Tachycardia - Treatment Issues!

The PALS teaching of SVT (Supraventricular Tachycardia) is to revert to the sinus rhythm as early as possible, to prevent the onset of CHF or it's progression. A patient, already presenting in the cardiogenic shock (pale, lethargy, PR 180 /mt.) due to the 2nd episode of SVT has no time for vagal maneuvers and must be given Adenosine by a rapid push method (1st dose 0.1mg/kg up to 6 mg and subsequent 0.2 mg/kg up to 12mg). Meanwhile, the preparations must be done for the synchronised cardioversion (1st dose 0.05 -1J/Kg) and subsequent dose of 2 J/Kg). If the cardiac functions improve, the need for other means of treating shock and mechanical ventilation would logically decrease. These methods are so specific that if they fail to terminate the SVT, one must revisit the diagnosis, because the next drug Amiodarone though used in SVT, but is also used in ventricular arrhythmias.

In the present case management, there is no mention of using adenosine or cardioversion. In both the episodes of SVT, there was heavy dependence on either Amiodarone or the secondary measures to combat a shock.

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Editorial Note: *Authors did not respond to the above letter.*

Prolonged Sedation Following Administration of Oral Midazolam

Midazolam is a commonly used drug for procedural sedation in children due to its rapid onset, short duration of action and hemodynamic stability, which may be associated with improved patient acceptance. It is also associated with shorter recovery time and minimal risk of vomiting and respiratory depression, making it the drug of choice for conscious sedation in pediatric patients. We report an unusual drug interaction between midazolam and azithromycin leading to prolonged sedation in a child.

A 2-year-old male child presented with laceration in the thigh after falling down from bicycle. There was no evidence of head injury or injury to any internal organs. *X*-ray of thigh did not reveal any fractures. Suturing was planned after giving local anesthesia. One milligram of parenteral preparation of midazolam (0.1 mg/kg) was given orally as a premedication for sedation. Procedure was successfully completed. The child continued to remain sedated even after the procedure. The child was monitored in intensive care overnight. He was hemodynamically stable throughout and there was no evidence of respiratory depression. Though the child was arousable, he slipped into sleep immediately after that. Child regained consciousness the next day, but remained drowsy. We could not identify the cause of this unexplained prolonged drowsiness. Detailed history revealed that the child had fever for 3 days for which he had been prescribed oral azithromycin by a practitioner. The dose of 200 mg once daily (20 mg/kg) was given as syrup formulation for 3 days before oral midazolam was given. The patient was discharged the next day and was normal at follow up for suture removal.

Literature search revealed the possibility of drug interaction between midazolam and macrolides, erthyromycin in particular, an inhibitor of CYP3A which is a cytochrome P450 isoform responsible for midazolam hydroxylation [1]. There are reports of drug interaction between erythromycin and oral midazolam, leading to prolonged sedation [2]. Such interaction is also possible with azithromycin [1]. Azithromycin has not been found to increase the plasma concentrations of oral midazolam in few studies [3,4], though both these studies were done on healthy adult volunteers and not in children. The chance of drug interaction is minimal with intravenous midazolam since it bypasses the altered presystemic metabolism and its pharmacokinetics is not affected to the same extent as after oral administration [5]. As azithromycin is commonly prescribed in pediatric patients, physicians should be aware of this possible drug interaction with oral midazolam. If midazolam has to be

used with macrolides, intravenous route should be preferred.

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Camphor Poisoning: Personal Experience

I wish to share my experience regarding camphor poisoning during last 32 years. I happen to see at least 5 to 6 camphor poisoning cases per year. Though I have not kept a record of these cases, my observations are as follows:

1. Camphor poisoning is exclusively seen in Hindus for whom camphor is an important component of puja material.
2. Todlers 1 to 3 years are commonly involved since they have easy access to camphor when they are playing arround grandparents busy in performing puja with camphor arround.
3. Most common presentation is afebrile seizures.
4. Camphor is so rapid acting that child gets seizures within seconds of camphor ingestion.
5. Very small doses can cause seizures. I remember a 1-year-old child brought with seizures who had consumed prasad of coconut piece just coated with camphor because both camphor and coconut were lying in close vicinity in same puja thali.
6. Camphor poisoning is so common in our region that I have made a dictum that any small child if brought with afebrile seizures for the first time in life, always ask history of camphor ingestion. In 50 to 60% cases I could get positive history. Generally parents do not provide history of camphor ingestion unless asked for and I have seen patients getting investigated in detail for that afebrile seizure episode in form of CSF/CT/EEG etc, which is unnecessary if you can extract the correct history.
7. Generally a single dose of IV midazolam was found to be enough and patient became totally normal within one or two hours, with no residual deficit.

All the above observations are based not on literature but purely on personal experience and evidence in pediatric practice over last 32 years.

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Hair Dye Poisoning [Paraphenylenediamine, Super Vasamol 33]

A 14-year-old girl was brought with consumption of around 50 mL of Super vasamol 33 hair dye one hour prior to presentation. She hailed from a village, had lost her father and consumed the dye with suicidal intention. Immediately, gastric lavage was given and she was shifted to PICU. On examination, her vitals were stable and there was no respiratory distress or upper airway obstruction. She developed cervico-facial edema within 4 hours of dye ingestion. Other systems examination was unremarkable. Her blood counts, blood Urea, creatinine, calcium, phosphrous, sodium, potassium, chloride were normal. Urine for albumin, sugar and blood was not detected. Urinary pH was 7.0 and microscopy was normal. Her blood sugar, arterial blood gases, PT and