Acute Management of Refractory and Unstable Pediatric Supraventricular Tachycardia

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Objective To characterize the management of acute pediatric supraventricular tachycardia (SVT), placing special emphasis on infants, patients refractory to adenosine (refractory SVT), and patients with hypotension, poor perfusion, or altered mental status (unstable SVT).

Study design Retrospective cohort study of patients 0-18 years of age without congenital heart disease who presented to our pediatric hospital from January 2003 to December 2012 for the treatment of acute SVT. Multiple logistic regression was applied to identify whether age was a risk factor for different SVT therapies. Model fit and residuals also were examined.

Results We identified 179 episodes for SVT. First dose of adenosine was effective in 72 (56%) episodes, and a second dose was effective in 27 of 54 (50%) episodes, leaving 27 (15%) episodes with refractory SVT. The response to the first dose of adenosine increased proportionally with age (OR 1.13, 95% CI 1.05-1.2). Only 1 of 17 episodes in infants responded to the first dose of adenosine. Refractory SVT was more frequent in infants vs older children ($\chi^2 = 5.9$ [1 df], $P = .01$). Unstable SVT was present in 13 episodes and was treated with adenosine and antiarrhythmics. Synchronized cardioversion was performed on 3 patients, 2 patients with unstable SVT, and 1 with refractory SVT.

Conclusion In children with SVT, young age is associated with decreased response to the first dose of adenosine and increased odds of adenosine-refractory SVT. In the treatment of unstable SVT, medical management with various antiarrhythmics before cardioversion may have a role in a subset of patients. Synchronized cardioversion rarely is performed for acute SVT. *(J Pediatr 2017;181:177-82).*

Supraventricular tachycardia (SVT) is the most common pediatric tachyarrhythmia in children. The use of vagal maneuvers and adenosine for the acute management of SVT is the current standard of care.[1,2] Adenosine is an endogenous nucleoside ubiquitous in humans. Its mechanism of action for decreasing the heart rate in SVT is through the binding of adenosine receptors in the sinoatrial, atrioventricular nodes, and atrial myocytes.[3] Since its first use in children in 1987,[4] adenosine has been established as a safe and effective medication for acute SVT, with quick onset and a short half-life of less than 10 seconds.

Current guidelines developed by the International Liaison Committee on Resuscitation and the American Heart Association (AHA) define adenosine dosing and administration for the first 2 doses as 0.1 and 0.2 mg/kg, respectively, or 6 and 12 mg, respectively, for older children. In cases of SVT with cardiovascular instability (hypotension, impaired perfusion) without altered mental status, it is recommended that adenosine be used only if it does not delay cardioversion, whereas immediate cardioversion is recommended for patients with SVT and altered mental status.[5] The International Liaison Committee on Resuscitation and the AHA recently identified important knowledge gaps and areas needing future investigation for the treatment of pediatric SVT related to the efficacy of various vagal maneuvers, choice of antiarrhythmic medication in patients refractory to the first 2 doses of adenosine (refractory SVT), and use of electrical therapy in patients with cardiovascular instability and altered mental status. In addition to these identified areas of uncertainty, recent reports suggest that infants might be less responsive to adenosine.[6-8]

We sought to perform an analysis of cases of pediatric SVT in patients without a history of congenital heart disease treated at our tertiary care facility, with a particular focus on infants, patients refractory to first 2 doses of adenosine, and patients presenting with hypotension, poor perfusion, or altered mental status (unstable SVT). Our primary hypothesis was that infants have lower response rates to

AHA	American Heart Association
ED	Emergency department
ICD-9	*International Classification of Diseases, Ninth Revision*
ICU	Intensive care unit
SVT	Supraventricular tachycardia

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adenosine and a greater prevalence of refractory SVT compared with children 1 year and older. Our secondary hypothesis was that patients with unstable SVT are treated rarely with synchronized cardioversion.

Methods

We included patients aged 0-18 years evaluated at our facility for acute SVT from January 1, 2003, through December 31, 2012. We excluded patients with a history of structural heart disease or previous cardiac surgery. To capture the preponderance of patients with acute SVT episodes in Western Pennsylvania, we reviewed the medical records of all pediatric patients who were (1) treated for SVT in the emergency department (ED), inpatient ward, or pediatric cardiac intensive care unit (ICU); (2) referred to our facility for further care after being treated at a referring facility; or (3) treated by paramedics and transported to our hospital. Our pediatric tertiary facility is the only children's hospital in Western Pennsylvania and therefore has a large catchment area. The study was approved by the institutional review board of the University of Pittsburgh.

Acute episodes of SVT were identified by 3 mechanisms. We first identified episodes of acute SVT by searching the electronic medical record for *International Classification of Diseases, Ninth Revision* (ICD-9) codes 427.0 (SVT), 427.89 (other arrhythmia), 427.1 (ventricular tachycardia), 427.41 (ventricular fibrillation), and 427.9 (cardiac arrhythmia). This query result was cross-referenced against ICD-9 code 99.62 for cardioversion to identify any potential patients who were treated by synchronized cardioversion and did not receive the ICD-9 code for SVT. In addition, a separately maintained cardiology database containing all patients with a diagnosis of SVT followed by our cardiology division was reviewed, and the medical records of all patients contained in this database and who were not captured by our ICD-9 code search were included. Patients with atrial tachycardia, atrial flutter, atrial fibrillation, and ventricular tachycardia were excluded from the analysis. For each patient identified via the use of these methods, we reviewed the medical record and identified each episode of acute SVT.

SVT was defined as tachycardia originating above the ventricles but not including atrial fibrillation or flutter. An episode of SVT was defined as documentation in the attending physician's clinical note and confirmation of the diagnosis on subsequent cardiology notes, specific appearance of electrocardiogram on a 12-lead electrocardiogram or telemetry monitor strip (tachycardia and absence of beat-to-beat variability), and treatment with vagal maneuvers, adenosine, other antiarrhythmics, or documentation of spontaneous resolution of SVT in the medical record. First presentation of SVT was defined as the first time a patient presented to our facility for treatment of SVT during the study period. Spontaneous conversion was defined as conversion to sinus rhythm in the absence of vagal maneuvers or administration of medications. Refractory SVT was defined as an episode of SVT that did not convert to sinus rhythm despite the administration of 2 doses of adenosine at or above the AHA-recommended doses. Unstable SVT was defined as the occurrence of hypotension, altered mental status, or documentation on physical examination of poor perfusion during an episode of SVT.[5] Hypotension was recorded as such if there was documentation of hypotension in the physician's medical record and the systolic blood pressure was lower than 2 × age (years) + 70 mm Hg. The general management of low blood pressure measurement in our ED consists of manual blood pressure measurement if the automated blood pressure measurement is below the normal range. Response to a specific maneuver was defined as conversion to sinus rhythm and maintenance of the sinus rhythm for at least 5 minutes after the respective intervention, which is consistent with definitions of conversion in previous publications of adenosine efficacy.[9] Some episodes responded to a specific therapy; however, the SVT recurred during the hospital stay, requiring further therapy. The subsequent SVT episodes occurring during the hospital stay were not included in the analysis.

Once identified as an acute SVT episode, each encounter was reviewed, including ED records, records of the hospital course, prehospital and transport records, and referring facilities' records when applicable. Where available, the results of echocardiogram were recorded.

Statistical Analyses

Research data were collected with the Research Electronic Data Capture (REDCap) software. Data were analyzed with the statistical package SigmaStat (Systat Software, Inc, San Jose, California). Data are presented as mean ± SD. The outcome variables of interest were spontaneous cessation of SVT, response to vagal maneuvers, response to first dose of adenosine, response to second dose of adenosine, and presence of refractory SVT, all of which were classified as one for "yes" and zero for "no." Multiple linear logistic regression models were fit to test whether age was a risk factor for different SVT therapies. Model fit statistics such as deviance and Pearson χ^2 statistic were examined, as well as Pearson residuals. No assumption violations were detected by examining the model fit and residual plots. The χ^2 test was used to compare the outcome for different treatments of acute SVT in the age groups: younger than 1 year vs 1 year and older, and weight groups: <10 kg vs ≥10 kg.

Results

During the 10-year study period, we identified 179 episodes of acute SVT in 134 patients. Of these patients, 110 had a single episode, and 24 had multiple episodes. Demographics for our patient population are presented in **Table I** (available at www.jpeds.com). Mean age was 7.4 ± 5.8 years, racial breakdown was 85% white and 15% black, and breakdown by sex was 62% male and 38% female patients. A history of SVT was recorded in 25% of patients, and 9.7% were treated with daily antiarrhythmics. The management of SVT in our population is presented in **Figure 1** (available at www.jpeds.com).