

Case Report

Marijuana Induced Transient Global Amnesia

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Abstract

Transient Global Amnesia is an acute onset of Anterograde and retrograde memory loss, with unknown etiology [1]. Attacks can occur during stressful periods, exertion, or with emotional circumstances, as long as some other aggravators such as immersion into cold water, and sexual intercourse [2]. Cognitive functions other than memory, specifically self-awareness, are spared [3].

Marijuana use has been linked to transient global amnesia in some cases [4-6], presumably through the effect of $\Delta 9$ -Tetrahydrocannabinol (THC) on the Cannabinoid receptors (CB1) that are located presynaptically in the lipid membranes of neurons, which results in neuromodulation with changing the number of neurochemicals such as glutamergic, GABAergic, and cholinergic systems, all of which end up affecting the memory [7].

INTRODUCTION

In today's world, the most used and most cultivated illicit drug is Cannabis. The drug is used in varying compositions and hence there could be changes in potency with use. There needs to be constant reassessment for this dynamic drug. Transient amnesia is one of the consequences of usage and needs to be reviewed, especially in current situation of increasing legalization of use.

CASE PRESENTATION

36 years old African American male with no past history, who came with a friend to the Emergency Department, his main complaint was a sudden uncomfortable feeling of the surroundings. The patient is a receptionist in a local hotel and reported an unusual feeling of everything being strange in the environment where he works daily, a feeling that was described by him as dreaming. A friend noticed his unusual actions, and brought him to the emergency department.

At presentation, the patient was anxious and wanted to understand what happened to him. He denied any history of similar episodes, was not complaining of any fever, chills, or recent trauma, but complained of a mild headache. No recent head trauma, chest pain, palpitation or SOB reported. His friend has noticed no changes in the gait, face and extremities movements, or any signs suggestive of neurological deficits.

On examination, the patient had atraumatic, normocephalic

head, he did not remember his birthday, yet was alert, and did appear reasonable with normal cognitive functions other than memory loss; he was anxious and asking many questions related to why he is in the hospital and what is happening to him.

No Carotid bruit appreciated, he had regular rhythm, rate, with normal first and second sounds, besides having normal lung auscultation and neither extra cardiac, nor pulmonic sounds. Neurological examination revealed no focal deficits, with normal sensory and motor exam.

As a workup for his memory loss, CT scan ruled out any brain abnormalities, MRI showed no areas of diffusion restriction, the ventricles and sulci were normal, beside normal brain parenchyma, and no mass, hemorrhage, or infarction, Carotid Doppler showed no stenosis. UDS revealed THC positivity.

The patient memory started recovering after 3 hours of arriving to the ED, at the time of discharge he was able to remember recent and old memories, no specific treatment was applied in the hospital neither he was sent home on any.

DISCUSSION

Transient Global Amnesia is an acute Anterograde and retrograde memory loss, with an incidence of 23.5 to 32 in 100,000 per year in those older than 50 years (1), its occurrence is claimed to be due to migraine, ischemia, seizures, venous congestion, and psychological disturbances [2,3]. Attacks can occur during

stressful events, exertion, or with emotional circumstances, and can be related to immersion into cold water, or sexual activity [2]. An increase of the incidence of TGA is found in those with phobic personality features, a finding proposed to be due to the fact that people with such a personality tend to hyperventilate during stressful or emotional events leading to vasoconstriction that in turn affects the cerebral perfusion including memory centers [2].

Self-awareness as well as other cognitive functions than memory is usually normal in TGA, if abnormal, the disease is excluded. Even whilst in active attack some patients can drive, cook, play music, and perform other complex tasks [3]. Having said that, the patient might not be aware of the memory loss, yet will be anxious and questioning about his health; those who were alone when they had the episode, called their relatives or friends asking for help even though they did not realize their memory loss [8].

The physiological mainstay players in TGA are the neurotransmitters. For an example, the emotional events increase the release of glutamate that suppresses the functionality of the hippocampus leading to memory loss [2]. The clinical (Hodges and Warlow's) criteria to diagnose TGA have to be met [4]; those include the presence of an observer to confirm that the patient had no trauma or loss of consciousness, and to notify the admitting team of any focal deficits at the beginning such as unstable gait, facial droop as an example. The patient should have no epileptic features, neither should be actively seizing, neurological imaging should suggest no other etiology, with a recovery in the first 24 hours as one of the criteria [1].

In TGA, CT scan is uniformly normal [3]. Studies are variable as to the role of DWI findings in diagnosis; while some reports highly suggested the diagnosis with specific characteristic features, others emphasized on the presence of normal or unrelated chronic changes [9,10]. The most specific radiologic finding prescribed was a punctate hyper-intense lesion of the lateral hippocampus [2,11,12].

Overall no difference in future cognitive functions or recurrence between those with and without DWI changes [13]

Dynamic duplex shows jugular-venous valve insufficiency in high percentage of TGA patients [3]. PET- scan studies have mostly shown hypo-perfusion in the acute settings with normalization after 48 hours, yet persistence of perfusion cessation was noticed with recurrent episodes [2]. In the case of repeated episodes, EEG is needed to exclude epilepsy [2].

TGA has a low tendency to recur (2.5-5%), with a good post-episode prognosis, no increased mortality, seizures, or ischemic events. no need to treat the episode, yet will be wise to monitor the patient up to the resolution of symptoms [1].

The effect of Δ^9 - Tetrahydrocannabinol (THC) on the Central Neurological System is driven from the activation of Cannabinoid receptors (CB1) [7] that normally guest the endogenous cannabinoid agonists, including anandamide and 2-arachidonoylglycerol which are arachidonic acid derivatives. CB1 is a G protein-linked receptor found mainly in the central and peripheral nervous systems, with high concentration in the hippocampus; the main center of memory in the brain [14,15].

How does Endocannabinoids act? They create a retrograde postsynaptic-presynaptic neuron effect that is called, Depolarization-induced Suppression of Inhibition (DSI), where the Action potential that reached the end of the presynaptic neuron creates a current of Ca^{+2} , that activates the release of the neurotransmitters, as well as the endogenous endocannabinoids (2AG and anandamide), the latter will attach and react with CB1 found on presynaptic neurons.

This signal inhibits Ca^{+2} and activates K^{+} channels stabilizing the membrane and inhibiting the release of many neurotransmitters such as acetylcholine, GABA, and Glutamate [15].

In the study conducted by Block et al. memory problems were found in chronic, more frequent, marijuana addict comparing to non-users [16].

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