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# Tropicamide-Induced Supraventricular Tachycardia During an Eye Exam

Daniel Miller, DO[1], Nicole Noff, MD[1], Gowri Swaminathan, MD[1], Natalie Balassiano, MD[1], Most S. Munira, MD[1]

[1] Icahn School of Medicine at Mount Sinai

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It is a well-established fact that commonly used ophthalmic medications have cardiovascular repercussions. Ophthalmologists are often aware of these effects as topical ophthalmic solutions are capable of producing severe cardiovascular side effects, including congestive heart failure, arrhythmias, and death. Cases such as this should be reported more in the world of medicine, as the adverse effects of medications not routinely used in the medical clinic can frequently get overlooked. The ophthalmic preparations are predominantly used to either facilitate diagnosis through mydriasis or treat glaucoma. Tropicamide is one such ophthalmic agent that is a short-acting cycloplegic and is commonly used to induce pupillary dilation through ciliary muscle paralysis for ophthalmic examinations. We present to you a rare case of supraventricular tachycardia (SVT) triggered by the use of Tropicamide ophthalmic solution during an eye exam in the outpatient setting.

## INTRODUCTION

Tropicamide is a muscarinic (cholinergic) antagonist with a pharmacological profile like that of atropine. As a weak drug in this class, tropicamide has a short-lived activity lasting about six hours, making it clinically useful in diagnosing refractive errors[1] and helping patients quickly return to their daily activities. It also causes paralysis of the ciliary muscle (cycloplegia) to prevent near vision accommodation, although it has only a little effect on far vision; it is said to have less impact on cycloplegia than mydriasis at low doses.[2,3] When applied topically to the cornea of the eye, tropicamide is rapidly absorbed; Vuori et al. observed that it reaches peak plasma concentration at 5 minutes.[4] Ocular side-effects of tropicamide include transient stinging and raised intra-ocular pressure; prolonged administration may cause local irritation, hyperemia, edema, and conjunctivitis (contact dermatitis).[5] Potential anticholinergic effects caused by tropicamide include dry mouth, high temperature, constipation, increased heart rate, and headache.[6] Due to the drug's low affinity for muscarinic receptors and very low receptor occupancy in plasma, tropicamide applied as an eye drop only rarely causes the aforementioned systemic effects.[4] The use of anticholinergic drugs carries a small risk of central nervous system (CNS) disturbance, including psychotic reactions and behavioral problems.[7] While adverse reactions to tropicamide are generally mild and transient, this case highlights the importance of considering cardiovascular effects as a potential adverse event. Cardiovascular side effects are less commonly reported and typically involve only mild increases in the heart rate*.* Herein, we present a case of supraventricular tachycardia (SVT) triggered by the use of tropicamide.

## CASE PRESENTATION

A 52-year-old female with a history of well-controlled hypertension and diabetes presented to the ophthalmology clinic for a routine eye examination. Upon arrival, the patient's blood pressure was 134/82 mm Hg, and her resting heart rate was 78 beats per minute (bpm). After a comprehensive eye examination, tropicamide 1% eye drops were instilled into both eyes for pupil dilation. Approximately 15 minutes after administration, the patient suddenly developed palpitations, chest discomfort, lightheadedness, and a sensation of rapid heartbeats. Clinic staff immediately performed a basic electrocardiogram, revealing a narrow-complex tachycardia consistent with supraventricular origin at 180 bpm. The patient was promptly transferred to the adjacent medical clinic for further evaluation. On examination, her blood pressure remained stable at 136/84 mm Hg, and her heart rate was 178 bpm. Physical examination did not reveal any other abnormal findings. Vagal maneuvers were attempted without success. The decision was made to administer adenosine. The patient experienced transient atrioventricular block following adenosine administration, which successfully terminated the SVT and restored sinus rhythm with a heart rate of 82 bpm. Further cardiac workup revealed no underlying structural or ischemic heart disease, including a comprehensive cardiac history, laboratory tests, echocardiography, and exercise stress testing. The patient was advised to avoid mydriatic agents in the future.

## DISCUSSION

Tropicamide has been referred to as a "seven-monther," which is the amount of time it takes to kill a young person without significant preexisting conditions.[8,9] Over the past several years, the misuse of eye drops such as tropicamide and cyclopentolate has increased tremendously due to their anticholinergic properties.[10] Abuse of these agents was previously limited to the former Soviet Union, but recent studies have shown that their misuse has spread beyond Eastern Europe[11]; tropicamide abuse via intravenous injection has been noted in several countries, including Turkey,[12] Italy,[13] France,[8] Tajikistan,[14] and Kazakhstan.[15] In clinical scenarios, tropicamide (0.5%) is commonly used to produce dilation of the pupils during a fundal examination; larger doses have been found to cause paralysis of the ciliary muscle, i.e., cycloplegia, in addition to mydriasis.[16] This is similar to the large dose concentration of tropicamide our 52-year-old patient received during his ophthalmic examination.

Tropicamide, an antimuscarinic agent, is rapidly absorbed when applied to the cornea of the eye. Vuori et al. observed that the drug reached peak plasma concentration at 5 minutes and decreased rapidly to undetectable levels after 2 hours.[4] Tropicamide produces mydriasis, i.e., pupillary dilatation, in 20-40 minutes after instillation and lasts about 6 hours. Cycloplegia is maximal approximately 30 minutes after administration, with complete recovery of accommodation generally within 6 hours, as noted by Sweetman et al.[17] Our presented case of the 52-year-old experienced the adverse effect of supraventricular tachycardia (SVT) right after tropicamide reached its peak plasma concentration, confirming the pharmacokinetics described in the literature.

Vuori et al. studied the plasma levels and systemic anticholinergic activity of tropicamide after ocular administration in eight women, which were monitored using radioligand binding techniques. They found that tropicamide bound to muscarinic receptors of rat brain with an apparent equilibrium binding constant (Ki-value in plasma) 220 ± 25 nM (mean ± SD, n=3). They also found that the drug occupied only about 8% of the muscarinic receptors, and this negligible receptor occupancy explains the low incidence of systemic anticholinergic side effects encountered during routine use of these drugs. Our 52-year-old patient represents this rarity because she experienced a life-threatening anticholinergic activity due to tropicamide in the form of SVT during a routine fundal exam.

Tropicamide exerts its mydriatic effect primarily by blocking muscarinic receptors in the iris sphincter muscle, leading to relaxation and subsequent pupil dilation. While adverse reactions to tropicamide are typically mild and transient, such as blurred vision and ocular discomfort, this case highlights the importance of recognizing potential cardiovascular effects as adverse events. Anticholinergic medications like tropicamide are known to cause tachycardia due to vagal blockade, but the occurrence of SVT is relatively uncommon. The overall prevalence of SVT is 2.29 per 1000 individuals, of which about 60% is AVNRT (atrioventricular nodal reentrant tachycardia).[18] Arora et al. observed that topical application of 1% tropicamide to the posterior left atrium significantly attenuated vagal stimulation-induced effective refractory period shortening in the posterior left atrium, pulmonary veins, and left atrial appendage.[19] In this case, the patient had a history of hypertension; although well-controlled, this can lead to structural and electrical remodeling of the heart, increasing the susceptibility to arrhythmias triggered by pharmacological agents like tropicamide. The management of tropicamide-induced SVT involves prompt recognition and intervention. Vagal maneuvers should be attempted initially, followed by administration of adenosine to terminate the arrhythmia, similar to our presented case.

## CONCLUSION

The occurrence of SVT following the administration of tropicamide, a commonly used mydriatic agent in ophthalmic practice, is a rare but noteworthy event. While tachycardia is a rare side effect of anticholinergic medications due to vagal blockade, SVT is an infrequent occurrence.

Tropicamide-induced SVT is a diagnosis of exclusion, requiring careful evaluation of the patient's medical history, physical examination, and cardiac workup. This case underscores the need for ophthalmologists and other healthcare professionals to be aware of tropicamide's potential cardiovascular side effects, particularly in patients with pre-existing cardiac conditions. A multidisciplinary approach to management, including ophthalmology and cardiology collaboration, is crucial to ensure patient safety and optimal outcomes.

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