Factors Influencing Digoxin Efficacy in Supraventricular Tachyarrhythmia: The Role of Hemoglobin and Renal Function

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Abstract

Aim: Supraventricular tachycardia (SVT) is a common arrhythmia requiring effective rate control. As a cardiac glycoside Digoxin is mostly utilized in infants for this indication. However, factors influencing its efficacy in managing SVT in adult patients are not thoroughly evaluated. Our study investigates the impact of hemoglobin and renal function on digoxin's ability to achieve rate control in adult SVT patients.

Methods: We conducted a retrospective, single-center observational study with 167 adult patients presenting with SVT and treated with digoxin. Patients were categorized into Rate Control Group (RCG, n=58) and Non-Rate Control Group (N-RCG, n=109) based on the success of rate control. Clinical and laboratory parameters, including hemoglobin, renal function (GFR, renal failure), and patient outcomes (discharge, hospital/ICU admission) of both patient groups were analyzed and compared.

Results: Patients achieving rate control (RCG) exhibited significantly higher hemoglobin levels (13.6 ± 2.5 g/dL vs. 12.7 ± 2.5 g/dL, p=0.010) and GFR (60.7 ± 27.3 vs. 58.7 ± 25.5 ml/min/1.73m2, p=0.015), with lower incidence of renal failure (3.4% vs. 17.4%, p=0.009) compared to N-RCG. Multivariate logistic regression revealed that hemoglobin remained a significant independent positive predictor of rate control (Adjusted OR: 1.154, 95% CI: 1.009-1.321, p=0.037), while renal failure and GFR did not retain statistical significance. No significant difference was observed in overall patient outcomes (discharge, hospital/ICU admission) between the groups (p=0.302).

Conclusions: Higher hemoglobin levels independently predict successful rate control with digoxin in adult SVT patients. While renal function is still critical for digoxin pharmacokinetics, its direct influence on rate control success may be confounded by other factors. Further research is warranted to explore the mechanisms linking hemoglobin and GFR to digoxin efficacy and the long-term clinical implications of rate control.

Keywords: *Digoxin; supraventricular tachyarrhythmia; anemia; heart rate control; renal failure*

1. Introduction

Supraventricular tachycardia (SVT) encompasses a variety of tachyarrhythmias originating above the ventricles, characterized by rapid heart rates typically ranging from 150 to 220 beats per minute. These rapid rhythms can lead to a spectrum of symptoms and in more severe or sustained cases, SVT can precipitate hemodynamic instability, myocardial ischemia, or even tachycardia-induced cardiomyopathy.[1] Effective and timely rate control is crucial for managing acute SVT episodes and preventing adverse cardiovascular sequelae.

Digoxin, a cardiac glycoside derived from the *Digitalis lanata* plant, had previously utilized as a rate-control agent in supraventricular tachycardia, particularly in cases of AV nodal reentrant tachycardia. Its primary mechanism of action involves the reversible inhibition of the myocardial sodium-potassium ATPase (Na+/K+ ATPase) pump, which leads to an increase in intracellular sodium in cardiac myocytes via the sodium-calcium exchanger. The resulting increase in intracellular calcium enhances myocardial contractility and produces a positive inotropic effect. Related to SVT manage-

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ment digoxin increases vagal tone, which significantly slows conduction through the atrioventricular (AV) node by prolonging its refractory period.[2] Despite its therapeutic utility, digoxin possesses a narrow therapeutic window, necessitating careful dosing and careful monitoring to avoid toxicity.

Baseline hemoglobin levels and renal function influence digoxin's pharmacokinetics, pharmacodynamics, and physiological response of the indiviudal. Furthermore, hemoglobin levels can impact the underlying physiological state of the patient, as anemia can lead to a hyperdynamic circulatory state and potentially making heart rate control more challenging and reducing digoxin's effectiveness.[3] Digoxin is predominantly eliminated by the kidneys and since its clearance is directly proportional to GFR, impaired renal function can also significantly prolong its half-life if doses.[4] Understanding the interplay between these parameters and digoxin's efficacy is crucial for optimizing therapeutic outcomes in adult SVT patients.

Our study aims to evaluate the specific factors, including hemoglobin and renal function, that influence achieving rate and rhythm control in adult patients presenting with supraventricular tachycardia and was administered digoxin.

2. Materials and Methods

2.1. Study Design and Setting

Our study was conducted as a retrospective single-center study. Data were collected from the emergency department of a tertiary hospital, spanning a four-year period from January 1, 2021, to December 31, 2024. Our study design adheres to the retrospect scope guideline proposed by Kaji et al. to mitigate potential biases inherent in retrospective analyses of emergency department data 5. Ethical approval for the study was obtained from the local ethics committee.

2.2. Patient Selection

Adult patients aged 18 years or older, who presented with a diagnosis of supraventricular tachycardia (SVT) and subsequently received digoxin treatment were identified through a retrospective review of the hospital automation system. The identification of SVT diagnoses was based on the International Classification of Diseases, 10th Revision (ICD-10) code I47.1. Exclusion criteria for patient enrollment included following: unavailability of complete data, presence of cardiac devices such as pacemakers, initial treatment involving electrical cardioversion, ST-elevated myocardial infarction on electrocardiogram (ECG), or documented treatment of any antiarrhythmic therapy within the 8 hours before digoxin administration. Accuracy of selected patient data forms was independently confirmed by a second emergency medicine specialist through random review to ensure data integrity. Patients were categorized into two distinct groups based on their response to digoxin therapy: the Rate Control Group (RCG), comprising those who achieved successful heart rate control, and the Non-Rate Control Group (N-RCG), consisting of those who did not achieve rate control.

2.3. Data Sources and Collection

Clinical and laboratory data were extracted via the hospital's electronic automation system. This included demographic details (age, gender), documented comorbidities, a history of concurrent medication use, digoxin dosages administered, the number of digoxin administrations, and the sequence of drug administration if multiple agents were used. Vital signs including pre- and post-administration blood pressure, heart rate, mean arterial pressure and oxygen saturation were recorded. Left ventricular ejection fractions (LVEF) at the time of admission were obtained from cardiology consultation notes. Blood test results on time of admission, such as blood lactate levels, base deficit, pH, partial oxygen pressure, and partial carbon dioxide pressure, were also collected.

2.4. Outcomes and Definitions

The primary outcome of this study was defined as the identification of factors influencing the achievement of the primary endpoint of rate control in patient population. Patient disposition outcomes were also evaluated including discharge status, hospital admission, and intensive care unit (ICU) admission rates, as indicators of overall clinical course following SVT management.

2.5. Statistical Analysis

All collected data were analyzed using SPSS 26.0 (Armonk, NY: IBM Corp.). Continuous variables were assessed for normal distribution using the Shapiro-Wilk test. Parameters demonstrating a normal distribution were presented as mean ± standard deviation, while those not normally distributed were expressed as median (interquartile range). Comparisons of continuous variables between the RCG and N-RCG were performed using the Student's t-test for normally distributed data and the Mann-Whitney U test for non-normally distributed data. Categorical variables were analyzed using the Chi-Square test or Fisher's exact test. Univariate and multivariate logistic regression analyses were employed to identify independent predictors of successful heart rate control. A p-value of less than 0.05 was considered as statistically significant.

3. Results

A total of 167 adult patients comprised 97 women (58%) and 70 men (42%) and received digoxin treatment for SVT were included in the analysis. Of all patients, 58 patients (34.7%) achieved successful heart rate control and were classified into the Rate Control Group (RCG), while 109 patients (65.3%) did not achieve rate control and constituted the Non-Rate Control Group (N-RCG). No statistically meaningful difference in mean age was observed between the RCG (p=0.135).

Significant differences were noted between the groups regarding some of the laboratory characteristics. Renal failure was significantly more prevalent in the N-RCG (17.4%, n=19) compared to the RCG (3.4%, n=2) (p=0.009), and the mean Glomerular Filtration Rate (GFR) was significantly lower in the N-RCG (58.7 ± 25.5 ml/min/1.73m2) compared to the RCG (60.7 ± 27.3 ml/min/1.73m2) (p=0.015). Hemoglobin levels were significantly higher in the RCG (13.6 ± 2.5 g/dL) compared to the N-RCG (12.7 ± 2.5 g/dL) (p=0.010). Other baseline characteristics including gender, systolic and diastolic blood pressure, baseline heart rate, prevalence of heart failure, LVEF, TnI, creatinine, Pro-BNP, calcium, potassium, and sodium, did not show statistically significant differences between the two groups.

The detailed clinical and laboratory characteristics of both groups are presented in Table 1.

Univariate and multivariate logistic regression analyses were also performed to identify factors predictive of successful heart rate control. In the univariate analysis, renal failure (Odds Ratio: 0.169, 95% Confidence Interval [CI]: 0.038-0.754, p=0.020), GFR (OR: 1.016, 95% CI: 1.003-1.030, p=0.020), and hemoglobin (OR: 1.190, 95% CI: 1.040-1.362, p=0.011) were all identified as statistically significant predictors of achieving rate control.

However, when these variables were subjected to multivariate logistic regression analysis to adjust for potential confounding factors, only hemoglobin retained its statistical significance as an independent positive predictor of rate control (Adjusted OR: 1.154, 95% CI: 1.009-1.321, p=0.037).

Table 1

Clinical and laboratory characteristics of RCG (Rate Control Group) and N-RCG (Non-Rate Control Group) with significance levels between groups.

Characteristic	N-RCG (n=109)	RCG (n=58)	P value
Age, years (mean ± s.d.)	70.4 ± 11.6	67.5 ± 13.3	0.135
Sex, female, n (%)	65 (59.6)	32 (55.2)	0.578
Systolic BP, mmHg (mean ± s.d.)	125.8 ± 30.0	131.6 ± 20.4	0.516
Diastolic BP, mmHg (mean ± s.d.)	90.4 ± 23.2	87.0 ± 15.9	0.448
Heart Rate, beats/min (mean ± s.d.)	161.1 ± 17.5	154.2 ± 17.8	0.698
Heart Failure, n(%)	37 (33.9)	14 (24.1)	0.190
Renal Failure, n(%)	19 (17.4)	2 (3.4)	0.009
LVEF, % median (IQR)	50.0 (20.0)	50.0 (13.2)	0.490
Hemoglobin, g/dL (mean ± s.d.)	12.7 ± 2.5	13.6 ± 2.5	0.010
TnI, ng/ml median (IQR)	23.8 (35.7)	35.6 (37.6)	0.748
Creatinine, mg/dL median (IQR)	1.07 (0.65)	1.17 (0.53)	0.250
GFR, (ml/min/1.73m2) (mean ± s.d.)	58.7 ± 25.5	60.7 ± 27.3	0.015
Pro-BNP, pg/ml median (IQR)	6504.0 (8328)	4087 (10221)	0.173
Calcium, mg/dL (mean ± s.d.)	9.0 ± 0.6	9.1 ± 1.0	0.199
Potassium, mEq/L (mean ± s.d.)	4.4 ± 0.7	4.2 ± 0.6	0.524
Sodium, mEq/L (mean ± s.d.)	136.8 ± 4.8	137.2 ± 3.6	0.239
Outcome, n(%)			
Discharge	53 (50.5)	26 (45.6)	0.302
Hospital admission	14 (13.3)	13 (22.8)	
ICU admission	38 (36.2)	18 (31.6)	

Table 2

Univariate and multivariate logistic regression analyses identifying predictors of successful rate control.

Variable	Univariate OR (95% CI)	Univariate P value	Multivariate Adjusted OR (95% CI)	Multivariate P value
Renal Failure	0.169 (0.038-0.754)	0.020	0.243 (0.049-1.199)	0.082
Hemoglobin	1.190 (1.040-1.362)	0.011	1.154 (1.009-1.321)	0.037
GFR	1.016 (1.003-1.030)	0.020	1.008 (0.993-1.024)	0.307
LVEF	1.004 (0.973-1.035)	0.808		
Pro-BNP	1.000 (1.000-1.000)	0.510		

Renal failure (Adjusted OR: 0.243, 95% CI: 0.049-1.199, p=0.082) and GFR (Adjusted OR: 1.008, 95% CI: 0.993-1.024, p=0.307) did not remain statistically significant in the multivariate model.

The results of the univariate and multivariate logistic regression analyses are summarized in Table 2.

The distribution of outcomes (discharge, hospital admission, ICU admission) did not significantly differ between the RCG and N-RCG groups (p=0.302).

4. Discussion

Supraventricular tachycardia (SVT) represents a diverse group of rapid cardiac rhythms originating above the ventricles, frequently presenting with acute and distressing symptoms. If left uncontrolled, these arrhythmias can lead to significant cardiovascular compromise, including myocardial ischemia, heart failure, or even a decline in overall hemodynamic stability.[1] Effective pharmacological intervention is often necessary to achieve prompt rate and

rhythm control. Digoxin, a well-established cardiac glycoside, has a potential role in the management of SVT due to its therapeutic utility that originates from its ability to enhance vagal tone, slowing conduction through the atrioventricular (AV) node and consequently reducing the ventricular response rate. Digoxin's combined effects of enhancing myocardial contractility and suppressing atrioventricular nodal conduction render it as a viable option for controlling ventricular rate in various supraventricular tachyarrhythmias.[2] However, the effectiveness of digoxin in achieving rate control in patients with supraventricular tachycardia can be significantly influenced by various factors, such as organ function markers and hematologic parameters. Our primary results indicate that higher baseline hemoglobin levels were associated with successful rate control. This association remained statistically meaningful and independent in the multivariate logistic regression analysis (95% CI: 1.009-1.321, p=0.037).

Hemoglobin levels and Digoxin Efficacy

Our finding of higher hemoglobin levels predicting more successful rate control with digoxin aligns with existing physiological understanding that anemia can impact cardiac function and exacerbate arrhythmias. Anemia, particularly severe iron-deficiency anemia, has been directly associated with SVT and recognized as a reversible cause of supraventricular tachyarrhythmias 6. Lower hemoglobin concentrations reduce the blood's oxygen carrying capacity, which in turn increases heart rate and cardiac output to maintain adequate tissue perfusion. This physiological response creates a high-output circulatory state, which can make rate control more challenging. In such hyperdynamic states, digoxin is shown to be less effective in achieving optimal heart rate.[7] In a retrospective cohort study evaluating more than nine million patients taken from the records of Korean National Health Insurance Service database without a history of AF, aged ≥40 years, and with Hb levels available for two years apart checkups, low or high Hb levels are associated with an increased risk of incident AF.[8] Another study conducted by Sertcakacilar et al. with critically ill patients who underwent routine hemoglobin and electrocardiography monitoring in the ICU revealed a negative association between anemia and new onset atrial fibrillation, with incidence of atrial fibrillation being 9.9% in the total population, and 12.8% in the patient group with anemia.[9] Supraventricular tachycardias appear to both occur more frequently and be exacerbated in the presence of anemia, which may contribute to the reduced effectiveness of digoxin for rate control in these patients. Another potential factor is impaired bioavailability of digoxin, which could further compromise its ability to achieve effective rhythm control. In a study by Pérez et al., involving 11 children with heart failure and 2 with anemia, the authors observed significant interpatient variability in the elimination half-life, volume of distribution, and total body clearance of digoxin. These findings suggest that individual differences in the metabolism and excretion of digoxin are reflected in altered pharmacokinetics, potentially affecting therapeutic efficacy.[10] Findings of our study seems to support this notion, with the independent predictive value of hemoglobin contributing to digoxin's effectiveness and suggesting that the underlying physiological state influenced by hemoglobin modulates the pharmacodynamic response to digoxin, even after accounting for other relevant clinical factors. This implies that addressing and optimizing hemoglobin levels could be a beneficial strategy to enhance the success of digoxin-based SVT management.

However, reports also indicate an association between digoxin treatment and the development of anemia as an adverse reaction in patients with atrial fibrillation and heart failure. For patients with atrial fibrillation, digoxin treatment was found to be more likely to be accompanied with anemia (OR 1.38, 95% CI 1.14–1.68, P = 0.001). Proposed mechanisms for this drug-induced anemia involve digoxin's interference with iron transport processes due to interacting genes.[11] This introduces a complex dynamic: while higher baseline hemoglobin levels enhance digoxin's acute efficacy in SVT, prolonged digoxin use might itself exacerbate anemia. This feedback loop suggests that patients successfully rate-controlled with digoxin could develop or worsen anemia due to the drug, which may lead to a loss of rate control over time or other complications. This emphasizes a need for long term monitoring of hemoglobin levels in patients receiving digoxin therapy.

Renal Function and Digoxin Efficacy

Digoxin's pharmacokinetics are heavily influenced by renal function. The drug is predominantly eliminated by the kidneys, and its clearance is directly proportional to the glomerular filtration rate (GFR). In patients with renal impairment digoxin's half-life is significantly prolonged, extending from a typical 36-48 hours in healthy individuals to 3.5-5 days in those with renal failure.[12] Our results showed patients in the N-RCG exhibited a significantly higher prevalence of renal failure, with 19 out of 109 patients (17.4%) having renal failure compared to only 2 out of 58 patients (3.4%) in the RCG (p=0.009). The mean Glomerular Filtration Rate (GFR) was significantly lower in the N-RCG (58.7 ± 25.5 ml/min/1.73m2) than in the RCG (60.7 ± 27.3 ml/min/1.73m2), with a p-value of 0.015. These univariate findings initially suggested an inverse association between renal impairment and the success of rate control. However, when these renal parameters were subjected to multivariate logistic regression analysis their statistical significance was not maintained. This observed shift in statistical significance from univariate to multivariate analysis suggests that while renal dysfunctions influence on acute rate control success with digoxin may be confounded by other parameters. This implies that in a clinical setting where digoxin dosing is adjusted for renal impairment, the patient's overall physiological function might be a more critical determinant of acute rate control success.

During chronic kidney disease, accumulated uremic toxins have been shown to significantly impact the cardiovascular system, contributing to not only uremic cardiomyopathy and left ventricular hypertrophy, but also increasing the propensity for arrhythmias. Studies have shown that uremic serum samples can inhibit Na+/K+-ATPase activity and increase calcium cycling in cardiac myocytes, and these effects can be attenuated by digoxin-specific antibodies. In the setting of dysfunctional Na+/K+-ATPase compromised by the uremia, the therapeutic effect of digoxin might be blunted, and achieving adequate rate control in SVT/arrhythmia patients with renal failure might necessitate different therapeutic strategies.[13,14]

Patients with renal failure frequently also known to experience a range of electrolyte disturbances, and these imbalances can significantly modulate digoxin's effects. Specifically, hypokalemia, hypomagnesemia, and hypercalcemia are known to alter myocardial sensitivity to digoxin, directly affecting the drug's effectiveness. Another potential explanation for the association between glomerular filtration rate and the efficacy of digoxin in rate control is that renal dysfunction is frequently accompanied by autonomic dysregulation. Digoxin's mechanism of action in SVT management largely relies on increasing vagal tone, which is a component of parasympathetic activity, to slow AV nodal conduction. However, digoxin is known to be less effective in states of increased sympathetic drive caused by autonomic disfunction due to renal impairment.[15] This suggests that the physiological state itself can contribute to a form of digoxin resistance for rate control.

There are a number of studies evaluating digoxin's efficacy in patients with renal impairment and arrhythmias in literature. Eslami et al. conducted a study with 87 CKD patients with atrial fibrillation and heart failure, whom were administered digoxin and their di-

goxin levels evaluated after last loading dose. They found significant relationship between GFR and serum digoxin concentration (p-value = 0.038), and observed the lower the GFR was, the higher was the digoxin levels, with a similar relationship maintaining between serum creatinine and digoxin levels (p value: .04).[16] In another retrospective study of 92 critically ill patients with AF, a median intravenous digoxin loading dose of 11 mcg/kg (750 mcg) achieved a median serum digoxin concentration (SDC) of 1.3 ng/mL and resulted in a 60% rate control success, defined as a heart rate less than 110 bpm within 24 hours with no reported digoxin-associated dysrhythmias. The authors stated their findings support dose adjustment for impaired renal function (median LD of 8.3 mcg/kg for CrCl < 30 mL/min) and suggest that a higher serum digoxin concentration may be acceptable for acute rate control in this population.[17] Adversely, digoxin is known for its toxicity in the presence of renal impairment and its efficiancy in supraventricular tachichardia may be affected by this changes. A study of over 120,000 hemodialysis patients found that digoxin use significantly increased the risk of death by 28%. This mortality risk was even higher with increasing digoxin levels and was most pronounced in patients with low predialysis potassium concentrations.[18] These findings, along with our own, suggest that digoxin should be prescribed with great caution in this vulnerable population, given its potential for reduced efficacy and increased risk of adverse effects.

4.1. Limitations

This study is subject to inherent limitations of its retrospective, single-center observational design, which may carry a risk of selection bias and confounding by unmeasured variables that could influence the observed results. Our sample size of 167 patients may have been underpowered to detect more subtle effects or to achieve statistical significance for all relevant outcomes. Furthermore, focus of our study being exclusively on adult patients limits the generalizability of the findings to pediatric populations where digoxin is mostly used for SVT management. Lastly, digoxin serum levels were not available for analysis. The inclusion of these levels would have provided valuable information regarding the association between renal function, hemoglobin levels and digoxin dosing.

5. Conclusion

Our study demonstrates that higher baseline hemoglobin levels serve as an independent predictor of successful rate control with digoxin in adult patients presenting with supraventricular tachycardia. This finding underscores the importance of a patient's overall physiological state, particularly oxygen-carrying capacity, in influencing the effectiveness of antiarrhythmic therapy. While renal function is a critical determinant of digoxin pharmacokinetics, its independent impact on achieving acute rate control may be less pronounced in a clinical setting. Future prospective, multi-center studies with larger cohorts are warranted to further evaluate the precise pathophysiological mechanisms linking hemoglobin and renal function to digoxin efficacy in SVT.

Statement of ethics

The study received approval from the Istanbul Basaksehir Cam and Sakura City Hospital Ethics Committee on 2025-119

genAI

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Conflict of interest statement

The authors declare that they have no conflict of interest.

Availability of data and materials

This Data and materials are available to the researchers.

Author contributions

Both authors contributed equally to the article. Both authors read and approved the final manuscript.

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