Case Report

A case of ventricular tachycardia precipitated by caffeine: a rare occurrence

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ABSTRACT

The effects of caffeine vary according to the dose and patient. Thus, there is no bright line defining thresholds. Here, we present an unusual case of ventricular tachycardia precipitated by a dose of 450-500 mg of caffeine in a healthy 33-year-old man.

Keywords: Arrhythmia, Caffeine toxicity, Coffee, Ventricular tachycardia, Wide QRS complex tachycardia

INTRODUCTION

Caffeine is a methylxanthine derived alkaloid. It is naturally found in beverages such as coffee, tea, and energy drinks. This compound is the primary constituent of coffee and is the most widely consumed psychostimulant. Caffeine has been known to enhance the mind and heart rate, and thus has drawn speculation as a contributor to arrhythmia.1 Deleterious effects of caffeine on cardiovascular health have been an extensively debated topic garnering considerable attention.6 However, recent clinical studies have not been able to indicate harmful effects such as arrhythmia.1,3,4 In fact, some have even concluded beneficial properties of caffeine1. Caffeine has been considered safe in moderate amounts of ≤400 mg daily intake in healthy adults and a lethal compound capable of inducing considerable toxicity when consumed excessively. Hence, it demonstrates dose-dependent effects.2

CASE REPORT

A 33-year old male presented to this cardiology outpatient department with chief complaints of palpitations for the past 2 days. A detailed history elicited palpitations with skipped beats. They were sudden in both onset and offset and occurred for a few seconds before spontaneous relief. There was no precipitating or relieving factors or diurnal variation. The patient was a non-smoker and non-alcoholic. He did not have history of chest pain, shortness of breath, easy fatigability, syncope, generalized body swelling, fever, cough, focal neurological deficit, joint pain, gastrointestinal or genitourinary complaints. He also did not have significant medical history. The patient was a body-building enthusiast. He exercised for 3 hours daily at the gym which involved a regimen of various aerobic exercises, stretching, and strength training. He had a history of increased caffeine intake for the past 1 year as part of a modified diet to suit his gym regime. He would consume approximately 8-9 sachets of ‘Nescafe’ coffee (each sachet weighed 1.5 gm and contained 47.5 mg of caffeine) with water and additionally, he would consume approximately 3-4 standard cups of tea (each contained around 26 mg of caffeine). Thus, he consumed approximately 450-500 mg of caffeine daily for the past 1 year. General physical examination was unremarkable. His blood pressure was 120/70 mm Hg and pulse rate was 66 beats per minute. Cardiovascular examination was
unremarkable. Review of other systems also did not show any abnormality. Laboratory investigations included complete blood count, liver function tests, kidney function tests, serum electrolytes, thyroid profile and lipid profile which were all normal. Baseline electrocardiography (ECG), chest x-ray and echocardiography of the patient were also normal. A 24 hour Holter recording was performed which revealed multiple supraventricular and ventricular ectopics and runs of ill-sustained ventricular tachycardia (VT) (Figure 1). Subsequently, the patient was put on oral beta-blockers and amiodarone and caffeine cessation was advised. After initiating the treatment, another Holter recording was obtained after 5 days which was normal. Amiodarone was gradually tapered over a period of 3 months on follow-up basis. The patient remained asymptomatic during the follow-up period. The repeat Holter done at 3 months follow-up was also normal. Since caffeine cessation was associated with termination of VT, it was assumed that the excessive caffeine intake precipitated VT in this patient.

![Figure 1: Electrocardiography (A): runs of ventricular tachycardia, (B): premature ventricular contractions and uplet, (PVC), and (C): multiple PVC.](image)

**DISCUSSION**

Caffeine demonstrates dose-dependent effects. Mild stimulation may be attained from doses in the range 50-200 mg, whereas lethal caffeine intoxication may occur after ingestion of >5,000 mg, although rare. However, some individuals have experienced toxicity and lethality at doses not normally associated with such outcomes. Such a case was observed in our patient, a 33-year-old male who experienced palpitations precipitated by a dose of 450–500 mg of caffeine. This may be justified by caffeine tolerance which is individual variation in susceptibility to effects of caffeine. Thus, this case highlights that there is no bright line defining thresholds of daily caffeine intake.

Caffeine intoxication demonstrates a vast clinical spectrum affecting several organs. The most common presenting features are tachycardia, seizures, palpitations, hypotension, vomiting, nausea, hypokalemia, and metabolic acidosis. There are no definitive symptoms and symptoms vary from patient to patient. However, symptoms such as hypokalemia, and severe vomiting have been suggested as a means to differentiate caffeine ingestion from other sympathomimetic agents.2

Whilst diagnosing our patient a few other possible diagnoses seemed likely. Firstly, supraventricular tachycardia (SVT) with aberrancy was considered as the patient’s ECG displayed runs of wide-complex tachycardia (>120 ms) on the 24-hour Holter recording. However, baseline ECG was unremarkable without identification of pre-existent fascicular block or functional bundle-branch block. Thus, a diagnosis of SVT with aberrancy was ruled out. Secondly, SVT with intramyocardial conduction delay was considered. This is another differential of wide-complex tachycardia in which delayed conduction is observable in ventricular hypertyrophy and dilatation, cardiomyopathy and congenital heart disease. However, our patient had no evidence of structural heart disease as documented on the ECG, thus this diagnosis was unlikely. Thirdly, ventricular paced rhythm was considered. This represents another differentiated diagnosis of wide-complex tachycardia as contemporary systems are associated with stimulus artefacts. However, history of ventricular pacing in our patient was negative, thus this diagnosis was unlikely too.

Currently there is no standard management for caffeine overdoses. Literature contains a range of patient-tailored treatment strategies. However, management of VT and premature heartbeat should involve assessment of hemodynamic status and associated etiology. If the patient is hemodynamically unstable, hemodialysis can assist in removal of caffeine from the plasma, reduce rhythm discordance and morbidity as observed in several cases. If there is no evidence of hemodynamic instability and a reversible cause cannot be identified, antiarrhythmics such as amiodarone can be administered. Several cases have also reported successful treatment with beta-blockers. In addition to advice towards caffeine cessation, this patient was prescribed amiodarone and beta-blockers.

**CONCLUSION**

Caffeine is a simple and uncomplicated compound when consumed in moderate doses. However, when consumed excessively it transforms into a highly complex compound which may culminate into serious fatalities. This case highlights fatal consequences such as ventricular tachycardia after moderate consumption of caffeine, a rare occurrence.

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