



# The relationship between blood glucose and nocturnal supraventricular tachycardia attacks in non-diabetic patients

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Received: 4 September 2018 / Accepted: 3 April 2019 / Published online: 30 April 2019  
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## Abstract

**Purpose** Decrease in the blood glucose level may trigger the tachycardia or bradycardia because it has an arrhythmogenic effect on the heart. Our purpose in this study was to investigate whether the blood glucose level has an effect on patients who attended to the hospital with nocturnal supraventricular tachycardia (SVT).

**Methods** We included 151 patients in our study who have SVT history. Plasma glucose levels which were taken during night hours, electrolytes, and 12 lead electrocardiography were evaluated.

**Results** There were 105 patients without nocturnal SVT attack and 46 patients with nocturnal SVT attack. Patients with nocturnal SVT attack, blood glucose level, potassium, calcium, and hemoglobin levels were significantly lower, hs-CRP was significantly higher, basal cycle length (BCL) was significantly short, and QT interval was significantly longer. It was found that blood glucose (O.R. = 0.904, 95% GA 0.828–0.986,  $p = 0.023$ ) and potassium levels (O.R. = 0.128, 95% GA 0.029–0.561,  $p = 0.006$ ) and basal cycle length (BCL) (O.R. 0.988, 95% GA, 0.980–0.996,  $p = 0.005$ ) values were in independently correlated with nocturnal SVT attacks.

**Conclusion** The decrease in blood glucose level of the patients who are being followed with SVT diagnosis might trigger the nocturnal SVT attacks.

**Keywords** Supraventricular tachycardia · Blood glucose · Non-diabetics · QT interval

## 1 Introduction

Supraventricular tachycardias (SVTs) are arrhythmias that begin and end suddenly, are characterized by high heart rate, and affect the life quality negatively [1]. Emotional stress and stimulant nutrients such as tea, coffee, and coke might trigger the attacks in the daytime. As the external stimuli decrease in number during sleep at night hours, the parasympathetic system increases its influence, and the body starts a general relaxing state [2].

Hypoglycemia is defined as the blood glucose level being below 70 mg/dL [3]. During hypoglycemia, the

sympathoadrenal system gets activated and starts trying to balance the blood glucose level [4, 5]. In addition, hypoglycemia might trigger the hypokalemia. The conducted studies have revealed that hypoglycemia has an arrhythmogenic potential and can cause sudden cardiac deaths as it results in direct sympathoadrenal system activation, hypokalemia, and a notable prolonging in QT interval [6–10].

While it is shown in some studies that the spontaneous hypoglycemia attacks seen at nights cause bradycardia, some other studies showed that they cause tachycardia by triggering atrial and ventricular arrhythmias [11–13]. In the literature, there is not a clear data mentioning that hypoglycemia triggers SVT attacks except from a few sporadic cases [14, 15]. Our purpose in this study is to find out whether the blood glucose level has an effect on these attacks in the patients who attended to the hospital with nocturnal SVT attacks.

## 2 Method

**Patient selection** In our retrospective study, patients who were admitted with SVT attack to the emergency department at

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**Table 1** Comparison of patients' demographic findings

	Without nocturnal SVT ( <i>n</i> = 105)	With nocturnal SVT ( <i>n</i> = 46)	<i>p</i>
Age (years)	49.1 ± 5.8	48.6 ± 5.5	0.602
Male gender, (%)	43 (41.0)	12 (26.1)	0.081
Systolic blood pressure (mmHg)	117.1 ± 12.5	119.4 ± 11.1	0.241
Diastolic blood pressure (mmHg)	77.1 ± 10.6	77.4 ± 10.9	0.488
BMI (kg/m <sup>2</sup> )	24.3 ± 1.5	24.5 ± 2.6	0.496
Smoking, <i>n</i> (%)	10 (19.0)	10 (21.7)	0.610
HT, <i>n</i> (%)	10 (9.5)	7 (15.2)	0.832
HPL, <i>n</i> (%)	10 (9.5)	2 (4.3)	0.347
SVT attack number, <i>n</i>	5.2 ± 2.2	4.9 ± 1.9	0.477
SVT attack duration (min)	52.8 ± 25.6	43.5 ± 27.7	0.066
Body temperature (°C)	36.3 ± 0.3	36.4 ± 0.4	0.259

HT hypertension, HPL hyperlipidemia, SVT supraventricular tachycardia

night hours (00:00–06:00 am) were included from hospital records between May 2015 and May 2018. Also, control group patients who were admitted to the arrhythmia clinic with SVT attack for treatment or observation and who did not have SVT attack at night were included. There were no daytime SVT episodes in patients with nocturnal SVT attack, and there was no nocturnal SVT attack in our control group patients. The patients with atrial fibrillation, diabetes, whose blood glucose levels were not measured at night hours, who have structural heart disease, pregnant, patients with SVT attacks both at night and at day (8 attacks in 6 patients), and whose laboratory data we could not access were excluded from the study. All patients were called by telephone to confirm that they had night SVT attacks. The demographical data of the patients were recorded. Ethical approval was taken from the regional ethics committee.

**The evaluation of laboratory findings** Blood glucose level, kidney function tests, sodium, potassium, calcium levels, complete blood count, and thyroid-stimulating hormone levels were examined from the routine blood tests taken at

**Table 2** Comparison of patients' medications

	Without nocturnal SVT ( <i>n</i> = 105)	With nocturnal SVT ( <i>n</i> = 46)	<i>p</i>
ACE ( <i>n</i> , %)	8 (7.6)	5 (10.9)	0.512
ARB ( <i>n</i> , %)	4 (3.8)	2 (4.3)	0.876
B-blocker ( <i>n</i> , %)	46 (43.8)	15 (32.6)	0.197
Ca channel blocker ( <i>n</i> , %)	12 (15.4)	7 (15.2)	0.518
Statin ( <i>n</i> , %)	4 (12.1)	43 (22.5)	0.176

ACE angiotensin-converting enzyme, ARB angiotensin receptor blocker, ASA acetylsalicylic acid, SVT supraventricular tachycardia

night hours (00:00–06:00 am) in patients with nocturnal SVT and at daytime hours in patients without nocturnal SVT were recorded during attacks.

**The evaluation of 12 derivations electrocardiography** 12 leads electrocardiography (ECG) were evaluated in patients with or without SVT, which was taken at 25 mm/s speed and 10 mm/mV amplitude (MAC 2000, GE Medical, Milwaukee, WI, USA). Tachycardia cycle length (TCL) was calculated from the SVT electrocardiogram of the patients. Basal cycle length (BCL) and QT and QTC intervals were measured from the electrocardiogram of patients without SVT (Bazget formula was depended on).

**Statistical analysis** Data were analyzed using SPSS for Windows 20.0 (SPSS, Chicago, IL, USA). Variables were divided into categorical and continuous groups. Categorical variables were expressed as frequencies and percentages and were analyzed using the Chi-square test. Kolmogorov-Smirnov test was used to determine whether continuous variables had normal distribution or not. Continuous variables were expressed as a mean ± standard deviation. Normal distributed variables were analyzed with independent samples *t* test. Not normal distributed variables were analyzed with the Mann-Whitney *U* test. Independent predictors were determined by the binomial logistic regression analysis using *p* < 0.05 variables. Receiver operating characteristic (ROC) analysis was used to calculate sensitivity and specificity values.

### 3 Findings

We included 105 patients without nocturnal SVT attack (average age 49.1 ± 5.8 years) and 46 patients with SVT attack (average age 48.6 ± 5.5 years) in our study. Ninety-eight of these patients had atrioventricular nodal re-entrant tachycardia (AVNRT), and 53 of them had atrioventricular reciprocal tachycardia (AVRT). The demographical data and medical treatment of both groups were similar to each other (Tables 1 and 2). When the laboratory findings were considered, blood glucose level (*p* < 0.001), potassium (*p* < 0.001), calcium (*p* = 0.014), and hemoglobin levels were significantly lower in the group of patients with SVT, hs-CRP (*p* = 0.008) was significantly higher, and the other findings were similar (Table 3). When ECG data of both groups were compared, it was found that BCL was significantly shorter (*p* < 0.001) and QT interval was significantly longer (*p* = 0.02) in the group of patients with SVT, and the other findings were similar to each other (Table 4). In the binomial logistics regression analysis, blood glucose (O.R. = 0.904, 95% GA 0.828–0.986, *p* = 0.023) and potassium levels (O.R. = 0.128, 95% GA 0.029–0.561, *p* = 0.006) and BCL values (O.R. 0.988, 95% GA

**Table 3** Comparison of patients' laboratory findings

	Without nocturnal SVT (n = 105)	With nocturnal SVT (n = 46)	p
Glucose (mg/dL)	91.0 ± 12.7	81.3 ± 13.4	< 0.001
Hypoglycemia, n (%)	9 (8.6)	14 (30.4)	0.001
BUN (mg/dL)	30.8 ± 8.5	29.9 ± 8.3	0.552
Cr (mg/dL)	0.8 ± 0.2	0.8 ± 0.1	0.412
Na (mmol/L)	137.1 ± 3.4	138.2 ± 2.7	0.601
K (mmol/L)	4.4 ± 0.4	3.7 ± 0.8	< 0.001
Calcium (mmol/L)	9.0 ± 0.5	8.6 ± 0.9	0.014
TSH	1.2 ± 0.7	1.6 ± 0.7	0.085
WBC (uL)	8.8 ± 2.0	9.1 ± 1.7	0.605
Hb (mg/dL)	13.1 ± 1.2	11.8 ± 1.6	< 0.001
Neutrophil (%)	61.6 ± 6.4	61.1 ± 11.1	0.724
Lymphocyte	24.8 ± 9.1	23.1 ± 9.3	0.304
NLR	3.6 ± 4.6	3.8 ± 3.6	0.883
Hs-CRP (mg/L)	0.7 ± 0.5	1.0 ± 0.6	0.008
Hs-TnT	9.8 ± 6.2	10.5 ± 4.8	0.494

BUN blood urea nitrogen, Cr creatinine, HDL high-density lipoprotein, Hs-CRP high-sensitive C reactive protein, Hs-TnT high-sensitive troponin T, Hb hemoglobin, LDL low-density lipoprotein, SVT supraventricular tachycardia, TSH thyroid-stimulating hormone, NT-proBNP N-terminal brain natriuretic peptide, WBC white blood cells

0.980–0.996,  $p = 0.005$ ) were independently correlated with nocturnal SVT attacks (Table 5). In the ROC analysis, it was seen that the blood glucose level, when taken as 86.5, predicted the night SVT attacks with 71.6% sensitivity and 73.9% specificity (AUC 0.795 (0.721–0.869),  $p < 0.001$ , Fig. 1). It was also determined that the potassium level, when taken as 3.9, predicted nocturnal SVT attacks with 73% sensitivity and 67% specificity (AUC 0.754 (0.659–0.848),  $p < 0.01$ , Fig. 2).

### 4 Discussion

Our study is the first one which was conducted with the patients with nocturnal SVT attacks, and a few important

**Table 4** Comparison of ECG findings

	Without nocturnal SVT (n = 105)	With nocturnal SVT (n = 46)	p
BCL (ms)	818 ± 91	747 ± 64	< 0.001
TCL (ms)	350 ± 29	353 ± 15	0.491
Basal QT	355 ± 36	371 ± 39	0.02
Basal QTc	410 ± 33	410 ± 32	0.982
QT dispersion	31 ± 14	29 ± 13	0.567

BCL basal cycle length, TCL tachycardia cycle length, QTc corrected QT, Disp dispersion, SVT supraventricular tachycardia

**Table 5** Independent predictors for nocturnal SVT episode

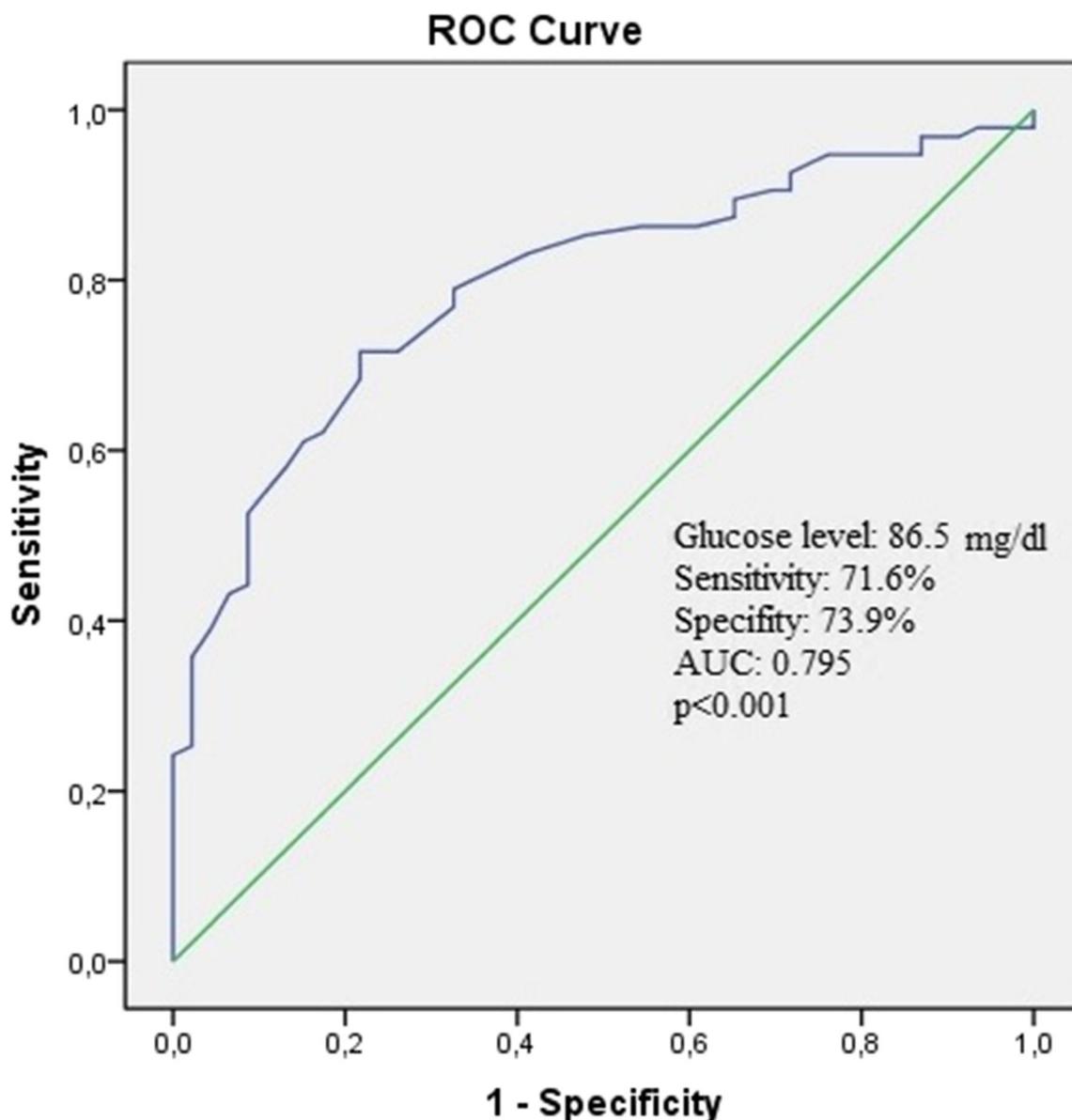
	Odds ratio	95% confidence interval	p
Glucose (every 1 mg/dL increase)	0.904	0.828–0.986	0.023
Hypoglycemia (present/absent)	1.324	0.159–11.006	0.795
Potassium	0.128	0.029–0.561	0.006
Calcium	3.377	0.313–36.457	0.316
Hemoglobin	0.428	0.171–1.071	0.07
Hs-CRP	0.969	0.937–1.002	0.062
Basal QT	1.005	0.988–1.028	0.458
BCL	0.988	0.980–0.996	0.005

Hs-CRP high-sensitive C reactive protein, BCL basal cycle length, SVT supraventricular tachycardia

findings were obtained. These are the significantly low blood glucose level and potassium level and significantly high basal heart rate in the patients with nocturnal SVT attacks.

In the guidelines of diabetes mellitus, hypoglycemia is defined as the blood glucose levels below < 70 mg/dL [3]. It was shown that hypoglycemia triggers the cardiac arrhythmias in both direct and indirect ways [7, 8, 16, 17]. In hypoglycemia, increase in heart rate, shortening of PR interval, ST segment depression, T wave flattening, and prolongation of QTC interval are observed due to the effect of catecholamine discharge [18]. We did not include the patients with diabetes into our study as the autonomic mechanisms break down in time. Although the number of the patients with hypoglycemia was found significantly higher in the group of patients with nocturnal SVT attacks in the univariate analysis, it was not determined as an independent variable in the multivariable analysis. The levels of blood glucose and potassium were found significantly low, basal heart rate was found significantly higher, and they were determined independently correlated with nocturnal SVT attacks. As a matter of fact, we did not expect the number of our patients with hypoglycemia higher as they were not diabetic and were not using diabetic drugs. The level of potassium might have decreased due to the decreased blood glucose level, and heart rate might have increased due to stress in the group of the patients with nocturnal SVT attacks. The decline in the levels of both blood glucose and potassium might have triggered SVT attacks. Besides, a stronger sympathoadrenal response might have been given against the decreased blood glucose levels, and heart rate might have increased in these patients compared to the groups of patients without nocturnal SVT. If we had measured the levels of epinephrine, norepinephrine, and stress hormones in the group of patients with night SVT, we could have found them higher.

In a meta-analysis in which many studies were evaluated, it was reported that silent hypoglycemia attacks might have



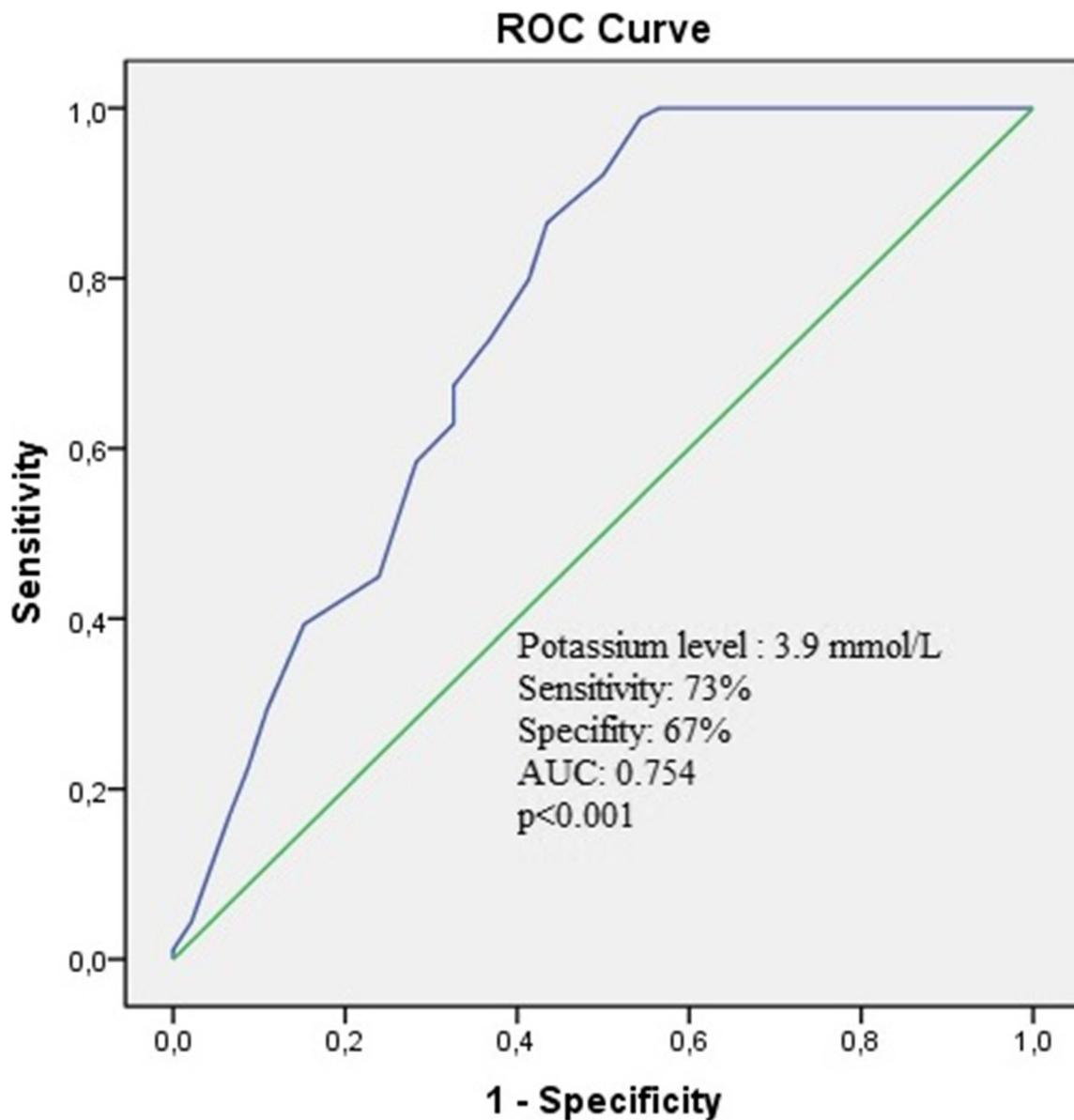
**Fig. 1** Receiver operating characteristic (ROC) curve analysis to determine predictive value of blood glucose level for nocturnal supraventricular tachycardia attacks

triggered the cardiac arrhythmias [19]. In a study which was conducted with the patients with type1 diabetes to investigate the diurnal effect of spontaneous hypoglycemia on cardiac arrhythmias, bradycardia and atrial ectopic beats were observed more often in nocturnal hypoglycemia [20]. In our study, it was determined that the low blood glucose level might trigger SVT attacks. As the patients are diabetic in this study, intake of exogenous insulin decreased the blood glucose level to hypoglycemia level. Due to improper function of sympathoadrenal mechanism and parasympathetic system's activation during sleep, bradycardia might have been observed. In our study, hormonal mechanisms might have blocked the occurrence of bradycardia as the patients were not diabetic.

Some medications (for example some beta-blockers) may mask the sympathoadrenal response during hypoglycemia [21]. In our study, the use of beta-blockers was similar in both groups. Therefore, we do not believe that the use of beta-blockers is effective in night SVT attacks.

## 5 Limitations

The most significant limitation of our study is that it is a retrospective study. We do not know if there is a real decrease in glucose and potassium levels because we do not know the basal laboratory values of the patients. The levels of epinephrine, norepinephrine, and stress hormones and insulin levels



**Fig. 2** Receiver operating characteristic (ROC) curve analysis to determine predictive value of serum potassium level for nocturnal supraventricular tachycardia attacks

were not measured. In order to say that blood glucose level triggers the night SVT attacks, studies that are conducted with loads of patients are needed.

## 6 Conclusion

In the patients who experience SVT attacks, it is important to pay attention to low night blood glucose level. The patients should be warned about this matter, and they can be recommended to take nutrients that can increase the blood glucose level before going to bed at nights.

## Compliance with ethical standards

The Adana City Training and Research Hospital Ethics Committee approved our retrospective study.

**Conflict of interest** The authors declare that they have no conflict of interest.

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