Extreme Transient Reflex Hypervagotonia Due to Pseudoephedrine Ingestion

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INTRODUCTION

A COMMON INGREDIENT in over-the-counter cold medications, pseudoephedrine has become a staple remedy in every household. Not many are aware, though, of its serious cardiovascular side effects. Pseudoephedrine is a stereoisomer of ephedrine. Pseudoephedrine causes direct presynaptic catecholamine release, blocks catecholamine reuptake and inhibits enzymes that breakdown catecholamines.[1] It is a non-selective agonist of alpha and beta adrenergic receptors, causing hypertension, dry mouth, anorexia, insomnia, vomiting, anxiety, tremor, restlessness, tachycardia, palpitations,[2,3,4] a dose-dependent response causing substantial elevations in systolic and diastolic blood pressures with higher doses of the drug,[5] coronary spasm and myocardial infarction.[6,7] The mean serum half-life of oral pseudoephedrine is 7 hours not taking into consideration the urine pH.[8,9] In this case report, a rare vasovagal cardiovascular effect of pseudoephedrine is described.

CASE PRESENTATION

A 46 year old male with history of obesity, hypertension, gastroesophageal reflux disease, no history of snoring, presented with sudden onset palpitations. Prior to admission, for a common cold, he took 30 ml of an over-the-counter cold preparation containing pseudoephedrine 30 mg/15 ml, twice daily for 2 days. Electrocardiogram revealed an atrioventricular nodal reentrant tachycardia at 127 beats per minute (BPM) with retrograde p waves 70 milliseconds from the onset of QRS complex. The arrhythmia reverted with 6 & 12 mg boluses of intravenous adenosine (Adenocard®, Sagent Pharmaceutical, Schaumburg, Illinois) to sinus rhythm at 88 BPM.

Twelve hours later, in the afternoon, while awake, the telemonitor showed an asymptomatic gradual slowing of sinus rate to 16 BPM and gradually progressive first degree atrioventricular block up to 220 milliseconds with subsequent return of sinus rate and PR interval to normal limits. The blood pressure was 157/93 millimeters of mercury. The subsequent course was uneventful, and he was discharged in stable condition. An outpatient Holter did not show significant bradycardia.

DISCUSSION

Only hypervagotonic states cause simultaneous suppression of the sinus node and atrioventricular node causing simultaneous transient sinus bradycardia with 1st degree AV block in patients not on antiarrhythmic medications. In our patient, hypervagotonia was cardioinhibitory with no vasodepressor component. Obstructive sleep apnea as a cause of hypervagotonia was ruled out due to non-occurrence of bradyarrhythmias during sleep. Pseudoephedrine is known to cause hypertension induced reflex increase in vagal tone and bradycardia.[10] It is proposed that a pseudoephedrine induced hyperadrenergic state caused vigorous contractions of ventricles, and activation of mechanoreceptors and afferent unmyelinated C fibers projecting centrally to the dorsal vagal nucleus of the medulla, leading to a paradoxical withdrawal of peripheral sympathetic tone and an increase in vagal tone, which in turn caused simultaneous bradycardia and 1st degree AV block.

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Ventricular hypercontractility due to catecholamine surge with resultant reflex bradyarrhythmias due to increased vagal tone are known side effects of pseudoephedrine. Our patient represents a rare case of extreme transient reflex hypervagotonia after pseudoephedrine ingestion which resolved spontaneously.

CONCLUSION

Clinicians must be aware of pseudoephedrine induced extreme vasovagal reactions causing simultaneous suppression of the sinus node and the atrioventricular node, so as to prevent unnecessary interventions and cardiac work up in patients who develop such side effects.

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EKG

Baseline EKG on admission showing AVNRT at 127 beats per minute (BPM) with retrograde p waves 70 milliseconds from the onset of QRS complex, reverting to sinus rhythm at 88 bpm after IV adenosine. Later, telemetry tracings showed asymptomatic gradual slowing of the sinus rate to 16/min and gradually progressive 1st degree AV block with subsequent return of sinus rate and PR interval to normal limits.