doi: 10.6515/ACS.202105_37(3).20210313B

Response to "Ventricular Tachycardia in Association with Propafenone Overdose" by Hyun Kuk Kim

Joshua Trebach, Sanjay Mohan and Robert S. Hoffman

To the editor,

We read with interest the article by Hyun Kuk Kim et al. entitled, "Ventricular Tachycardia in Association with Propafenone Overdose." We applaud the authors for eliciting the history regarding the supratherapeutic ingestion of propafenone, however, we respectfully disagree with the diagnosis of ventricular tachycardia (VT).

Propafenone's pharmacologic properties include blocking sodium and calcium channels and antagonizing beta-adrenergic receptors. The history of repeated supratherapeutic ingestion of propafenone followed by a very wide QRS complex tachycardia (WCT) should prompt an interpretation of the electrocardiogram not as VT, but instead, aberrantly conducted supraventricular tachycardia resulting from sodium channel blockade (SCB). At the very least, SCB must be on the differential for this WCT. The first line treatment for SCB would be 2 mEq/kg sodium bicarbonate IV followed by a repeat electrocardiogram to monitor for QRS narrowing and more sodium bicarbonate boluses as needed.[1-3] Should an antidysrhythmic medication be required, lidocaine is preferred, as it can safely manage both ventricular tachycardia and SCB-induced WCT.[4]

We found it unfortunate, though unsurprising, that the patient experienced a cardiac arrest after verapamil administration. As demonstrated by Sasyniuk et al., inducing bradycardia in WCT secondary to SCB will narrow the QRS complex due to use-dependent kinetics.[5] However, this may lead to cardiovascular collapse because of the combined negative inotropic effects of the two drugs. We therefore strongly advise against the use of beta-adrenergic antagonists or calcium channel blockers in patients with potential sodium channel blocker toxicity.

CONFLICT OF INTEREST

All the authors declare no conflict of interest.

REFERENCES

1. Nattel S, Mittleman M. Treatment of ventricular tachyarrhythmias resulting from amitriptyline toxicity in dogs. *J Pharmacol Exp Ther* 1984;231:430-5.
2. Pentel P, Benowitz N. Efficacy and mechanism of action of sodium bicarbonate in the treatment of desipramine toxicity in rats. *J Pharmacol Exp Ther* 1984;230:12-9.
3. Sasyniuk BI, Jhamandas V. Mechanism of reversal of toxic effects of amitriptyline on cardiac Purkinje fibers by sodium bicarbonate. *J Pharmacol Exp Ther* 1984;231:387-94.
4. Pentel PR, Benowitz NL. Tricyclic antidepressant poisoning. Management of arrhythmias. *Med Toxicol* 1986;1:101-21.
5. Sasyniuk BI, Jhamandas V, Valois M. Experimental amitriptyline intoxication: treatment of cardiac toxicity with sodium bicarbonate. *Ann Emerg Med* 1986;15:1052-9.

Received: February 10, 2021 Accepted: March 13, 2021
Division of Medical Toxicology, Ronald O. Perelman Department of Emergency Medicine, NYU Grossman School of Medicine, New York, NY, USA.
Corresponding author: Dr. Joshua Trebach, Division of Medical Toxicology, New York City Regional Poison Control Center, 455 First Avenue, Room 123, New York, New York 10016, USA. Tel: 212-447-4534; E-mail: joshua.trebach@nyulangone.org