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Initial 12 mg Versus 6 mg Adenosine for Supraventricular Tachycardia in the Emergency Department

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ABSTRACT

Objectives: This study aimed to compare the efficacy and safety of an initial 12 mg versus 6 mg adenosine dose for sinus rhythm conversion in patients presenting with supraventricular tachycardia (SVT) in the emergency department (ED).

Methods: This prospective observational study was conducted between February 2025 and January 2026. Adult patients (≥ 18 years) presenting to the ED with hemodynamically stable SVT confirmed by 12-lead electrocardiography (ECG) were included. Patients were categorized into two groups according to the initial adenosine dose administered (6 mg vs. 12 mg). To address potential selection bias, 1:1 propensity score matching (PSM) was performed. The primary outcome was successful first-dose sinus rhythm conversion. Secondary outcomes included adenosine-related adverse effects and SVT recurrence during the ED stay.

Results: A total of 142 patients were analyzed ($n = 71$ per group). First-dose conversion was significantly higher with 12 mg compared with 6 mg (83.1% vs. 52.1%). In the PSM cohort ($n = 104$), the 12 mg dose maintained its superior efficacy (82.7% vs. 53.8%). The PSM-adjusted odds ratio for successful conversion was 4.12 (95% confidence interval [CI]: 1.85–9.14), with a number needed to treat (NNT) of 3.8 (95% CI: 2.5–8.3). SVT recurrence was numerically lower in the 12 mg group (1.4% vs. 9.9%). Adverse effects were similar between groups.

Conclusions: An initial 12 mg adenosine dose was associated with higher first-dose sinus rhythm conversion than 6 mg, while adverse effects were similar between groups.

1 | Introduction

Supraventricular tachycardia (SVT) is among the most common tachyarrhythmias encountered in the emergency department (ED) and is typically characterized by a sudden onset and regular narrow-QRS complexes [1]. While vagal maneuvers are the recommended initial approach for hemodynamically stable patients, pharmacological therapy is required if these interventions fail to achieve rhythm conversion [2]. Current guidelines recommend intravenous adenosine as the first-line agent owing to its short half-life, rapid onset of action, and transient atrioventricular (AV) nodal conduction blockade [2, 3]. However, this stepwise approach may prolong ED treatment time and lead to repeated exposure to adenosine-related adverse effects, such as chest discomfort, dyspnea, flushing, and transient sinus arrest [2, 3].

Recent studies have suggested that the standard initial dose of 6 mg may be insufficient to achieve sinus rhythm conversion in some cases, while administration of a 12 mg dose of adenosine in patients refractory to the initial dose has been associated with higher cumulative success rates [4, 5]. However, evidence regarding 12 mg as an initial dose remains limited concerning success rates, SVT recurrence, and adverse effect incidence;

consequently, no clear consensus has been established [6, 7]. Therefore, this study aimed to compare the effectiveness of 6 mg and 12 mg initial doses of adenosine in achieving sinus rhythm and to evaluate the associated adverse effects and recurrence rates in patients presenting with SVT.

2 | Methods

2.1 | Study Design and Setting

This prospective observational study was conducted in the ED of a tertiary care hospital. Consecutive adult patients presenting with SVT between February 1, 2025, and January 31, 2026, were assessed for eligibility. Patients aged ≥18 years with SVT confirmed by 12-lead electrocardiography (ECG), consistent with atrioventricular nodal reentrant tachycardia (AVNRT) or atrioventricular reentrant tachycardia (AVRT), who were hemodynamically stable and received intravenous adenosine, were included in the study. The study protocol was approved by the Aksaray University Health Sciences Scientific Research Ethics Committee (approval no. 2025/13; January 16, 2025) and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants or their legal representatives.

Exclusion criteria comprised hemodynamic instability (defined as systolic blood pressure < 90 mmHg or the presence of clinical signs of instability, including altered mental status, signs of shock, ongoing chest pain suggestive of myocardial ischemia, or acute heart failure), the need for immediate electrical cardioversion, SVT termination following vagal maneuvers, wide-complex tachycardia (QRS > 120 ms), a prior history of radiofrequency catheter ablation, or refusal to provide written informed consent.

2.2 | Data Collection

Demographic characteristics, vital signs, comorbidities, history of SVT, caffeine consumption (defined as intake within 4 h prior to adenosine administration), administered adenosine dose, treatment response, and adverse effects were prospectively recorded using a standardized data collection form. All patients underwent a standard 12-lead ECG in the supine position at a paper speed of 25 mm/s, which was independently and blindly reviewed by two experienced ED physicians. Any discrepancies were resolved by consensus. SVT was diagnosed according to current international guidelines using 12-lead ECG [2, 3]. All patients underwent a standardized, modified Valsalva maneuver before adenosine administration [8].

Eligible patients received intravenous adenosine as a rapid bolus, immediately followed by a 20 mL normal saline flush. The initial adenosine dose (6 mg or 12 mg) was determined at the discretion of the treating physician based on clinical judgment. Adenosine was administered via a proximal intravenous line (preferably the right antecubital vein) using a T-connector or stopcock over 1–2 s. If sinus rhythm was not achieved after the initial dose, further management was performed according to current guidelines [2, 3]. All patients were continuously monitored with cardiac monitoring during their ED stay. Adverse events were prospectively recorded immediately after administration of the initial adenosine dose (6 mg or 12 mg). Clinicians used a standardized verbal inquiry to assess common adenosine-related symptoms, including chest tightness, flushing, shortness of breath, headache, nausea, and feeling of impending doom. Adverse event recording was limited to the effects of the first dose to minimize potential confounding from additional doses administered during the ED stay.

2.3 | Outcomes

The primary outcome was successful sinus rhythm conversion after the initial adenosine dose, confirmed by ECG documentation. Secondary outcomes included adenosine-related adverse effects and SVT recurrence during the ED stay. SVT recurrence was defined as ECG-documented SVT during the ED observation period after successful conversion.

2.4 | Statistical Analysis

Sample size was calculated a priori using G*Power software (version 3.1.9.7). Based on previous studies, expected SVT termination rates were estimated at approximately 55% for the 6 mg initial adenosine dose and 80% for the 12 mg dose [6, 9, 10]. For the comparison of two independent proportions with a two-sided alpha level of 0.05% and 80% power, a minimum of 66 patients per group (total $n = 132$) was required. To account for potential attrition or missing data, at least 70 patients per group were planned. To ensure independence of observations, only the first ED visit per patient was included in the analysis, and repeat presentations were excluded. All statistical analyses were performed using IBM SPSS Statistics (version 22.0; IBM Corp., Armonk, NY, USA). Normality was assessed using the Shapiro–Wilk test and visual inspection of histograms. Normally distributed variables were expressed as mean ± standard deviation (SD) and compared using the independent samples *t*-test. Non-normally distributed variables were presented as median (25th–75th percentile) and compared using the Mann–Whitney U test. Categorical variables were expressed as counts and percentages and compared using the Pearson chi-squared test or Fisher's exact test, as appropriate.

To address potential selection bias related to physician discretion in dose selection, a propensity score matching (PSM) analysis was conducted. Propensity scores were estimated using a logistic regression model including age, sex, and history of SVT as covariates. These variables were selected based on clinical relevance and baseline imbalance, as history of SVT was the only baseline characteristic significantly different between groups. Additional variables (e.g., caffeine consumption and body mass index) were not included in the model to avoid overfitting because they were already well balanced at baseline. PSM was performed in a 1:1 ratio using the nearest-neighbor method without replacement with a caliper width equal to 0.2 times the standard deviation of the logit of the propensity score [11, 12]. Patients outside the region of common support were excluded from the matched analysis. Covariate balance after matching was assessed using standardized mean differences (SMD), with

an absolute SMD <0.1 indicating adequate balance between groups. SMD was calculated as the difference in means or proportions divided by the pooled standard deviation. Covariate balance was assessed using SMD rather than significance testing, as SMD is independent of sample size.

Associations between clinical variables and successful first-dose conversion were evaluated using univariable and multivariable logistic regression analyses. A direct (enter) modeling strategy was used for the multivariable logistic regression analysis, in which all clinically relevant variables (initial adenosine dose, age, sex, and history of SVT) were included simultaneously without stepwise or automated variable selection procedures. This approach was preferred to avoid data-driven model selection and to improve the generalizability of the findings. Prior to multivariable modeling, diagnostic checks were performed to ensure the validity of logistic regression assumptions. Multicollinearity was evaluated using variance inflation factors (VIF), and no evidence of problematic multicollinearity was identified. The linearity-in-the-logit assumption for continuous variables was assessed using the Box–Tidwell approach. Influential observations were examined using Cook's distance, and no influential data points were identified. No evidence of complete or quasi-complete separation was observed. Overall model adequacy was evaluated by assessing the magnitude and precision of effect estimates and ensuring consistency between crude and propensity score–matched analyses. Model calibration was assessed using the Hosmer–Lemeshow goodness-of-fit test. Effect estimates were reported as odds ratios (ORs) with 95% confidence intervals (CIs). To enhance clinical interpretability of the treatment effect, additional measures of effect size were calculated for the primary outcome, including risk ratios (RR), absolute risk differences (RD), and the number needed to treat (NNT). All statistical tests were two-sided, and a p value <0.05 was considered statistically significant.

3 | Results

A total of 142 patients were included in the final analysis, with 71 patients in each initial adenosine dose group (6 mg and 12 mg) (Figure 1). The mean age of the study population was 52.7 ± 12.5 years, and 63 patients (44.4%) were male (Table 1). Before PSM, baseline characteristics such as age, sex, body mass index, vital signs, and comorbidities were generally comparable between groups. However, a history of SVT was more common in the 12 mg group than in the 6 mg group (70.4% vs. 53.5%; SMD = 0.35). Smoking status and caffeine consumption did not differ significantly between groups. The rate of successful conversion after the initial adenosine dose was higher in the 12 mg group than in the 6 mg group (83.1% vs. 52.1%), corresponding to an absolute difference of 31.0% (95% CI 16.2–45.8).

The matched cohort included 104 patients (52 per group), while 38 patients were excluded because they fell outside the region of common support or no suitable match could be identified within the predefined caliper width ($0.2 \times$ SD of the logit of the propensity score). After matching, all baseline covariates were well balanced, with SMD <0.1. In the matched cohort, the success rate was higher with the 12 mg initial dose than with the 6 mg dose (82.7% vs. 53.8%) (Table 2). SVT recurrence during the ED stay was lower in the 12 mg group than in the 6 mg group (1.4% vs. 9.9%); however, the difference did not reach statistical significance.

Crude and PSM estimates for factors associated with successful SVT conversion are presented in Figure 2. An initial 12 mg adenosine dose was associated with higher odds of successful conversion compared with 6 mg (crude OR 4.52, 95% CI 2.06–9.91; PSM OR 4.12, 95% CI 1.85–9.14). History of SVT, age, and sex were not associated with successful conversion in the PSM analysis. Additional effect size estimates, including risk ratio (RR), risk difference (RD), and NNT, were calculated. The crude RR was 1.59 (95% CI 1.24–2.05), whereas the PSM estimate was 1.42 (95% CI 1.15–1.74). The corresponding NNT values were 3.2 (95% CI 2.2–6.2) and 3.8 (95% CI 2.5–8.3) (Table 3).

Chest tightness was the most frequently reported adverse effect and occurred at similar rates in both groups (84.5% vs. 88.7%) (Table 4). Other adverse effects, including flushing, shortness of breath, headache, nausea, and a sense of impending doom, were also comparable between groups.

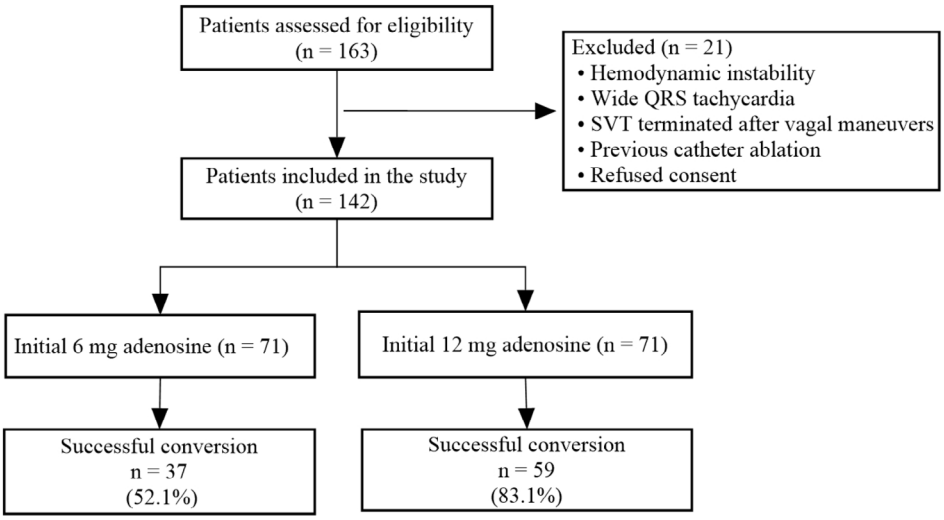

FIGURE 1 | Flow diagram of the study population.

TABLE 1 | Baseline characteristics according to initial adenosine dose before and after propensity score matching.

	Before matching ($n=142$)			After matching ($n=104$)[a]		
Variable	**6 mg ($n=71$)**	**12 mg ($n=71$)**	**SMD**	**6 mg ($n=52$)**	**12 mg ($n=52$)**	**SMD**
Age, years	51.1 ± 12.8	54.2 ± 12.1	0.25	52.8 ± 11.9	53.4 ± 11.5	0.05
Male sex	33 (46.5)	30 (42.3)	0.08	24 (46.2)	23 (44.2)	0.04
Body mass index, kg/m^2	28.3 ± 4.0	29.0 ± 3.8	0.18	28.7 ± 3.9	28.9 ± 3.7	0.05
Baseline systolic BP	123 (114–142)	124 (116–139)	0.07	124 (115–140)	124 (116–138)	0.02
Baseline diastolic BP	79 (70–85)	78 (72–90)	0.11	78 (71–86)	78 (72–88)	0.03
Heart rate at baseline	178.2 ± 12.6	177.1 ± 11.6	0.09	177.8 ± 12.2	177.3 ± 11.4	0.04
History of SVT	38 (53.5)	50 (70.4)	0.35	31 (59.6)	33 (63.5)	0.08
Hypertension	23 (32.4)	32 (45.1)	0.26	20 (38.5)	21 (40.4)	0.04
Diabetes mellitus	19 (26.8)	21 (29.6)	0.06	14 (26.9)	15 (28.8)	0.04
Atrial fibrillation	4 (5.6)	6 (8.5)	0.11	3 (5.8)	4 (7.7)	0.07
Asthma/COPD	5 (7.0)	7 (9.9)	0.10	4 (7.7)	5 (9.6)	0.06
Heart failure	2 (2.8)	4 (5.6)	0.14	2 (3.8)	3 (5.8)	0.09
Dyslipidemia	13 (18.3)	9 (12.7)	0.15	8 (15.4)	7 (13.5)	0.05
Beta-blocker/CCB use	19 (26.8)	23 (32.4)	0.12	15 (28.8)	16 (30.8)	0.04
Current smoker	15 (21.1)	24 (33.8)	0.28	13 (25.0)	15 (28.8)	0.08
Caffeine consumption	16 (22.5)	12 (16.9)	0.14	11 (21.2)	10 (19.2)	0.05

Note: Data are presented as mean ± SD, median (25th–75th percentile), or *n* (%). BP: blood pressure; SVT: supraventricular tachycardia; COPD: chronic obstructive pulmonary disease; CCB: calcium channel blocker.
[a]Propensity score matching was performed using 1:1 nearest-neighbor matching with a caliper width of 0.2 of the SD of the logit of the propensity score. SMD: standardized mean difference; values < 0.1 indicate adequate covariate balance. Balance for the matched cohort was assessed using SMD rather than significance testing (*p*-values), as SMD provides a measure of balance independent of sample size.

TABLE 2 | Clinical outcomes by initial adenosine dose in the primary and propensity score–matched analyses.

Outcome	Initial 6 mg	Initial 12 mg	Risk difference, % (95% CI)	*p*
Primary analysis ($n=142$)	$n=71$	$n=71$		
Successful conversion	37 (52.1)	59 (83.1)	31.0 (16.2 to 45.8)	< 0.001
Failure of initial dose	34 (47.9)	12 (16.9)	−31.0 (−45.8 to −16.2)	< 0.001
Matched analysis ($n=104$)	$n=52$	$n=52$		
Successful conversion	28 (53.8)	43 (82.7)	28.9 (13.5 to 44.3)	< 0.001
SVT recurrence during ED stay	7 (9.9)	1 (1.4)	−8.5 (−16.2 to 0.1)	0.063
Length of stay in ED, minutes	255 (210–310)	240 (200–290)	—	0.112

Note: Data are presented as *n* (%) or median (25th–75th percentile). ED: emergency department; SVT: supraventricular tachycardia; CI: confidence interval. Primary analysis includes the full cohort ($n=142$). Matched analysis represents the propensity score–matched cohort ($n=104$). Risk differences with 95% confidence intervals were used to estimate treatment effects. A *p* value < 0.05 was considered statistically significant.

4 | Discussion

An initial 12 mg adenosine dose may be clinically relevant in the ED setting, where rapid rhythm termination is desirable to relieve symptoms, shorten treatment time, and reduce the need for repeated drug administration, which may increase cumulative adverse effects, prolong patient discomfort and anxiety, and increase ED resource utilization. In this context, the 12 mg dose was associated with higher conversion rates than the conventional 6 mg dose, without an apparent increase in adenosine-related adverse effects. These findings may have implications for the traditional stepwise dosing strategy in hemodynamically stable patients with SVT.

Current literature indicates that adenosine is associated with high success rates in the acute treatment of SVT; however, these outcomes appear to be largely influenced by stepwise dose escalation strategies rather than the initial dose alone [2, 3]. In patients refractory to the standard initial 6 mg dose, escalation to higher doses has been shown to increase the

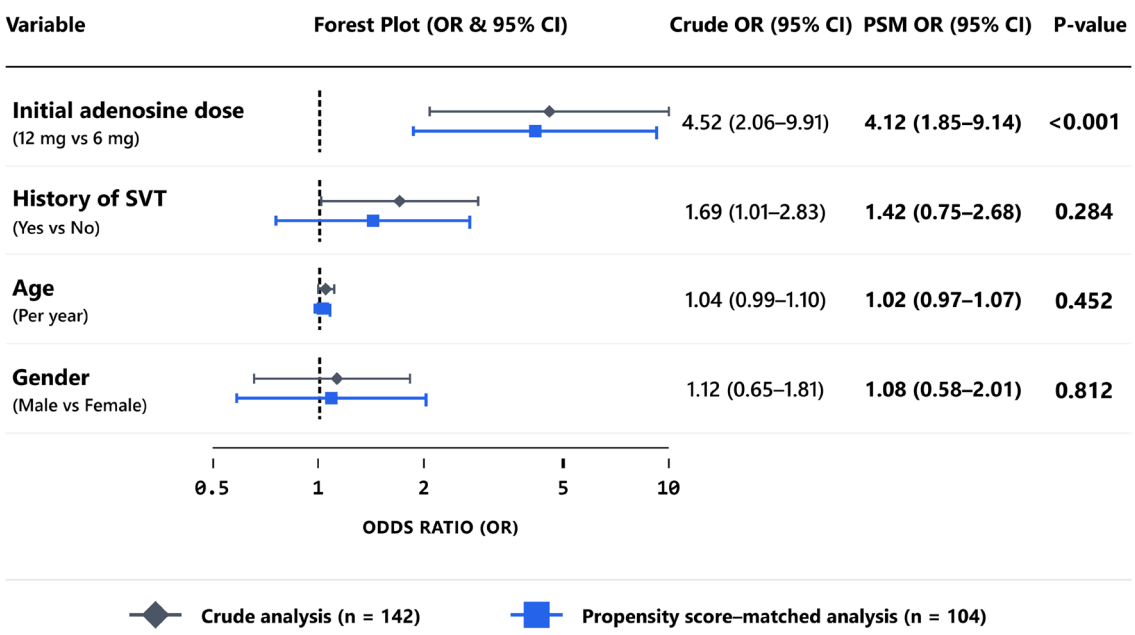

FIGURE 2 | Forest plot of crude and propensity score-matched odds ratios for successful SVT conversion.

TABLE 3 | Effect estimates for successful SVT conversion according to the initial adenosine dose (12 mg vs. 6 mg).

Measure	Crude estimates (95% CI)	PSM-adjusted estimates[a] (95% CI)
Odds Ratio (OR)	4.52 (2.06–9.91)	4.12 (1.85–9.14)
Risk Ratio (RR)	1.59 (1.24–2.05)	1.42 (1.15–1.74)
Risk Difference (RD)	31.0% (16.2–45.8)	26.4% (12.1–40.7)
Number Needed to Treat (NNT)	3.2 (2.2–6.2)	3.8 (2.5–8.3)

Abbreviations: CI: confidence interval; SVT: supraventricular tachycardia.
[a]PSM-adjusted estimates were derived from the propensity score–matched cohort ($n = 104$).

TABLE 4 | Adenosine-related adverse effects according to the initial adenosine dose (6 mg vs. 12 mg).

Adverse effect	Initial 6 mg ($n = 71$)	Initial 12 mg ($n = 71$)	p
Chest tightness	60 (84.5)	63 (88.7)	0.460
Flushing	22 (31.0)	27 (38.0)	0.377
Shortness of breath	16 (22.5)	17 (23.9)	0.843
Headache	6 (8.5)	4 (5.6)	0.512
Nausea	7 (9.9)	11 (15.5)	0.313
Feeling of impending doom	6 (8.5)	5 (7.0)	0.754
Transient bradycardia	0 (0.0)	1 (1.4)	0.500
Prolonged asystole > 3 s	1 (1.4)	1 (1.4)	NA
Atrioventricular block	0 (0.0)	0 (0.0)	NA

Note: Data are presented as *n* (%). Adverse effects represent dose-specific events occurring after the initial adenosine administration. *p* values were calculated using the chi-squared test or Fisher's exact test as appropriate.

likelihood of sinus rhythm conversion [4, 5, 10, 13, 14]. In a Cochrane meta-analysis of seven randomized controlled trials, Alabed et al. reported an overall success rate of 89.7% for adenosine in terminating SVT. This high efficacy was largely attributed to stepwise dosing protocols in which higher doses were administered when the initial 6 mg bolus failed [13]. Similarly, Xiao et al. [4] and Alghadeer et al. [5] reported that the initial 6 mg dose achieved sinus rhythm conversion in only 30% and 55% of patients, respectively, whereas cumulative success rates increased to 61% and 77% following escalation to 12 mg. Comparable findings were reported by Şirin et al., who observed a conversion rate of 48% with an initial 6 mg dose in vagal-refractory SVT, which increased to 67.5% after escalation to 12 mg in patients who did not respond to the first dose [14]. Collectively, these studies suggest that the standard initial 6 mg dose may be insufficient to achieve sinus rhythm conversion in a substantial proportion of patients, often requiring additional dosing. *Across these studies, approximately 45% to 70% of patients required dose escalation to 12 mg to achieve successful rhythm conversion*. Consistent with these observations, our findings suggest that a higher initial adenosine dose may

be associated with improved first-dose conversion in patients presenting with SVT in the ED.

Recent studies have also supported the potential efficacy of a 12 mg initial adenosine dose in the treatment of SVT. In a retrospective analysis, Krug et al. [6] reported that an initial 12 mg dose was associated with a significantly higher first-dose conversion rate compared with 6 mg (79.1% vs. 56.4%). However, the retrospective design of the study, the lack of assessment of SVT recurrence, and the absence of evaluation of potential confounding factors such as administration technique and caffeine consumption represent important methodological limitations that may affect the interpretation of these findings. Moreover, adenosine-related adverse effects were not comprehensively assessed. Similarly, in a large-scale prehospital study, Fernandez et al. [7] reported that an initial 12 mg adenosine dose was associated with a reduced need for repeat dosing, higher rates of clinical improvement, and lower hospital admission rates compared with 6 mg. Nevertheless, SVT outcomes were based on paramedic-reported clinical improvement rather than ECG-confirmed restoration of sinus rhythm. In addition, ED recurrence rates and detailed adverse effect profiles were not evaluated. Consequently, the impact of prehospital adenosine administration on the subsequent ED clinical course and short-term rhythm stability could not be fully determined. In contrast to prior retrospective and prehospital studies [6, 7], our prospective ED-based study provides further evidence regarding the potential efficacy and safety of a 12 mg initial adenosine dose. By addressing key methodological limitations of earlier studies, our findings suggest that this dosing strategy may be considered a potential alternative initial treatment strategy, although confirmation in randomized studies is required.

In our study, the robustness of the observed treatment effect was supported by both PSM and regression analyses. After balancing baseline characteristics through PSM, the higher conversion rate associated with the 12 mg dose remained consistent. The PSM analysis demonstrated higher odds of successful conversion with the 12 mg dose compared with 6 mg. Additional effect size measures suggested a clinically meaningful benefit, with an estimated NNT of approximately four patients, indicating that one additional successful conversion may be achieved for every four patients treated with an initial 12 mg bolus.

5 | Limitations

This study has several limitations that should be acknowledged. First, although the data were collected prospectively, the study was observational in design and did not involve randomization. The choice of the initial adenosine dose was left to the treating physician's discretion rather than being determined by a predefined dosing protocol, which may have introduced selection bias and residual confounding. While statistical adjustment methods were applied, the possibility of unmeasured confounding cannot be completely excluded. Specifically, although caffeine use and BMI were well balanced between groups after matching, residual confounding from factors such as quantified caffeine intake or absolute body weight—both of which may influence adenosine efficacy—may still be present, as BMI may not fully capture weight-related physiological variability. Additionally, although the study protocol required the use of a proximal intravenous line (preferably the right antecubital vein), the exact venous access site was not systematically recorded for each patient, which may have influenced the efficacy of adenosine administration. Second, this was a single-center study conducted in a tertiary-care ED, which may limit the generalizability of the findings to other clinical settings. Third, the assessment of adenosine-related adverse effects relied partly on patient-reported symptoms and clinical observation during the ED stay; therefore, mild or transient symptoms may have been underreported. Nevertheless, continuous cardiac monitoring allowed objective detection of major arrhythmic events and conduction disturbances. Fourth, invasive electrophysiological studies were not performed; therefore, definitive classification of SVT subtypes and detailed evaluation of the electrophysiological mechanisms underlying the response to adenosine were not possible. Consequently, without electrophysiological confirmation, some cases may have represented other adenosine-responsive tachyarrhythmias (e.g., atrial tachycardia) or spontaneously terminating narrow-complex tachyarrhythmias. Finally, SVT recurrence was assessed only during the ED observation period, and longer-term recurrence after discharge was not evaluated. Although the observed effect size was clinically meaningful, the relatively wide confidence interval for the primary outcome indicates some uncertainty in the precision of the estimate and warrants cautious interpretation. Despite these limitations, the prospective design and ED setting offer clinically relevant insights into initial adenosine dosing strategies for SVT management.

6 | Conclusion

An initial 12 mg adenosine dose was associated with higher first-dose sinus rhythm conversion rates than a 6 mg dose in hemodynamically stable patients with SVT. This association persisted after adjustment for baseline characteristics. The incidence of adenosine-related adverse effects was similar between the two dosing strategies. These findings suggest that initiating treatment with a 12 mg adenosine dose may represent a potentially effective alternative approach for the acute management of SVT in the ED. However, given the observational design of the study, these results should be interpreted as an association rather than evidence of causality.

Author Contributions

Ekrem Taha Sert: writing – original draft, writing – review and editing, conceptualization, methodology, investigation, formal analysis, data curation. **Kamil Kokulu:** conceptualization, methodology, investigation, data curation, writing – review and editing. **Oğuz Yürük:** methodology, investigation, writing – review and editing. **Emin Hüseyin Akar:** investigation, data curation, writing – review and editing. **Muhammed Ali Topuz:** investigation, data curation, writing – review and editing.

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The authors have nothing to report.

Ethics Statement

The study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki and was approved by the Aksaray University Health Sciences Scientific Research Ethics Committee (approval number: 2025/13).

Consent

All authors have read and approved the final version of the manuscript and consent to its publication.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request. The data are not publicly available due to privacy and ethical restrictions.

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