Cardiovascular collapse during amiodarone infusion in a hemodynamically compromised child with refractory supraventricular tachycardia

Sunil Saharan, Seshadri Balaji
Department of Pediatrics, Division of Pediatric Cardiology, Doernbecher Children's Hospital, Oregon Health and Science University, Portland, U S A

ABSTRACT

We describe a 7-week-old female infant who presented with refractory supraventricular tachycardia (SVT). During amiodarone infusion, she developed hypotension and cardiac arrest requiring extracorporeal membrane oxygenation (ECMO) support. After successful control of SVT using procainamide infusion, she was weaned from ECMO and discharged home on oral flecainide. We conclude that infants with acidosis, ventricular dysfunction, and prolonged refractory SVT may poorly tolerate intravenous amiodarone.

Keywords: ECMO, Refractory, SVT

INTRODUCTION

Most SVT episodes can be terminated with vagal maneuvers or intravenous (IV) adenosine bolus. Rarely, SVT can be refractory to initial treatment with adenosine or be recurrent after only brief conversion.[1] There is no well-defined treatment algorithm for these cases.

Intravenous amiodarone is a commonly used antiarrhythmic medication recommended for management of refractory arrhythmias in both pediatric and advanced life support algorithms (PALS, ALS).[2,3] The recommendation to use amiodarone for refractory arrhythmias in pediatric population is extrapolated from adult studies. The PALS algorithm is to consider either IV amiodarone (5 mg/kg IV/IO over 20-60 minutes and can be repeated twice) or procainamide for SVT if it does not respond to adenosine or electrical cardioversion. IV amiodarone does have adverse effects, including hypotension, acute liver dysfunction, and serious pulmonary toxicity.[4] There have been very few reported cases of cardiovascular collapse in literature following amiodarone infusion.[5-7] We report a case of severe cardiovascular collapse during amiodarone infusion that was rescued with ECMO and intravenous procainamide infusion.

Access this article online

Quick Response Code:

Website: www.annalspc.com

DOI: 10.4103/0974-2069.149519

CASE REPORT

A 7-week-old healthy female infant was admitted to an outside hospital with vomiting of two-week duration along with poor feeding and cold extremities noticed by her parents on the day of admission.

An initial diagnosis of SVT with left bundle branch block was made [Figure 1]. There was no evidence of either pre-excitation or non-conducted P waves on the electrocardiogram during brief periods of sinus rhythm, suggesting that it was due to an AV node-dependent mechanism. The presence of P waves suggested that it was likely due to a concealed accessory pathway [Figure 2]. The SVT briefly responded to multiple administrations of adenosine with immediate recurrence. Electrical cardioversion was not attempted because it was clear that "conversion" to sinus rhythm was not the limiting factor for this patient, and that the focus needed to be on the maintenance of sinus rhythm. Therefore, she was started on an esmolol infusion, after which she developed hypotension. At the same time, she was noted to have metabolic acidosis. At this point, she was intubated, started on mechanical ventilation, and transferred to our centre for further management.

During transport esmolol infusion was discontinued secondary to bradycardia and hypotension and with

Address for correspondence: Dr. Sunil Saharan, Department of Pediatrics, Doernbecher Children's Hospital, 707 SW Gaines Road, Mail code: CDRC-P, Portland, OR, 97239, USA. E-mail: saharan@ohsu.edu

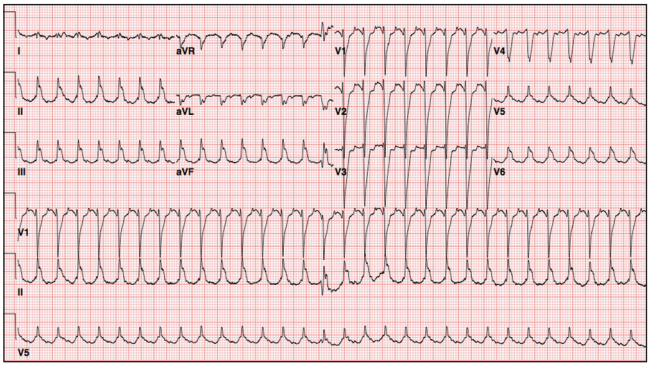

Figure 1: Narrow complex tachycardia with left bundle branch block

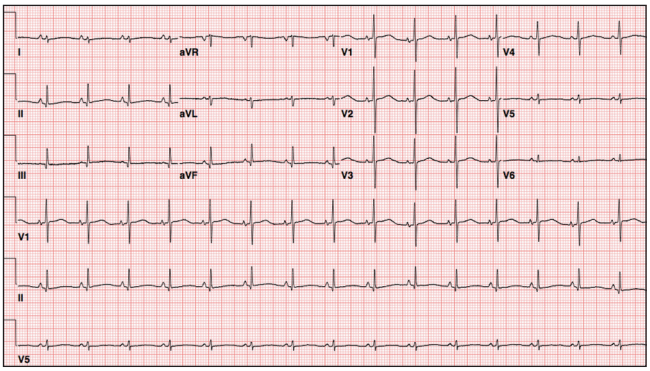

Figure 2: Normal sinus rhythm following treatment. ECMO: Extracorporeal membrane oxygenation, IV: Intravenous, PALS: Pediatric advanced life support, SVT: Supraventricular tachycardia

this, the SVT recurred. Upon arrival at our institution, the esmolol infusion was restarted, but she again developed hypotension with bradycardia; therefore, it was discontinued and an amiodarone bolus of 5 mg/kg was given IV over 60 minutes. This converted her rhythm from persistent SVT to intermittent runs of SVT. Since it appeared that amiodarone was helping to control the SVT, a second bolus of 5 mg/kg was started with a plan to infuse over 60 minutes. Throughout this time she had a significant metabolic acidosis with pH around 7.2 on the blood gas and lactate of above 15 mmol/L. Her echocardiogram showed severe left ventricular dysfunction while she was in sinus rhythm between episodes of SVT. During the second bolus of amiodarone she suddenly developed severe bradycardia and hypotension following which cardio-pulmonary resuscitation (CPR) was commenced. She was successfully placed on to venoarterial extra cardiac membrane oxygenation (ECMO) support. IV procainamide bolus (10 mg/kg) was given followed by an infusion initially at 20 mcg/kg/min with gradual increase to 60 mcg/kg/min (second bolus of 5 mg/kg in between) leading to conversion of SVT to sinus rhythm after about 14 hours from the initial bolus.

She was decannulated from the ECMO circuit on day 4 on infusions of milrinone and procainamide. On day 5, she was transitioned to oral flecainide followed by successful extubation from mechanical ventilation. A metabolic and genetic workup for cardiomyopathy was negative. During the hospital stay she also developed acute kidney injury, transient transaminitis, and thrombocytopenia all of which resolved gradually. She had normal biventricular function at discharge. At follow up 2 months post discharge, she has done well on oral flecainide and enalapril.

DISCUSSION

There is no commonly accepted treatment regime for cases refractory to initial therapy of SVT using either vagal maneuvers or adenosine. Therapies used in the context of refractory SVT include IV and oral digoxin, IV and oral beta-blockers, oral flecainide, oral sotalol, IV procainamide, IV amiodarone, electrical cardioversion, and transesophageal overdrive pacing. Calcium channel blockers are now rarely used for acute refractory SVT in infancy as they were reported to cause acute collapse and even death in infants.[8] While IV sotalol is available, there has been very little experience with its use. Flecainide is available only in oral form in the United States. Furthermore, all the IV antiarrhythmic medications available except digoxin (beta blockers, calcium blockers, procainamide, sotalol, and amiodarone) can cause acute hypotension. While hypotension associated with amiodarone is well documented, few cases of cardiovascular collapse in neonates have been reported.[6]

There are no clear guidelines regarding management of hemodynamically unstable patients with refractory SVT other than PALS recommendation of use of amiodarone or procainamide.[2] Amiodarone is a complex drug with multiple mechanisms of action and has been called the "king" of antiarrhythmics. However, the pre-eminence of amiodarone has recently been questioned.[9]

Amiodarone is thought to cause hypotension due to histamine release secondary to the solvent polysorbate 80.[10] Certain canine species, especially dogs, have been shown to be intolerant of this commonly used diluent, and some have suggested that it may be poorly tolerated by humans also.[11] Recently, a newer IV form of amiodarone (PM101) has been approved by FDA that uses cyclodextrin instead of polysorbate 80 and benzyl alcohol as solvent in order to reduce hemodynamic side effects and improve compatibility with other medications.[12]

Amiodarone also has calcium channel-blocking properties, and infants have been shown to tolerate calcium channel blockers poorly.[13] A number of reports of cardiovascular collapse with IV verapamil led to recommendations against its use in infants.[8,14] Whether pre-treatment with IV calcium can mitigate the hemodynamic effects

of amiodarone is not known. Our patient did not tolerate either esmolol or amiodarone, and this likely represents that the patient was in a fragile state with no cardiac output reserve. Radiofrequency ablation was not considered due to age and size of the child and also initial favorable response to intravenous amiodarone, which prompted us to administer the second bolus.

While procainamide was successful in controlling the arrhythmia in our patient, it too can cause negative inotropy and be deleterious for patients with ventricular dysfunction.[15] In a recent paper, Chang et al. suggested that procainamide may be more successful in controlling SVT than amiodarone with no increase in adverse effect frequency.[16] However, as expounded in an accompanying editorial by Saul and LaPage, the study had many flaws and in particular, the two population groups were not uniformly distributed as more cases with underlying congenital heart disease were in the amiodarone group.[17]

CONCLUSION

Our case illustrates the potential dangers of using IV amiodarone in hemodynamically unstable infants with refractory SVT. It is possible that a newly released form of amiodarone (PM101) may avoid the adverse effects associated with the currently available form of amiodarone in children.

REFERENCES

1. Salerno JC, Seslar SP. Supraventricular tachycardia. Arch Pediatr Adolesc Med 2009;163:268-74.
2. Kleinman ME, Chameides L, Schexnayder SM, Samson RA, Hazinski MF, Atkins DL, *et al.* Part 14: Pediatric advanced life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122:S876-908.
3. Neumar RW, Otto CW, Link MS, Kronick SL, Shuster M, Callaway CW, *et al.* Part 8: Adult advanced cardiovascular life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122:S729-67.
4. Connolly SJ. Evidence-based analysis of amiodarone efficacy and safety. Circulation 1999;100:2025-34.
5. Veltri EP, Reid PR. Sinus arrest with intravenous amiodarone. Am J Cardiol 1986;58:1110-1.
6. Ng GY, Hampson Evans DC, Murdoch LJ. Cardiovascular collapse after amiodarone administration in neonatal supraventicular tachycardia. Eur J Emerg Med 2003;10:323-5.
7. Jan SL, Fu YC, Lin MC, Hwang B. Precordial thump in a newborn with refractory supraventricular tachycardia and cardiovascular collapse after amiodarone administration. Eur J Emerg Med 2012;19:128-9.
8. Radford D. Side effects of verapamil in infants. Arch Dis Child 1983;58:465-6.
9. Saul JP, Scott WA, Brown S, Marantz P, Acevedo V, Etheridge SP, *et al.*; Intravenous Amiodarone Pediatric Investigators. Intravenous amiodarone for incessant tachyarrhythmias in children: A randomized, double-blind, antiarrhythmic drug trial. Circulation 2005;112:3470-7.
10. Masini E, Planchenault J, Pezziardi F, Gautier P, Gagnol JP. Histamine-releasing properties of Polysorbate 80 *in vitro* and *in vivo*: Correlation with its hypotensive action in the dog. Agents Actions 1985;16:470-7.
11. Munoz A, Karila P, Gallay P, Zettelmeier F, Messner P, Mery M, *et al.* A randomized hemodynamic comparison of intravenous amiodarone with and without Tween 80. Eur Heart J 1988;9:142-8.
12. Souney PF, Cooper WD, Cushing DJ. PM101: Intravenous amiodarone formulation changes can improve medication safety. Expert Opin Drug Saf 2010;9:319-33.
13. Epstein ML, Kiel EA, Victorica BE. Cardiac decompensation following verapamil therapy in infants with supraventricular tachycardia. Pediatrics 1985;75:737-40.
14. Perry JC, Garson A Jr. Diagnosis and treatment of arrhythmias. Adv Pediatr 1989;36:177-99.
15. Chapman MJ, Moran JL, O'Fathartaigh MS, Peisach AR, Cunningham DN. Management of atrial tachyarrhythmias in the critically ill: A comparison of intravenous procainamide and amiodarone. Intensive Care Med 1993;19:48-52.
16. Chang PM, Silka MJ, Moromisato DY, Bar-Cohen Y. Amiodarone versus procainamide for the acute treatment of recurrent supraventricular tachycardia in pediatric patients. Circ Arrhythm Electrophysiol 2010;3:134-40.
17. Saul JP, LaPage MJ. Is it time to tell the emperor he has no clothes?: Intravenous amiodarone for supraventricular arrhythmias in children. Circ Arrhythm Electrophysiol 2010;3:115-7.

How to cite this article: Saharan S, Balaji S. Cardiovascular collapse during amiodarone infusion in a hemodynamically compromised child with refractory supraventricular tachycardia. Ann Pediatr Card 2015;8:50-2.

Source of Support: Nil, **Conflict of Interest:** None declared