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Yohimbine Induced Type II Myocardial Injury: An Underrecognized and Dangerous Adverse Effect

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Abstract Yohimbine is an Indoloquinolizidine alkaloid derived from the bark of the African tree *Pausinystalia johimbe*, as well as from the bark of the unrelated South American tree *Aspidosperma quebracho-blanco*. It is commonly sold as a dietary supplement for the purpose of enhancing libido, weight loss and natural bodybuilding aids. Yohimbine has high affinity for the α_2-adrenergic receptor, moderate affinity for the α_1 receptor, dopamine D_2 receptor, and weak affinity for the dopamine D_3 receptors and some of the serotonin receptors. Depending on dosage, Yohimbine can either increase or decrease systemic blood pressure (through vasoconstriction or vasodilation, respectively). We present a 51-year-old male who presented with substernal chest pressure and the sensation of a "pounding heart" after starting Yohimbine as a dietary supplement. He was found to have type II myocardial injury characterized by elevated troponin levels. We propose that the mechanism for this was 1) elevation of blood pressure and sinus tachycardia causing myocardial ischemia due to increased myocardial oxygen demand and supply mismatch, 2) cardiac norepinephrine release causing direct non-ischemic norepinephrine mediated myocardial injury and 3) increased sympathetic outflow mediated coronary vasoconstriction and spasm causing ischemia.

Keywords: *Yohimbine, Hypertension, Norepinephrine, α_2 receptor, Type II NSTEMI, Tachycardia, Erectile Dysfunction*

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1. Introduction

Yohimbine is an Indoloquinolizidine alkaloid derived from the bark of the African tree *Pausinystalia johimbe*, as well as from the bark of the unrelated South American tree *Aspidosperma quebracho-blanco* [1,2,3]. Yohimbine is an alpha-2 adrenergic antagonist. While Yohimbine behaves as an aphrodisiac in some mammals, it does not do so in humans [8]. In the United States, Yohimbine preparations are sold as dietary supplements for enhancing libido, weight loss and as aids for bodybuilding [8]. Because Yohimbine has the highest affinity for the α_2 receptors, small doses can increase blood pressure by causing a relatively selective α_2 blockade [4]. We are presenting a 51-year-old male who presented with substernal pressure and the sensation of a "pounding heart" after recently starting a new "energy pill" called Yohimbine and was found to have type II Non-ST Segment Elevation Myocardial Infarction (NSTEMI).

2. Case Report

A 51-year-old male with a past medical history of hypertension, hypercholesterolemia, obstructive sleep apnea and active smoker presented to the emergency department complaining of a "pounding heart" and substernal pressure of 1 day in duration. He reported that he recently started a new "energy pill" called Yohimbine. Home medications included Losartan, Amlodipine and Rosuvastatin. At presentation, his heart rate (HR) was 132 beats per minute (bpm) and his blood pressure (BP) was 173/108 millimeters of mercury (mmHg). Physical examination was unremarkable. Laboratory data revealed an initial Troponin-I of 0.03 nanogram per milliliter (ng/mL) which trended up to 0.10 ng/mL after six hours, and subsequently returned to 0.04 ng/mL twelve hours later. Thyroid stimulating hormone was within normal ranges. Electrocardiogram initially revealed sinus tachycardia at 108 bpm, which later normalized (Figure 1 and Figure 2). Echocardiography revealed a normal left ventricular ejection fraction (LVEF) of 60%, with no wall motion or valvular abnormalities. Stress Myocardial Perfusion Imaging revealed a small area of reversible, mildly decreased perfusion in the anteroseptal and apical segment of the left myocardium. Subsequently, a coronary angiogram demonstrated only a mild 30% distal stenosis involving the distal Left Anterior Descending coronary artery. The patient's palpitations and chest discomfort subsided. HR & BP normalized in the hospital.

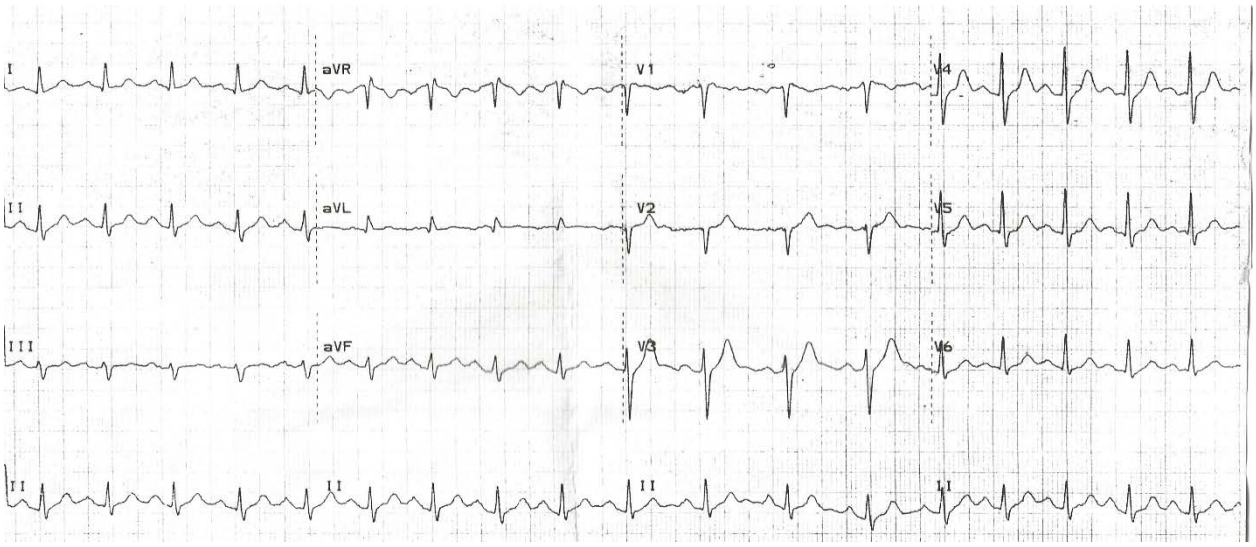

Figure 1. EKG showing sinus tachycardia at 108 bpm

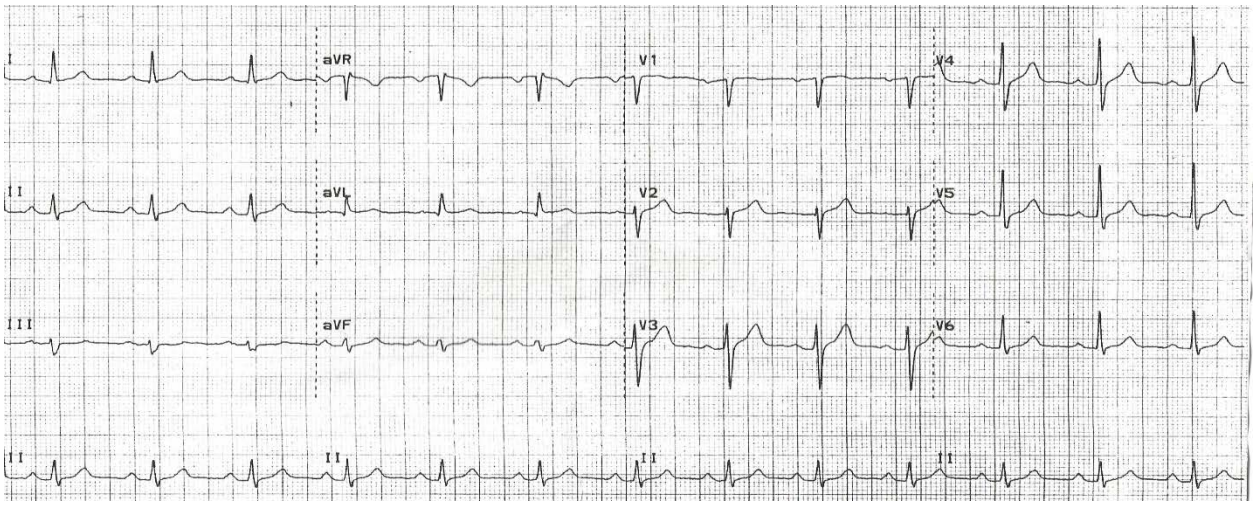

Figure 2. EKG showing resolution of sinus tachycardia

3. Discussion

Yohimbine is an Indoloquinolizidine alkaloid derived from the bark of the African tree *Pausinystalia johimbe* and from the bark of the unrelated South American tree *Aspidosperma quebracho-blanco [1,2,3]*. Yohimbine has high affinity for the α_2-adrenergic receptor, moderate affinity for the α_1 receptor, dopamine D_2 receptor, and weak affinity for the dopamine D_3 receptors and some of the serotonin receptors [4,5,6]. Depending on dosage, Yohimbine can either increase or decrease systemic blood pressure (through vasoconstriction or vasodilation, respectively). Because Yohimbine has the highest affinity for the α_2 receptor, small doses can increase blood pressure by causing a relatively selective α_2 blockade. Higher doses of oral Yohimbine may create numerous side effects, such as rapid heart rate, overstimulation, anomalous blood pressure, cold sweats, and insomnia. Yohimbine has been studied as a potential treatment for erectile dysfunction but there is insufficient evidence to rate its effectiveness. In some studies, the efficacy of Yohimbine as monotherapy in patients suffering from erectile dysfunction population is likely to be modest [2,7,8]. Yohimbine blocks the pre- and post-synaptic α_2 receptors. Blockade of post-synaptic α_2 receptors causes only minor corpus cavernosum smooth muscle relaxation, due to the fact that the majority of adrenoceptors in the corpus cavernosum are of the α_1 type. Blockade of pre-synaptic α_2 receptors facilitate the release of several neurotransmitters in the central and peripheral nervous system, such as nitric oxide and norepinephrine. Nitric oxide released in the corpus cavernosum is the major vasodilator contributing to the erectile process [8,9]. In our patient, we propose that Yohimbine caused Type 2 myocardial injury in the form of troponin elevation by: 1) elevation of BP and sinus tachycardia causing myocardial ischemia due to increased myocardial oxygen demand and supply mismatch [10], 2) Yohimbine induced cardiac norepinephrine release, causing direct non-ischemic norepinephrine mediated myocardial injury, a phenomenon also implicated in myocardial injury in the setting of heart failure and cocaine toxicity [11,12], and 3) increased sympathetic outflow mediated coronary vasoconstriction and spasm causing ischemia [13].

4. Conclusion

There is need for public awareness of the sinister cardiovascular side effects of Yohimbine to the extent of

causing myocardial injury, as it will continue to be a preferred alternative to prescription erectile dysfunction medications due to its quick onset of action when taken orally, its affordability and availability over the counter. We also urge clinicians to remember to inquire about Over-the-counter substances when taking a patient history.

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