 (mmHg)	97.60 ± 2.44	98.43 ± 1.93	0.07
$PaCO_2$ (mmHg)	40.37 ± 1.58	39.83 ± 1.56	0.085

Table 2: Comparison of UCG between PSVT group and control group.

UCG	PSVT	Control	P
LA (mm)	30.71 ± 3.31	29.94 ± 2.89	0.328
RA (mm)	44.31 ± 3.66	40.71 ± 3.01	0.524
LVD (mm)	47.34 ± 3.34	48.63 ± 2.86	0.106
LVS (mm)	29.17 ± 2.44	29.14 ± 2.93	0.964
RV (mm)	29.11 ± 2.45	29.37 ± 1.88	0.633
PA (mm)	22.37 ± 2.16	21.37 ± 1.83	0.051
LVEF (%)	67.77 ± 4.15	68.60 ± 3.88	0.405

Discussion

Paroxysmal supraventricular tachycardia risk, as shown by the study, was influenced by a number of clinical factors such as: Alcohol intake, smoking, and stress. A number of studies demonstrate the causal relationship between some risk factors and arrhythmias [6,7].

Scridon et al. [8] identified plasma vWF concentrations in patients with atrial fibrillation were significantly higher than that in sinus rhythm group. Cortés et al. [9] described vWF levels in patients with atrial fibrillation were higher than that of healthy people. The

Table 3: Respiratory rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, heart rate comparison $(\overline{x} \pm S)$.

	Before	After	P-Value
RR (rate/minute)	19.42 ± 1.65	19.22 ± 1.64	0.555
SBP (mmHg)	102.83 ± 6.69	123.62 ± 6.65	0
DBP (mmHg)	63.80 ± 4.72	71.38 ± 4.49	0
MAP (mmHg)	76.77 ± 4.61	88.71 ± 3.93	0
HR (rate/minute)	166.48 ± 10.47	81.13 ± 6.40	0

Table 4: Comparison of vWF and Endocan levels during and after reversion of PSVT and control group $(\overline{x} \pm S)$.

group	n	vWF (pg/ml)	Endo (ng/ml)
	100	3193.31 ± 657.61	51.93 ± 11.43
Reversion of psvt	100	2603.01 ± 476.85	46.37 ± 8.11
Control group	35	1341.86 ± 425.69	30.23 ± 8.32
χ^2/Φ		122.316	58.703
P-value		0.000	0.000

Table 5: Comparison of vWF and Endocan concentrations by time (T) in the time of onset of PSVT group $(\overline{x} \pm S)$.

group	n	vWF (pg/ml)	Endo (ng/ml)
T ≤ 3h	32	2427.84 ± 424.33^D	43.47 ± 11.49^D
3h<t ≤ 24h	32	3053.81.70 ± 475.64^D	51.09 ± 9.66^D
t>24h	36	3626.03 ± 376.21*	56.17 ± 9.91*
χ^2/Φ		47.737	13.458
P-value		0	0

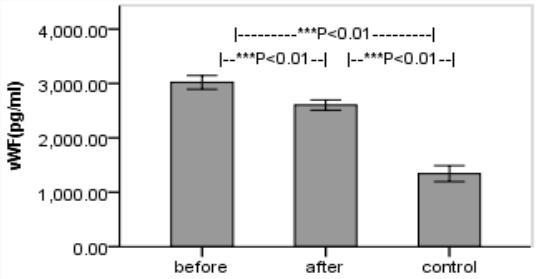

Figure 1: The concentrations of vWF during and after reversion of PSVT and control group.

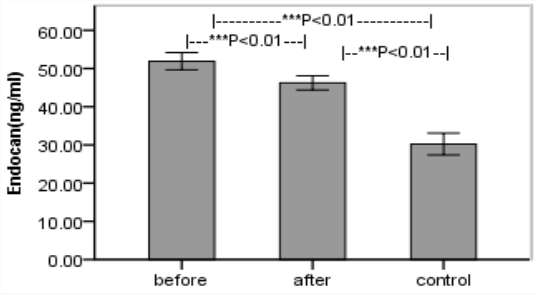

Figure 2: The concentrations of Endocan during and after reversion of PSVT and control group.

recent study showed plasma vWF was significantly elevated in atrial fibrillation patients compared with controls [10]. Further studies showed that there were actually differences between 3 types of atrial fibrillation (paroxysmal, persistent, or permanent), with the highest

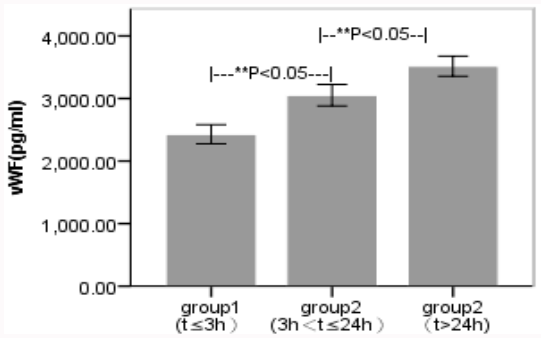

Figure 3: Comparison of changes of vWF concentrations in the sub-groups during the attack of PSVT.

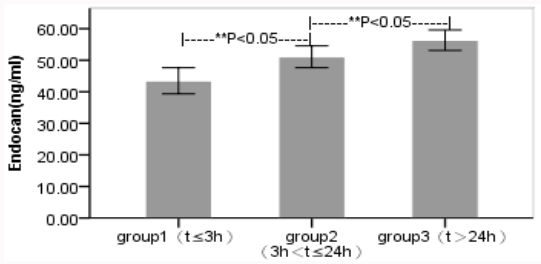

Figure 4: Comparison of changes of Endocan concentrations in the sub-groups during the attack of PSVT.

levels of vWF in patients with permanent atrial fibrillation [9,11].

VWF was recognized as a plasma biomarker of endothelial injury/dysfunction and elevated levels can predict adverse cardiovascular outcomes [12]. In this study, we showed, for the first time, that vWF levels were significantly elevated in PSVT group as compared to controls. Our further research showed that vWF levels were increased with prolonged attack time. The elevation of vWF was proposed to be attributed to endothelial damage. The shear stress of vascular wall affects the integrity and function of endothelial cells, resulting in the secretion of vWF by vascular endothelial cells [13].

Our study also showed that endocan levels in the chamber of heart tachycardia were significantly higher and increased gradually during the disease attack, suggesting that PSVT may exist in the process of activation of endothelial function. Endocan was a kind of endothelial cells to secrete soluble chondroitin sulfate proteoglycans [14]. There were many reports that Endocan is highly expressed in tumors, sepsis, obesity, or inflammatory diseases, and Endocan is a novel marker of vascular endothelial cell function [15]. Balta et al. [16] explored that Endocan was a marker of cardiovascular disease in patients with psoriasis. Sevket et al. [17] found that the serum Endocan levels in patients with hypertension were significantly higher than control group. Wang et al. [18] found that the levels of serum Endocan in 164 hypertensive patients were independently correlated with coronary heart disease and the severity of coronary heart disease. Xiong et al. [19] also found that the levels of serum Endocan in patients with hypertension were correlation with coronary heart disease. These conclusions suggested high Endocan levels can make the risk of atherosclerosis increased. Kose et al. [20] assessment of patients with acute coronary syndrome serum Endocan levels were significantly higher than the healthy control group and were increased with prolonged attack time. On the cause of the rise of endocan, some studies found increased vascular laminar shear led to downregulation

of endothelial Nitric Oxide Synthase (eNOS) activity [21,22], decreased eNOS activity contributed to the decrease of Nitric Oxide (NO) bioavailability [22]. Supraventricular tachycardia associated with vascular endothelial dysfunction although the mechanism was not clear. The relationship between PSVT and vascular endothelial dysfunction was caused by oxidative stress.

Limitations

The effects of drugs on vWF and Endocan during and after reversion of PSVT weren't considered. The detection of vWF and Endocan was susceptible to a variety of factors, while the half-life and stability weren't the same.

Conclusion

In summary, alcohol intake, smoking, and stress were risk factors for paroxysmal supraventricular tachycardia. The changes of vWF and Endocan levels were not only observed in cardiovascular disease but also in arrhythmia. This phenomenon may be one of homeostasis regulation functions as the body's stress compensation mechanism.

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