Use of Adenosine in the Treatment of Supraventricular Tachycardia in a Pediatric Emergency Department

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Objective: Supraventricular tachycardia (SVT) is the most frequent arrhythmia requiring treatment in childhood, with an estimated incidence of 1/100 to 1/250 children. The treatment of choice of the acute event is intravenous adenosine. This study aimed to determine if doses of adenosine higher than previously described are needed to successfully revert SVT in children.

Methods: This is a retrospective study of SVT cases in a tertiary hospital from January 2007 to December 2011.

Results: A total of 44 episodes of SVT were recorded in 26 patients. Mean age was 3.1 years. In 39 patients (89%), adenosine was administered, reverting to stable sinus rhythm in 29 episodes, which represents an effectiveness of 75%. In relation to the number of doses administered, 12 patients (30%) received a single dose, with a mean (SD) response dose of 112 (35) μg/kg; 16 (41%) received 2 doses, with a mean (SD) response dose of 188 (55) μg/kg; and 9 (24%) received 3 doses, with a mean (SD) response dose of 249 (108) μg/kg. Finally, in 2 patients (4%), 4 doses of adenosine were administered, with only 1 of them responding to a dose of 300 μg/kg. The mean (SD) dose that reverted the SVT to normal sinus rhythm was 173 (84) μg/kg, and the mean (SD) number of doses administered was 1.7 (0.8) (range, 1–4). Sixty-six percent were discharged home, without the need to be transferred to pediatric intensive care unit or pediatric ward.

Conclusions: Most of the patients with SVT episodes require treatment with more than 1 dose of adenosine. Doses higher than the usually described in the guidelines are necessary to revert SVT. Most patients can be discharged home from the emergency department, without the need of hospital admission.

Key Words: supraventricular tachycardia, adenosine, doses

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Supraventricular tachycardia (SVT) is a fast rhythm that originates above the bundle of His, with or without participation of the auriculoventricular (AV) node and may on occasions be caused by the abnormal automatism of a particular group of cells.[1] Reentrant SVT is the most frequent form of tachycardia in childhood. It is most commonly the result of electric conduction through accessory pathways between the atria and the ventricles in infants and children. In other cases, it is secondary to intranodal reentry at the AV node, which is more common in adolescents and adults.[2,3] Most of the patients with SVT episodes do not have any structural heart disease, although there are some cases related to congenital heart disease such as Ebstein anomaly.[4,5]

Supraventricular tachycardia is the most frequently encountered arrhythmia requiring treatment in childhood, with an estimated incidence of 1/1000 to 1/250 children.[6–8] The drug of choice for the short-term episode is intravenous adenosine for its short half-life, approximately 5 seconds, and its great effectiveness.[9–11] Adenosine has a negative dromotropic effect at the level of the AV node when rapidly administered through an intravenous line. This slowing and, even at times, interruption of the conduction of the electric impulse at the AV node can restore the normal sinus rhythm in patients with reentrant SVT. Its use can also be a valuable diagnostic tool because the slowing down of the AV conduction at the AV node can unmask F waves of an atrial flutter.

METHODS

This retrospective study includes all the patients with SVT diagnosed in the pediatric emergency department (ED) of a tertiary hospital between January 2007 and December 2011. This ED assists a mean of 106,000 emergencies per year.

All the patients younger than 14 years who presented to the ED with an episode of SVT, requiring hospital admission or not, were included. Exclusion criteria include diagnosis at another center where SVT had reverted to sinus rhythm on arrival to our ED. The diagnosis of SVT was confirmed in all patients with a 12-lead electrocardiogram. As a first step, vagal maneuvers were performed, such as the application of a cold bag in infants or Valsalva maneuvers in collaborative children, while an intravenous access of the greatest possible caliber was obtained, preferably in an upper extremity. All children presenting with SVT received adenosine in rapid boluses, according to Pediatric Advanced Life Support guidelines, using incremental doses of 100, 200, and 300 μg/kg. The initial dose of adenosine was 100 μg/kg, administered through a 3-way stopcock for a rapid flushing of the line with saline after the injection of adenosine. A positive response to adenosine was considered the reversion to stable sinus rhythm.

The clinical files from the ED were revised, obtaining clinical and epidemiological data such as age, sex, underlying structural heart disease, previous episodes, presentation, associated congestive heart failure (CHF), spontaneous resolution of the episode, vagal maneuvers used, drugs administered, and destination on discharge from the ED (home, pediatric ward, pediatric intensive care unit [PICU]). The ways of presentation were described as asymptomatic, unspecific symptoms, tachycardia detected by the parents, chest pain, palpitations, or presence of CHF. The files in relation to admission to the ward or PICU and the discharge reports were used to obtain data about in-hospital stay, progress, and treatment.

Statistical analysis was performed using the SPSS statistical package version 15.0. A database was created in SPSS for

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the variables. A descriptive analysis of the variables with punctual estimation and 95% confidence interval was performed, treating the continuous variables as means, SDs, or medians, depending on the distribution of the variables. The categorical variables were presented in frequencies and percentages. A statistical analysis using χ^2 test was performed to assess the association between age and presentation.

RESULTS

Between January 2007 and December 2011, 44 episodes of SVT were treated at the ED of our institution (26% male and 74% female) in a total of 26 patients, representing a prevalence of 0.08 × 1000 of the emergencies during the study period. The median age was 3.1 years, with a range of 1 month to 13.5 years. Only 2 patients showed associated structural heart disease, namely, an unbalanced AV canal and a moderate mitral insufficiency. The mean (SD) heart rate during the episodes of tachycardia was 229 (38) beats per minute, with a range between 165 and 300.

In 22% of the cases, it was the first episode of SVT, and in the majority (78%), it was a second or further episode. Sixteen percent of the episodes were in infants younger than 1 year, although if we only account for first episodes, infants represented 40% of the debuts.

In relation to the way of presentation, there was a marked difference when dividing the sample into those older and younger than 3 years. In children younger than 3 years, 8 patients presented with tachycardia that was detected by the parents; unspecific symptoms were stated in 7 cases; and 3 were asymptomatic cases. On arrival at the ED, 2 patients showed signs of CHF. In the group of patients older than 3 years, the most frequent reasons for consultation were palpitations in 12 patients and chest pain or dyspnea in 5 of them. There were significant statistical differences ($P < 0.05$) for unspecific symptoms in patients younger than 3 years and for tachycardia stated by the patient in the age group of patients older than 3 years.

Of the 44 episodes, 5 patients (11%) did not receive adenosine because 3 of them ended spontaneously, 1 reverted with vagal maneuvers, and 1 with a diagnosis of Coumel tachycardia ended after the administration of propranolol. In 39 patients (89%), adenosine was administered, reverting to stable sinus rhythm in 29 of these episodes, representing an effectiveness of 75%. Ten patients (25%) did not respond to the administration of adenosine: 4 patients received β-blockers, 3 received amiodarone, 2 had to be cardioverted, and 1 received amiodarone plus cardioversion (Fig. 1). One patient died (case number 2), a 27-day-old newborn who presented with a junctional ectopic tachycardia with severe biventricular dysfunction on admission that responded to neither adenosine nor amiodarone. Table 1 shows the characteristics of the SVT episodes, the response to treatment, and the follow-up on discharge.

In relation to the number of doses of adenosine administered, 12 patients (30%) received a single dose, with a mean (SD) response dose of 112 (35) μg/kg; 16 (41%) received 2 doses, with a mean (SD) response dose of 118 (55) μg/kg; and 9 (24%) received 3 doses, with a mean (SD) response dose of 249 (108) μg/kg. Finally, in 2 of the patients, 4 doses of adenosine were administered, with only 1 of them responding to a dose of 300 μg/kg (episode 11). The mean (SD) dose that reverted the SVT to sinus rhythm was 173 (84) μg/kg, and the mean (SD) number of doses administered was 1.7 (0.8) (range, 1–4).

In 2 episodes that did not respond to adenosine in the ED, a flutter and a reentry (episodes 30 and 32), cardioversion restored in both cases the sinus rhythm. In 3 episodes (episodes 19, 31, and 44) with well-tolerated SVTs that had previously presented with tachycardia and were receiving prophylactic treatment with propranolol at the onset of symptoms, an extra dose of β-blocker was administered, reverting to sinus rhythm.

In relation to where treatment was performed, 33 patients (75%) received treatment exclusively in the ED, 7 (16%) were admitted to the PICU after the initial treatment in the ED, and 4 (9%) presenting with CHF were admitted directly to the PICU without starting any treatment in the ED.

Of the 40 patients treated initially in the ED, 29 (66%) were discharged home, 7 were later admitted to the PICU, and another 4 were admitted directly to the PICU, totaling 11 admissions to the PICU (25% of the episodes). Four patients (9%) were admitted to a pediatric ward (Fig. 2). The mean length of stay in the ED was of 13.5 hours.

The main criteria for admission to critical care (11/44 patients, 25% of the episodes) were nonresponse to adenosine in

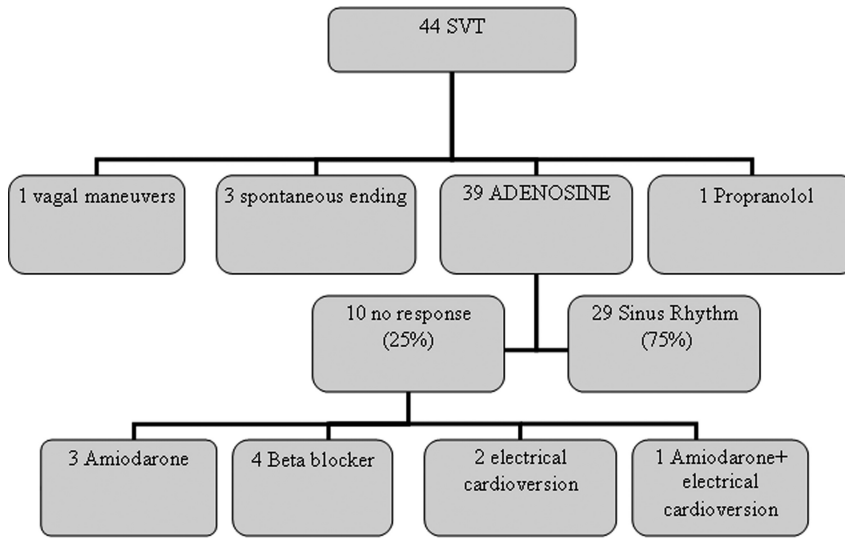

FIGURE 1. Treatments administered and response to the administration of adenosine.

6 patients, the presence of heart failure and hemodynamic instability in 4 patients, and the need for monitoring in a 1-month-old patient (episode 3). Additional doses of adenosine were required in the PICU in 7 episodes, being effective in 4. Patients who did not respond to adenosine received amiodarone (3 patients), esmolol (2 patients), and cardioversion plus amiodarone in episode 33, which corresponded to an atrial flutter.

The diagnoses of the 44 episodes, obtained from the medical records after follow-up, were 31 accessory pathway reentries, 6 Wolff-Parkinson-White syndromes, 3 permanent reciprocating tachycardia of the union (Coumel tachycardia), 2 atrial flutter, an intranodal reentry, and 1 junctional ectopic tachycardia.

DISCUSSION

The most frequent arrhythmia with clinical repercussion in childhood is SVT. The prevalence observed in our series with respect to the ED emergencies attended is 0.08×1000, the real prevalence being probably higher because many episodes go unnoticed, end spontaneously, or do not require hospital care.

Although most of SVTs occur in structurally normal hearts, sometimes, it is associated with congenital heart defects. In our study, 2 of the 26 patients (8%) had structural heart disease, although none had Ebstein anomaly.

Supraventricular tachycardia is more common in children younger than 1 year, especially younger than 4 months. In our review, children younger than 1 year represent 40% if we consider only the onset of the first episode, a slightly lower figure than found in the literature, where up to 50% of the children with SVT are described as presenting with their first episode within the first year of life. When tachycardia appears beyond the first year of age, prognosis is less favorable, with spontaneous remission rates of 15% to 20% at 12 years.[12]

It is well-known that the way episodes of SVT present varies according to age.[13] Newborns and children younger than 1 year present with nonspecific symptoms such as cyanosis, pallor, irritability, trouble with their feedings, tachypnea, or sweating or sometimes may be detected on a routine check. On the contrary, older children such as adolescents may complain of palpitations, shortness of breath, chest pain, dizziness, or syncope. In our study, this difference shows statistical difference, in the group of patients younger than 3 years the most frequent complaint being "the tachycardia evidenced by parents." One reason for this could be that many episodes corresponded to recurrences, so the parents were sensitized and trained in the detection of episodes of tachycardia.

It is important to underline that the nonspecific symptoms of younger children involves a greater delay in diagnosis, the reason why patients in this age group may present with a higher incidence of CHF and cardiovascular collapse. This was the form of presentation in 2/7 children younger than 12 months. In patients older than 3 years, the most frequent complaint at presentation was the symptoms referred by the patient, palpitations being the most frequent symptom.

When suspecting an episode of SVT, the first study to be performed is a 12-lead electrocardiogram. Although most of the SVT episodes occur in structurally normal hearts, after an episode of SVT, children should be evaluated as outpatients by a pediatric cardiologist. This is especially important in cases with wide QRS, when there is poor tolerance of the episode, no response to treatment, or when preexcitation or other abnormalities are found in the basal electrocardiogram.[14]

For the short-term management of the episode, the antiarrhythmic drug of choice in stable children is intravenous adenosine, once there is no response to vagal maneuvers. In our series, only 1 patient responded to these maneuvers when proceeding to the canalization of a venous access. It is important to bear in mind that adenosine has to be administered through a vein of good caliber, preferably in the upper limbs, as close as possible to the heart. The infusion rate will be the fastest possible, and the subsequent flushing with physiological saline must be performed to accelerate the arrival of the drug in the coronary circulation. In the unstable patient, the treatment of choice is synchronized electrical cardioversion with an initial dose of 0.5 to 1 J/kg. Even so, if we have a venous access, we can try an initial dose of adenosine because it is useful to establish the mechanism of the tachycardia, especially in cases of atrial flutter.

The administration of adenosine in childhood is effective in the cessation of the SVT in 70% to 85% of cases.[15–17] In our series, it was administered in 89% of episodes, being effective in 75% of the cases. It should be noted that none of the patients who were directly admitted to the PICU with signs of severe heart failure responded to adenosine. Two cases, 1 with Coumel tachycardia and a flutter, were arrhythmias in which adenosine did not restore sinus rhythm. In the other 2 cases, the lack of response could be in relation to the presence of severe heart failure and low output, with the consequent slowing of the venous circulation.

The initial dose of adenosine in SVT is a highly controversial issue. Pediatric Advanced Life Support guidelines recommend a starting dose of 100 μg/kg (maximum of 6 mg), increasing to 200 μg/kg (maximum of 12 mg).[18] In our series, the average of the first dose administered that reverted to sinus rhythm was 120 μg/kg, 188 μg/kg in those who received 2 doses, 249 μg/kg in those who received 3 doses, and 300 μg/kg in the only patient who responded after a fourth dose. Only 24% of the patients, who responded to adenosine, responded after a single dose (120 μg/kg). These results suggest that in our setting, the initial dose recommended by the guidelines for the administration of adenosine is insufficient. The overall average cumulative dose that patients received was 275 μg/kg, with a range from 100 to 900 μg/kg, without causing undesirable adverse effects. Because adenosine is a drug with few adverse effects owing to its very short half-life, it seems advisable to recommend a higher initial dose, at least 200 μg/kg. Dixon et al[19] performed a retrospective study collecting 53 episodes of SVT and proposed a minimum initial dose of 100 μg/kg in children and 150 or 200 μg/kg in infants. Possible causes that could justify the need of higher doses in infants include the following: greater body surface area, smaller venous access, and a poorer hemodynamic state on diagnosis. Other studies have also demonstrated the lack of effectiveness of lower doses of adenosine.[20,21] Losek et al[15] found that a higher total dose was needed when the first administered dose was low. This could also have economic implications because it probably implies a higher pharmaceutical cost.

It is important to stress the importance of an appropriate initial management in the ED. Our study shows that SVT in children can be treated in the pediatric ED and that two thirds of these patients can be discharged without the need for hospital admission. The admission to PICU of patients who have an episode of SVT depends primarily on 3 factors as follows: the clinical condition of the child, the absence of response to treatment in the ED, and the presence of known heart disease. The main reason of admission in our series was the absence of response to adenosine and, in 2 cases, the presence of heart failure or signs of cardiovascular collapse. Despite this, most of the episodes of SVT evolved well, with resolution of the episodes and recovery of systolic function. Only

TABLE 1. Characteristics of the Episodes and Response to Treatment in the ED

	Age, mo	Response to Adenosine ED	Response to Adenosine PICU	Response Dose, μg/kg	Total Dose, μg/kg	No. Doses	Other Treatments	Type of SVT	Evolution Destination after ED
1	0, 9	—	No		600	3	Amiodarone	WPW	Direct to PICU
2	1	No	No		600	3	Amiodarone	JET	PICU/ exitus
3	1	Si		150	250	2		Occult accessory pathway reentry	PICU
4	2	No	Yes	300	600	2		Occult accessory pathway reentry	PICU
5	5	Yes		200	300	2		Occult accessory pathway reentry	Home
6	10	Yes		200	300	2		Occult accessory pathway reentry	Home
7	10	Yes		100	100	1		Occult accessory pathway reentry	Ward
8	16	Spontaneous ending						Occult accessory pathway reentry	Ward
9	17	Yes		200	500	3		Occult accessory pathway reentry	Home
10	18	No	Yes	200	600	2		Occult accessory pathway reentry	PICU
11	23	Yes		300	900	4		WPW	Home
12	23	Yes		150	250	2		Occult accessory pathway reentry	Home
13	28	Yes		200	200	1		WPW	Home
14	28	Yes		400	900	3		Occult accessory pathway reentry	Home
15	30	Yes		150	250	2		Occult accessory pathway reentry	Home
16	33	No	No		700	4		WPW	PICU
17	33	Yes		200	300	2		Occult accessory pathway reentry	Home
18	34	—			300	2	Esmolol	Coumel	PICU direct
19	35	No			200	2	Propranolol	Coumel	Home
20	36	Yes		200	300	2		Occult accessory pathway reentry	Home
21	36	Yes		100	100	1		Coumel	Ward
22	37	Yes		200	400	2		Occult accessory pathway reentry	Home
23	38	Yes		150	150	1		Occult accessory pathway reentry	Home
24	38	Spontaneous ending						Occult accessory pathway reentry	Home
25	39	Yes		200	300	2		Occult accessory pathway reentry	Home
26	51	Ceased with vagal maneuvers						Occult accessory pathway reentry	Home
27	56	No	Yes	300	500	2		Occult accessory pathway reentry	PICU
28	75	Yes		200	450	3		Occult accessory pathway reentry	Home
29	87	Yes		100	100	1		Occult accessory pathway reentry	Home
30	91	No			100	1	Cardioversion	Flutter	Home
31	96	No			200	2	Propranolol	Occult accessory pathway reentry	Planta
32	106	No			300	1	Cardioversion	Occult accessory pathway reentry	Home

(Continued on next page)

TABLE 1. *(Continued)*

	Age, mo	Response to Adenosine ED	Response to Adenosine PICU	Response Dose, μg/kg	Total Dose, μg/kg	No. Doses	Other Treatments	Type of SVT	Evolution Destination after ED
33	108	—	No		100	1	Amiodarone cardioversion	Flutter	PICU direct
34	108	Yes		100	100	1		Occult accessory pathway reentry	Home
35	113	No	Yes	400	700	3		Occult accessory pathway reentry	PICU
36	120	Spontaneous ending						Occult accessory pathway reentry	Home
37	156	Yes		150	350	3		Intranodal pathway	Home
38	156	Yes		100	100	1		Occult accessory pathway reentry	Home
39	156	Yes		250	550	3		Occult accessory pathway reentry	Home
40	156	Yes		100	100	1		Occult accessory pathway reentry	Home
41	158	—	No		450	3	Amiodarone	Occult accessory pathway reentry	PICU direct
42	163	Yes		100	100	1		Occult accessory pathway reentry	Home
43	163	Yes		100	200	2		WPW	Home
44	163	—					Propranolol	WPW	Home

JET indicates junctional ectopic tachycardia; WPW, Wolff-Parkinson-White.

1 patient in our series died: a neonate with junctional ectopic tachycardia, in whom the arrhythmia could not be controlled despite electrical cardioversion and amiodarone treatment.

Our work, being retrospective, presents the limitations inherent to the collection of data. Further studies would be needed to determine the most effective dose, taking also into account

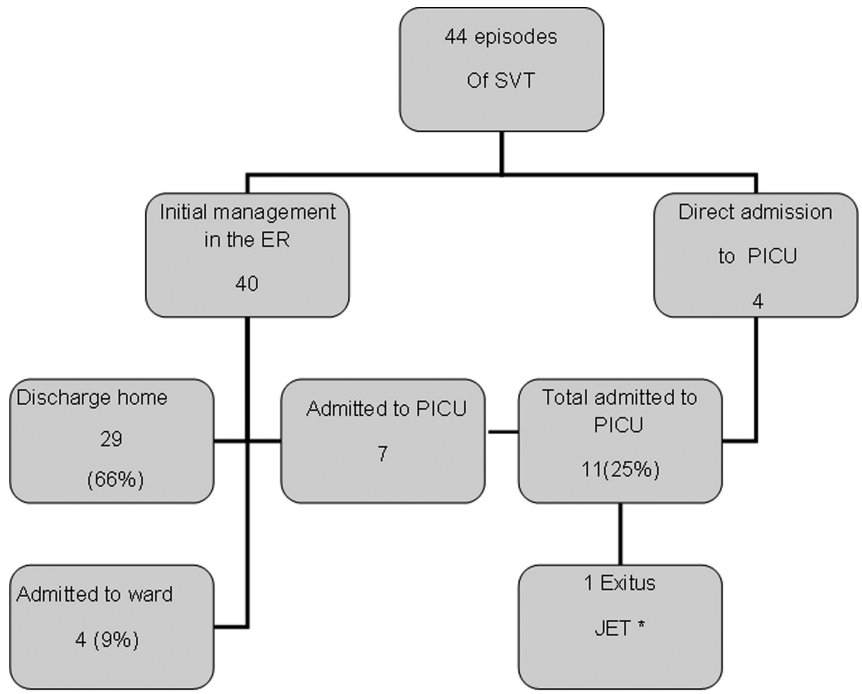

FIGURE 2. Follow-up of SVT episodes. JET indicates junctional ectopic tachycardia.

the type of venous access, peripheral or central; the speed of administration; the flushing of the line; age; and cardiovascular status, among other factors.

In conclusion, the forms of presentation of SVT vary and depend on age. Most patients require treatment with adenosine, needing more than 1 dose for the termination of the arrhythmia. Doses higher than those described in the guidelines are needed to subjugate the episode of SVT and restore normal sinus rhythm. We finally point out that SVT episodes in the pediatric age group can be effectively treated in the emergency area, without the need for hospital admission.

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