Khat and stroke

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Abstract

Khat chewing, though a tradition followed majorly in African countries, has of late spread widely across the globe due to faster transport systems and advanced preservation techniques. Many complications such as psychosis, arterial hypertension, angina pectoris, and myocardial infarction have been reported in connection to khat abuse. We present a case of a young man who presented with acute onset left-sided weakness. He was a known khat addict for over three decades. A diagnosis of left hemiplegia due to right middle cerebral artery infarction was established. Detailed evaluation revealed no significant underlying cause for stroke. Since the main central nervous system effects of khat are comparable with those of amphetamines and there are established reports of stroke in amphetamine abuse, the former was assumed to be the etiological factor. The patient was discontinued from taking khat and was managed conservatively. The subject showed significant recovery with no further complications or similar episodes during follow-up. To the best of our knowledge, this is the second case of stroke associated with khat. Since the management is essentially conservative, a vigilant history eliciting of khat abuse in prevalent countries would cut down unnecessary healthcare costs.

Key Words

Amphetamine-like, khat, stroke

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Introduction

Khat (Gat, Qat) is been a part of culture for centuries in many countries in the Middle East. Khat is the leaves of the plant *Catha edulis* chewed for their stimulant effect. It is produced in large quantities in Yemen, Somalia, and East Africa.[1] The pleasure derived from khat chewing is attributed to the euphoric actions of cathinone, a substance with sympathomimetic amine properties similar to amphetamine.[2] The consumption of the leaves was limited to the African continent for centuries as the stimulant effects are seen in only fresh leaves. Of late, the problem has spread to Asia, Europe, and American continents, courtesy airlift and preservation of freshness.[3] Long-term use of Khat has been attributed to development of psychotic symptoms and other health adverse effects.[4]

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Pharmacological studies on khat showed that there are the following three main alkaloids present in khat leaves: cathinone, cathine, and ephedrine. Cathinone is the main active substance in khat leaves. It is amphetamine-like substance that releases endogenous catecholamine from peripheral and central neurones.[4] Its major metabolites are nor-pseudoephedrine and ephedrine. These two substances have weaker sympathomimetic activities and central stimulant properties.[5]

Of late, a number of complications due to chronic khat chewing are being reported.[1,2] However, central nervous system (CNS) complications in these conditions are rare. We report a case of stroke in a middle-aged male, which was probably due to chronic khat use.

Case Report

A 47-year-old man from Sana'a, Yemen, presented with sudden onset left-sided weakness with deviation of mouth to the right side since day 1. History of comorbidity-Type 2 diabetes, hypertension, or ischemic heart disease was absent. There was no history of alcohol, drug intake, or smoking. The patient was a regular Khat chewer since 30 years (5-6 packets a day—each packet containing nearly 100 g—over 6 to 8 hours spread over the day). There was no significant family history.

On examination, patient was conscious, oriented, and afebrile. Pulse was 80/minute, regular, good volume with no carotid bruit. Blood pressure measured 200/100 mmHg. The patient was right handed. CNS examination revealed left-sided hemiplegia (upper and lower limb strength 0/5) with left-sided upper motor neuron facial palsy. Fundus examination was normal. There were no signs of aphasia, agraphia, alexia, apraxia, or sensory lesions on further neurological examination. Cardiovascular, respiratory, and abdominal systems were normal.

Routine investigations (hemoglobin - 11.4 g%, total white cell count- 6.6 x 10^9 cells/l, neutrophils/lymphocytes/monocytes/eosinophils - 69/26/3/2, Erythrocyte Sedimentation Rate-6, random blood sugar - 96 mg%, serum electrolytes [sodium - 137 mmol/l, potassium - 3.5 mmol/l, calcium - 1.16 mmol/l], urine analysis) were normal. Electrocardiography and transthoracic echocardiography were normal. Carotid and vertebral artery Doppler studies were normal. Computed tomography of brain (plain and contrast) showed acute right middle cerebral artery infarction. Magnetic resonance angiogram of vertebral, carotid, and cerebral arteries was normal. A detailed work-up for common etiologies of stroke in the young people, which included lipid profile (total cholesterol - 140mg/l, high density lipoprotein- 35 mg/l, low density lipoprotein - 85 mg/l, triglycerides - 120 mg/l), anticardiolipin antibodies (IgG - 6.8, IgM - 2.1), protein-C (0.9) and protein-S (0.8), serum homocysteine level (13 μmol/l), and antiphospholipid antibody (IgG - 7.5, IgM - 3.0), were normal. tests for infections (Hepatitis B and C, Human Immunodeficiency Virus) and syphilis were negative.

A diagnosis of left hemiplegia due to right middle cerebral artery infarction was established and the patient was treated for ischemic stroke as per hospital protocol. The patient was discontinued from taking khat. Blood pressure was stabilized by antihypertensive. Following conservative treatment and physiotherapy, the patient recovered gradually over months. After six months, the strength in the left limbs was 4/5.

Discussion

Cathinone acts on CNS, inducing catecholamine release from postsynaptic storage, and effects include tachycardia, vasoconstriction, and increase in blood pressure, respiratory rate, and body temperature. All these effects including the role of catecholamines inducing platelet aggregation, increase the risk of cardiovascular events.[6]

Miranda and O'Neill reported stroke-like complications following amphetamine abuse.[7] Since khat is similar to amphetamines, the complication arising due to abuse can be assumed to be similar. Literature review showed this article to be the second of its kind where stroke is seen as a complication of long-term khat chewing. Vanwalleghem *et al*. reported a case of stroke associated with khat in a Somalian immigrant in Belgium.[8] Gulf-RACE group studied the association of khat chewing and the risk of stroke by using a multinational prospective design of hospitalized acute coronary syndrome (ACS) patients and found that khat chewers were more likely to die after ACS and more likely to develop post-ACS stroke and cardiogenic shock.[9]

In the present case, the patient had no high-risk factors which could lead to stroke. The common causes of stroke in this age group were ruled out by detailed work-up. Also, the patient did not manifest with any other cerebral complication following khat discontinuation. All these suggest khat chewing as a strong contender as the etiology for stroke.

The major limitation in this report is that transesophageal echocardiography for a clear view of atrial septum and atrium and its appendage could not be done due to unavailability. Also, the genetic predisposition to khat-associated vascular changes in this population, as suggested by Alkadi *et al*., could not be ruled out due to lack of appropriate technology.

Conclusion

In a multiethnic environment, immigration to developed countries in search of better standard of living is very common. Knowledge about the neurological manifestations of this ancient habit of khat chewing is essential to be considered as differential in similar presentations, especially in immigrants. Since the management of such cases is essentially conservative with discontinuation of khat use, an early diagnosis is important to minimize cost of healthcare. Although there is no known documentation of khat use in India, the association of same with stroke is to be borne in mind owing to increasing global migration and medical tourism in our country.

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