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Case Report

KHAT ASSOCIATED CVS COMPLICATIONS: A CASE REPORT OF ETHIOPIAN MALE

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ABSTRACT

Introduction:

Khat or Catha edulis an indigenous flowering plant produces Cathinone, an alkaloid that is used as a drug of abuse in the African horn. As socio-cultural drug of abuse khat or qat has its roots of use dating back to 17th century. It is used as stimulant and increasing libido, alertness and concentration. Cathinone is a schedule I drug though it escapes regulatory restrictions in some countries. The sympathomimetic effects of cathinone are multifold including cardiovascular, neurological and gastrointestinal. The cardiovascular and neurological effects of cathinone mimics amphetamine.

Case presentation:

A 25 years Ethiopian male was referred to emergency with symptoms of chest pain and dyspnea. He underwent echocardiography, cardiac MRI and Cardiac X ray which showed acute renal failure, tachycardia and heart failure. He was a khat consumer. He was resuscitated with ionotropic agents and was discharged. He returned with reduced Left ventricular ejection fraction, dyspnea and ankle swelling after 7 weeks. Patient was recompensated and was suggested to withdraw Cathinone abuse which could lead to sudden cardiac death.

Conclusion: Cardiotoxic effects and symptoms of Cathinone needs to address properly and professionally.

List of abbreviations

MI	Myocardial infarction
LVEF	Left ventricular ejection Fraction
CMRI	Cardiac Magnetic resonance imaging

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INTRODUCTION

Khat or qat is an indigenous flowering plant and had remained as drug of abuse in the Yemeni culture in the 16th century. More than 20 M peoples across the Arabian peninsula and middle consume khat leaves as a part of their socio-cultural traditions. The use of khat is more dominant in male users as compared to female. The use of khat leaves is limited to these communities residing in US and Europe. The Somalia, Yemeni and Ethiopian communities living in USA, Germany and France and UK are major import consumers of khatleaves [1-3].

Today fresh leaves and twigs of the plant are chewed for their amphetamine like effects [3]. Khat includes alkaloids including like Cathinone, cathine, amphetamine and noradrenaline. Khat is now emerging as an alternative to amphetamine abuse and considered as natural amphetamine [4]. Cathinone is the β- keto analogue of amphetamine [5, 6].Cathinone is a schedule I drug while Cathine is Schedule IV drug albeit there is a lack of regulatory control as the legal status of these chemicals are not well defined[6, 7]. The abuse of khat is the major cause of its abuse. A single gathering ends with a consumption of 200 g- 500 g of the fresh leaves supplied in a banana wrapping[2, 8]. Approximate 90% of the alkaloidal agents are extracted through periodic chewing of leaves and are later spitted[9]. The physiological effects of khat includes sensations of happiness, energy boost, euphoria, spike in concentration and imagination. The cardiac, neurological, psychological and gastrointestinal effects appears with chronic use of khat leaves[7]. The indirect sympathomimetic activity of cathinone produced after 3-4 h causes elevations of heart rate and blood pressure and increases chances of arrhythmias, acute myocardial infarction in habitual users as reported by previous studies[10, 11]. The incidence of khat associated myocardial infarction increase in patients at risk[12]. Amphetamine is present along with cathinone in qat leaves. Abusive consumption of amphetamine is linked to cardiotoxicity and mortality. Amphetamine manifests prolonged increase in heart rate, elevated systolic blood pressure and respiratory rate for several hours [13]. Cardiotoxic effects of methamphetamine dual modal, catecholamine toxicity and direct effects on cardiovascular tissues

[9]. Besides cardiotoxicity, chronic intake of amphetamine present in qatleaves correlates with idiopathic pulmonary hypertension and consequently leads to prolong hypertension and further cardiomyopathy [14]. Furthermore, amphetamine Abuser have higher percentage of echocardiographic Left ventricular (LV) and reduction in LVejection [14]. Heart failure is now attributed primarily to elevated levels of catecholamine [15]. As evident from similarity between cardiomyopathy caused by amphetamine andcatecholamine inducedcardiomyothy[16]. Lastly, coronary artery disease and possible MI is linked with amphetamine abuse. Myocardial contrast echocardiography is used to detect earlier abnormalities in microcirculation and reduction of cardiac perfusion [17, 18]. Myocardial infarction can precipitate without coronary artery disease. Physical examination aids in assessing signs of myocardial infarction. 12-lead ECG, Urine analysis, echocardiography, 3d Speckle tracing imaging markers, cardiac MRI are some of the diagnostic tools for tracing cardiac damage.

CASE REPORT

A 25 years adult Ethiopian male was presented to emergency department complaining of abdominal pain in upper region with cardiogenic shock. His physical examination showed pale, cold and sweaty skin, a grade 2 systolic murmur, increased heart rate (tachycardia) with blood pressure (107/80), which was below par. He was suffering from difficulty in breathing and his ankles were also swelled from 2 weeks. His medical history showed that he was not suffering from a disease in past. He confessed methamphetamine with alcohol and tobacco from past 4.5 years.

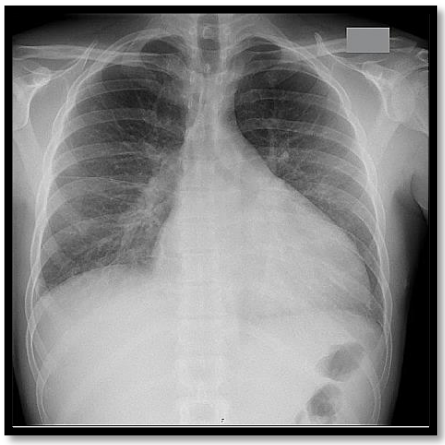

Figure 1: Cardiomegaly and decongested Lungs in X-ray

Laboratory diagnostics showed acute renal failure and elevated hepatic enzymes.

He was referred for ECG. The initial ECG showed sinus tachycardia (107 bpm) and delayed R segment progression. X ray showed cardiac dilatation and pulmonary edema as shown in figure 2. Furthermore, echocardiography revealed global hypokinesia with a left wall ventricular ejection fraction (LVEF) of 20% and moderate mitral regurgitation. He was immediately admitted in ICU andionotropic therapy along with vasopressors were initiated to mitigate symptoms. Left cardiac catheterization ruled out coronary heart disease. Additionally, cardiac Magnetic resonance imaging ruled out incidence of myocarditis or takotsubo cardiomyopathy. The prevalent symptoms were attributed crystal meth induced cardiomyopathy. Heat failure was treated with Lisinopril 5 mg once daily dosing. He was closely monitored in outpatient department after equipping with defibrillator vest. He was discharged after a week but he was admitted after 7 weeks due to difficulty in breathing and ankle swelling.

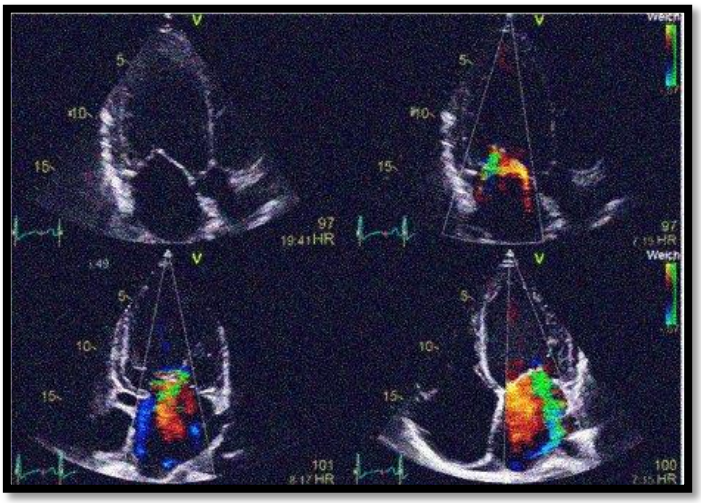

Figure 2: Left Ventricular dilatation with moderate to severe mitral value regurgitation in Transthoracic Echocardiography

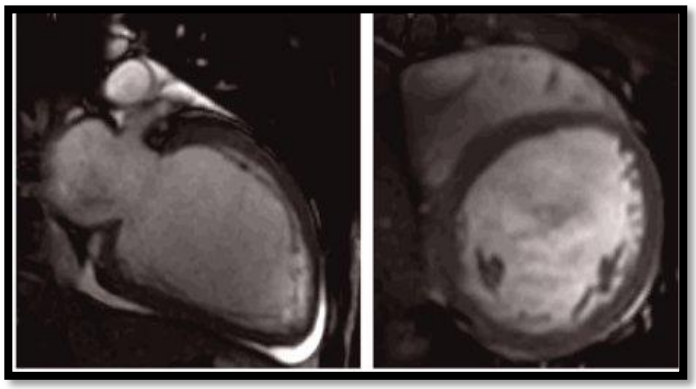

Figure 3 : Left Ventricular Enlargement in Cardiac MRI

He admitted poor medication compliance as well as continuous consumption of MA. Echocardiography showed another LVEF. He was recompensated, and was discharged with proper outpatient care setting. He was advised for drug abuse rehabilitation as these complications can prove fatal any time or next time.

DISCUSSION

Khat or qat is a regionally produced drug of abuse of east Africa and middle east particularly Yemen and consumed during social gatherings during cultural events [19]. There are more a dozen varieties of Catha edulis growing in different regions of east Africa and Yemen. More than 44 types of khat plants are cultivated in these regions [20]. The sympathomimetic effects of active chemical cathinone and cathine present in the fresh harvested leaves are related to amphetamine in their sympathomimetic effects [21]. Beside amphetamine like cathinone, actual amount of amphetamine are also present in the leaves of khat[22]. Overconsumption of khat or qat causes increased blood pressure, increased risks of acute myocardial infarction [23]. Amphetamine is another chemical abused and qat has significant amount of amphetamine in its leaves. The overuse of amphetamine causes vasoconstrictions, pulmonary arterial hypertension, fatty plaques, arrhythmias, cardiogenic shock and cardiomyopathy [24]. Khat leaves containing sympathomimeticscauses' catecholamine toxicity because of increased affinity for dopamine and norepinephrine toxicity [25] Amphetamine abusers are at risk of MI as reported in a previous study. Concentric myocardial hypertrophy, coupled with infarcts in different regions coupled with perivascular and interstitial fibrosis, cellular vacuolization and destructed myocytes in one case report [26]. The cardiogenic effects of cathinone and related ingredients in khats are reversed positively when consumption of khat is stopped as improvement in heart rate, R-peak amplitude reduction, QRS duration and RR intervals which decreases chances of premature ventricular contraction and arrhythmia [27]. Like cathinone, amphetamine carries same ratio of acute myocardial infarction and cardiovascular toxicity potential. These cardiotoxic effects can prove fatal if not managed on time [28].

CONCLUSION

Patient suffering from khatleaves associatedcardiotoxicity was properly rehabilitated after differential diagnosis.

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