Child Neurology Open
Volume 8: 1-3

DOI: 10.1177/2329048X21995296
journals.sagepub.com/home/cno
SAGE

Infant Stroke Associated With Left Atrial Thrombus and Supraventricular Tachycardia

Rachel Pauley, MD[1], Elise L. Mercier, MD[2], Ashutosh Kumar, MBBS[3], William H. Trescher, MD[3], and Gayatra Mainali, MBBS[3]

Abstract

We report a rare case of cardioembolic stroke in the setting of supraventricular tachycardia (SVT) in an infant. After a week of irritability, a 10-week-old male presented to the emergency department with SVT requiring treatment with adenosine. He developed right-sided hemiparesis and focal motor seizures. Imaging of the brain showed ischemic infarct in the left middle cerebral artery (MCA) territory. Echocardiogram showed a newly formed large left atrial intracardiac thrombus. A coagulopathy workup was negative. He was treated with beta-blocker and anticoagulation therapy. He had mild residual right hemiparesis. During childhood, he developed medically refractory focal epilepsy from the left hemisphere, requiring epilepsy surgery at age 10. A child presenting with sustained SVT can be at increased risk for intracardiac thrombi and stroke.

Keywords

arterial ischemic stroke (AIS), supraventricular tachycardia (SVT), intracardiac thrombi, focal epilepsy, hemiparesis, developmental delay, ADHD

Received September 2, 2020. Received revised December 23, 2020. Accepted for publication January 12, 2021.

Introduction

The incidence of arterial ischemic stroke (AIS) in the pediatric population is estimated to be 5 to 10 per 100,000 children per year.[1] Risk factors of AIS include cerebral arteriopathy, cardiac disorders, systemic conditions, head and neck disorders, infection, atherosclerosis, and prothrombotic states.[2] These risk factors were identified in 91% of patients with AIS.[2] Over 30% of patients had underlying cardiac disease, consisting of structural abnormalities, arrhythmias, cardiac surgery or catheterization, left ventricular assist device, and ECMO.[2] However, only 2 out of the 676 children with AIS had an arrhythmia in the International Pediatric Stroke Study.[2] Supraventricular tachycardia (SVT), the most common childhood arrhythmia, is generally considered a treatable condition with an excellent prognosis.[3,4] We present a case of a previously healthy infant, without any known risk factors, who developed a cardioembolic stroke in the setting of sustained SVT. The aim of this case report is to investigate SVT as a potential risk factor for cardioembolic stroke in children and discuss long-term sequelae.

Case Presentation

A 10-week-old previously healthy male presented to his primary care physician with a 1-week history of increased irritability and decreased oral intake. In the office, his heart rate was 280 beats per minute. He was sent to the emergency department where an EKG showed SVT. He was converted to normal sinus rhythm following several adenosine boluses. A repeat EKG revealed Wolff-Parkinson-White syndrome as the underlying cause of SVT. He was started on propranolol.

[1] Department of Neurology, Division of Child Neurology, NYU Langone Medical Center, New York, NY, USA
[2] Department of Pediatrics, Division of Child Neurology, St. Christopher's Hospital for Children, Philadelphia, PA, USA
[3] Department of Pediatrics, Division of Child Neurology, Penn State Health College of Medicine, Hershey, PA, USA

Corresponding Author:
Rachel Pauley, MD, Department of Neurology, New York University School of Medicine, 462 1st Ave RM 7W12C, New York, NY 10016, USA.
Email: rachel.pauley@nyulangone.org

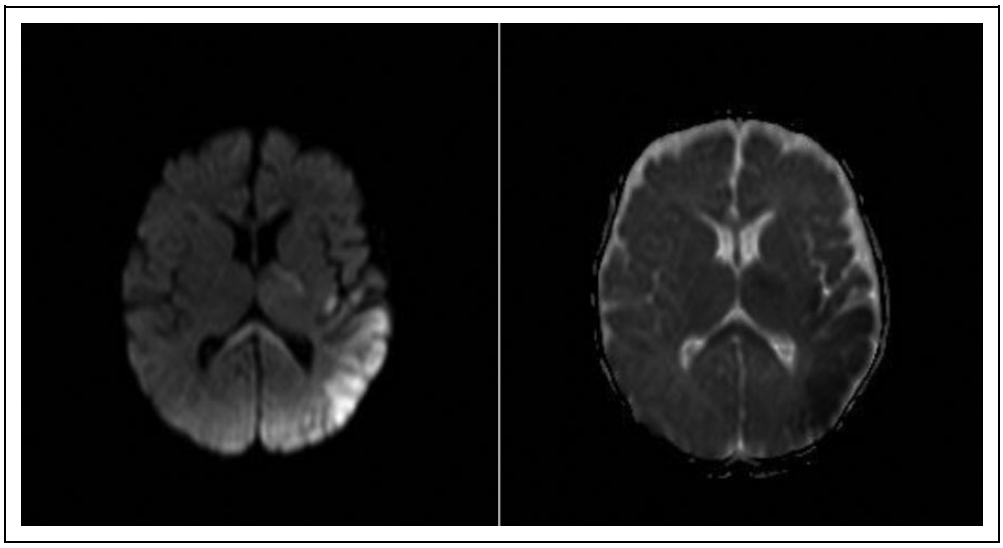

Figure 1. Diffusion weighted magnetic resonance imaging (left) with apparent diffusion coefficient mapping (right) revealing acute infarct within the left posterior middle cerebral artery territory.

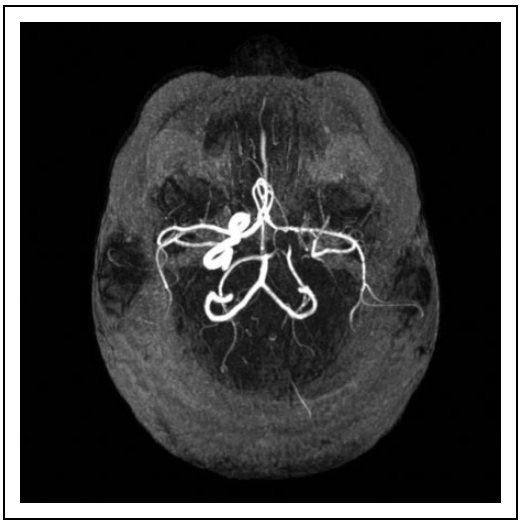

Figure 2. Magnetic resonance angiogram revealing absence of flow in left internal carotid artery distal to the carotid bifurcation.

His initial echocardiogram described mild left atrial enlargement, mild mitral insufficiency, but otherwise normal structure and function.

He had a second episode of SVT on his first day of hospitalization, which resolved with adenosine. The following day, he had 3 episodes of right arm clonic movements with right gaze deviation, each lasting around 2 minutes. He had diminished spontaneous movement of the right arm and leg on physical exam prompting neuroimaging. A head computed tomography (CT) without contrast showed hypodensity and sulcal effacement in the left MCA distribution. Magnetic resonance imaging (MRI) identified a left MCA infarct involving the frontal, parietal, and temporal lobe, including the thalamus and internal capsule (Figure 1). A head and neck magnetic resonance angiogram (MRA) with and without contrast revealed occlusion of the left internal carotid artery (ICA) distal to the carotid bifurcation (Figure 2). On the fourth day of hospitalization, a repeat echocardiogram showed a 5×7 mm left atrial appendage thrombus. This thrombus was present on subsequent review of his initial echocardiogram but was less mobile and distinct. Hypercoagulable workup (including PT/PTT/INR, protein C, protein S, antithrombin III, prothrombin G20210, lupus anticoagulant, factor V Leiden, and homocysteine) was unremarkable. An electroencephalogram (EEG) showed focal cerebral dysfunction over the left hemisphere without epileptiform activity.

He was discharged on phenobarbital, propranolol, enoxaparin, and aspirin. In the absence of seizures, his phenobarbital was tapered and discontinued 2 weeks later. Serial echocardiograms revealed a structurally normal heart and resolving thrombus, with complete resolution at 9 months. His enoxaparin and propranolol were discontinued after 1 year. From a cardiac perspective, he remained stable until he had a recurrent episode of SVT at 2 years of age in the setting of a febrile illness. The episode of SVT resolved after an adenosine bolus. His parents declined surgical ablation, and he was treated with atenolol for 1 year.

Neurodevelopmentally, he met typical milestones in infancy, except for a mild speech delay. Motor function was not clinically impaired; he was left-hand dominant and right-hand dysfunction was detected only with fine motor testing. At age 4, he developed attention deficit hyperactivity disorder (ADHD). When he was 6-years-old, he was hospitalized for new-onset seizures and diagnosed with focal epilepsy from the left frontal-temporal-parietal region. His seizures became medically refractory to multiple antiseizure medications. He was referred to a surgical epilepsy center where he had stereo-EEG (depth electrodes) for localization and subsequent left fronto-parietal craniotomy for resection of epileptogenic tissue at 10-years-old. After surgery, his seizures improved,

but he developed right hemiplegia, right hemisensory loss, and right homonymous hemianopsia.

Discussion

SVT is the most common childhood arrhythmia, with approximately half of SVT cases presenting within the first year of life.[3,4] Symptoms of SVT in infants are nonspecific, including lethargy, poor feeding, vomiting, and diaphoresis.[4] Infants may have higher risk of complications due to prevalence and delayed diagnosis. Overall, complications are infrequent in children and include cardiomyopathy, congestive heart failure, and death.[3]

SVT is a risk factor for AIS in adults, although the incidence remains largely unknown.[5] In the pediatric population, AIS is a rare complication of SVT with only a few cases reported in the literature. Children were less than 5 years of age at time of diagnosis of SVT and stroke.[6–8] Including our patient, 3 children had infarcts in the left MCA territory, and 1 child had infarcts in multiple territories.[6–8] Nearly all cases of stroke were considered to be cardioembolic; one patient had left ICA narrowing on angiogram which may have contributed.[6–8] However, only our patient had an identified intracardiac thrombus on echocardiogram.

SVT can cause intracardiac thrombosis in the left-side of the heart.[9–12] The proposed mechanism of thrombosis is stasis of blood flow in the setting of poor contractility or enlargement of the left atrium or ventricle.[5,9,10] Aside from the patient with left ICA narrowing, all children with SVT and subsequent stroke had atrial enlargement and/or ventricular dysfunction on echocardiogram.[6–8] These findings suggest an increased risk for intracardiac thrombus formation. In addition, prothrombotic workups were negative or unknown in these patients.[6–8]

As many as 82% of children with AIS have focal neurologic signs at symptom onset and 31% of children present with seizures.[2] Children with AIS secondary to SVT had neurologic deficits including lethargy, aphasia, right hemiparesis, and a right hemifield defect.[6–8] Our patient developed right hemiparesis and focal seizures, which lead to the diagnosis of AIS. No other child was noted to have seizures.[6–8]

Over 75% of patients with AIS have long-term neurologic deficits.[1] Neurologic sequelae for our patient included mild speech delay, fine motor deficits, ADHD, and medically refractory focal epilepsy. Following epilepsy surgery, he developed major neurologic deficits of right homonymous hemianopsia, right hemiplegia, and right hemisensory loss. However, his overall quality of life improved due to a significant reduction in seizure burden.

Conclusions

SVT is a common condition in children which typically responds to medical treatment and cardiac ablation techniques in appropriate settings. Intracardiac thrombus formation is rare, but in this case, a left atrial thrombus led to an embolic stroke with long-term neurologic sequelae.

Declaration of Conflicting Interests

The authors have no potential conflicts of interest regarding research, authorship, and/or publication of this article. Case reports are exempt from our institutional IRB. Consent was obtained from parents prior to publication.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

ORCID iD

Rachel Pauley, MD https://orcid.org/0000-0003-2949-1264

References

1. Amlie-Lefond C. Evaluation and acute management of ischemic stroke in infants and children. *Continuum (Minneap Minn)*. 2018; 24(1):150-170.
2. Mackay MT, Wiznitzer M, Benedict SL, Lee KJ, DeVeber GA, Ganesan V. Arterial ischemic stroke risk factors: the international pediatric stroke study. *Ann Neurol*. 2011;69(1):130-140.
3. Schlechte EA, Boramanand N, Funk M. Supraventricular tachycardia in the pediatric primary care setting: age-related presentation, diagnosis, and management. *J Pediatr Health Care*. 2008;22(5):289-299.
4. Vos P, Pulles-Heintzberger CF, Delhaas T. Supraventricular tachycardia: an incidental diagnosis in infants and difficult to prove in children. *Acta Paediatr*. 2003;92(9):1058-1061.
5. Rujirachun P, Wattanachayakul P, Winijkul A, Ungprasert P. Paroxysmal supraventricular tachycardia and risk of ischemic stroke: a systematic review and meta-analysis. *J Arrhythm*. 2019;35(3):499-505.
6. Atluru VL, Epstein LG, Gootman N. Childhood stroke and supraventricular tachycardia. *Pediatr Neurol*. 1985;1(1):54-56.
7. Müller GI, Ulmer HE, Bauer JA. Complications of chest thump for termination of supraventricular tachycardia in children. *Eur J Pediatr*. 1992;151(1):12-14.
8. Zapson DS, Riviello JJ, Bagwell S. Supraventricular tachycardia leading to stroke in childhood. *J Child Neurol*. 1995;10(3): 239-241.
9. Russo G, Trappan A, Benettoni A. Unusual left atrial appendage thrombus in a neonate with normal heart after sustained supraventricular tachycardia. *Int J Cardiol*. 2008;131(1):e17-e19.
10. Hanséus K, Björkhem G. Left ventricular thrombosis during infancy: report of two cases. *Pediatr Cardiol*. 1995;16(4):182-185.
11. Ganesh AB, Mathew RC. Left atrial thrombus in a neonate with normal heart after sustained supraventricular tachycardia. *J Clin Diagn Res*. 2014;8(7):PD01-PD02.
12. Mitchelson BL, Skinner JR, Donnell CO. Multiple atrial thrombi in a neonate presenting with supraventricular tachycardia. *HeartRhythm Case Rep*. 2015;2(1):71-73.