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Bojana Živanović[1], Ante Silić[2]
[1]General Hospital „Dr. Tomislav Bardek", Koprivnica
[2]University Psychiatric Hospital Vrapče, Zagreb

Brain atrophy and first episode psychosis in chronic cannabis use: case report

Abstract
The question of harmful effects of cannabis and its etiological relation to psychotic disorders versus its potential medical benefits has never been more present and controversial as it is today.
In our paper we present a young patient with first episode psychosis decompensation who admits heavy use of cannabis use over a long period of time on a daily basis. Through a routine differential diagnostics, CT brain scan revealed a diffuse cerebral atrophy unrelated to any specific neurological disorder.
We would like to point out probable causality of heavy cannabis use with clinical presentation and observed morphological changes.
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Key words: *cannabis, atrophy, brain, psychosis*

INTRODUCTION

Cannabis is one of the most common and worldwide used illicit drug [1]. It was used far back in the Ancient China (2727 B.C.) [2]. The question about its harmful effect on human organism compared with it is possible useful impact and place in treatment psychiatric disorders and somatic diseases has never been more controversial nowadays.
Numerus studies have been conducted in this area and their results are subject of dispute. Some of them bring theories about a positive impact of cannabis on cognitive functions [3, 4] and its place in treatment psychical disorders [5]. Much attention is being directed towards the active components of cannabis- cannabinoids such as cannabidiol (CBD), cannabigerol (CBG), and tetrahydrocannabivarin (THCV), because of their potential antipsychotic properties [5, 6]. A large number of opposed studies gives arguments for the toxic impact of cannabis on neuroanatomic structures and brings out results about the atrophy of grey and white brain matter after a long term use of cannabis [7].
There are also studies which describe an atrophy of specific brain areas abounding in numerous cannabinoid CB1 receptors such as the cingulate cortex and hippocampus [8, 9]. Among cannabis users, adolescents make the most sensitive group, in which the exposure to cannabis can cause severe and long term consequences in cognitive functions [10]. The adolescents brain is different from the adult's because of the incomplete maturation of its structures and the brains also differ in anatomy (neuronal connections and morphology) as well as neurochemistry [10]. Chronic exposure of the endocannabinoid system of adolescents to cannabis can dramatically influnce changes in brain maturation and cause long-lasting neurobiological changes that ultimately affect the cognitive function and increase the risk of developing a psychotic disorder [11–14].
The atrophy of grey and white matter is related to the frequency of cannabis use (how much and how often it is smoked) [15]. Chronic users have a significant atrophy of grey matter in brain abounding in cannabinoid receptors CB1, and it is associated with frequency of use and the numbers of the years of smoking cannabis [16].
In this case report, we present a young male patient who presents as a first psychoic episode and has confirmed brain atrophy after chronic use of cannabis.

CASE REPORT

A 19 years old male patient, unemployed. Family anamnesis does not show any records of psychiatric diseases. He was born as a younger of two children in the family of carefully monitored pregnancies.

Adres do korespondencji:
Bojana Živanović, MD, Trainee of psychiatry
General Hospital „Dr.Tomislav Bardek",
Department of psychiatry
Željka Selingera bb
Koprivnica 48000, Croatia
e-mail: zivanovic_bojana@yahoo.com
Tel.: +385919750213

Our patient started stammering at the age of three and he had been visiting a speech therapist for years. For the rest of the early and late childhood psychomotor development proceeded as normal. As a child he was very shy, having a hard time making friends. As he was finishing the elementary education, the family moved to another part of the city and it took him some time to adjust to new surroundings.

Not interested in learning, he finished the elementary school with difficulty, but in high school he adapted very well and made friends. He enrolled at a professional school. In the second grade of high school he smoked cannabis for the first time. In the beginning, it was smoked occasionally at weekends with his new friends. For the last year and a half, he smoked cannabis every day. As he admitted, he smoked three or more "joints" daily. The first psychic symptoms started about year and half before hospitalization. It began with hearing a kind of noise from time to time, but for the last six months these intensified, turning into imperative, commentary voices of religious character. He had difficulty falling asleep. To calm himself, as he was frightened and tired, he smoked more intensively, thinking that it might help him. He also started to withdraw. On the day of hospital admission, the patient came home, as his parents described, "strange and filthy", displaying disorganized behavior after being away from home for a couple of hours.

When addmited the patient was conscious, disoriented, he displayed markedly disorganized speech and behavior. His psychomotor functions were mildly enhanced, his mood dysphonic. Ductus of his speech was completely incoherent, he had dissociated judgment, distorted perception of relations around him. He presented ideas of persecution and phenomena of derealization and mind reading. No aggressive behavior or suicidal thoughts were observed upon the admission. The patient verbalized hallucinations in the form of voices that were giving him orders. Cognitive function was reduced secondarily, with low insight about his state.

In his somatic presentation we can point out the fresh bruises on dorsal side of both his fists made while punching a boxing bag repeatedly as he states in order to lower the tension.

During period of hospitalization, laboratory tests did not show any deviations (complete blood work, blood glucose, electrolytes, urea, hepatogram, creatinine, urin). The results of thyroid hormones test were within normal ranges and so were the EEG results. Neuroradiological analysis was done, in which the MSCT of the brain displayed a mild difuse cerebral atrophy (Fig. 1). "Supratentorial atrophy in frontal apical and temporoparietal, liquor spaces of cortical sulci and subarahnoidal spaces are on both sides wider and somewhat deeper, also Sylvian fissures on the both sides."

Psychological examination applied tools were: interview, RBS, BVRT, LB, MMPI, PIE, TNR, PTM. "In the intellectual function test patient scored on a level below the standard, probably compatible or somewhat reduced (psychogenic) in comparison with the primary capacity. Mild disorders of recent memory/concentration of functional type were recorded. No graphomotorical deviations. No signs of psychoorganic changes. Personality examination suggested pattern of behavior, mainly in reducing voluntary patterns and interests, unsystematic paranoid ideas (of relations, influences, values, size, the sense of being in danger), sense of the non-realistic (altered surroundings), hallucinatory sensations ('voices' of threatening, degrading and imperative character) affective dissociation (fear of people, of losing control, disintegration, solitude, emotional deprivation, suppressed aggressive energy), stereotypes in thought.

He described having low concentration for years (resembling prodromal signs of psychotic process). During the previous year he had been experiencing hallucinations and diffuse sense of being vulnerable with others (peers and strangers) which he ascribes to cannabis abuse".

Psychopharmacological therapy started with risperidone and anxiolytic, and because of persistent psychotic symptoms clozapine therapy was also introduced. Progressively the patient's psychological condition grew steady and productive symptoms reduced. His mood was stabilized, and overall behavior and speech became less disorganized. After the treatment, the patient was released in stable remission, with recommended continuation of regular psychiatric control and treatment.

DISCUSSION AND CONCLUSION

In this article we have depicted an adolescent who developed florid psychotic decompensation after a long term use of cannabis, and in whom differential diagnosis discovered a diffuse atrophy of the brain uncommon at his age. MRI examination of the patient brain was not performed so we could not compare the results, but his MSCT scan showed an evident diffuse brain atrophy. The level of evidence that we possess today undoubtedly indicates that cannabis is bad for health and its use carries significant risks.

A recent study has shown reduced volume of hippocampus and amygdala in chronic users of cannabis, but there are no alterations on orbitofrontal, anterior and paracingulate cortices nor the pituitary gland, which adds to the conclusion that chronic cannabis use has a selective impact on the morphology of the mediotemporal lobe [16]. This is just one of a great number of studies

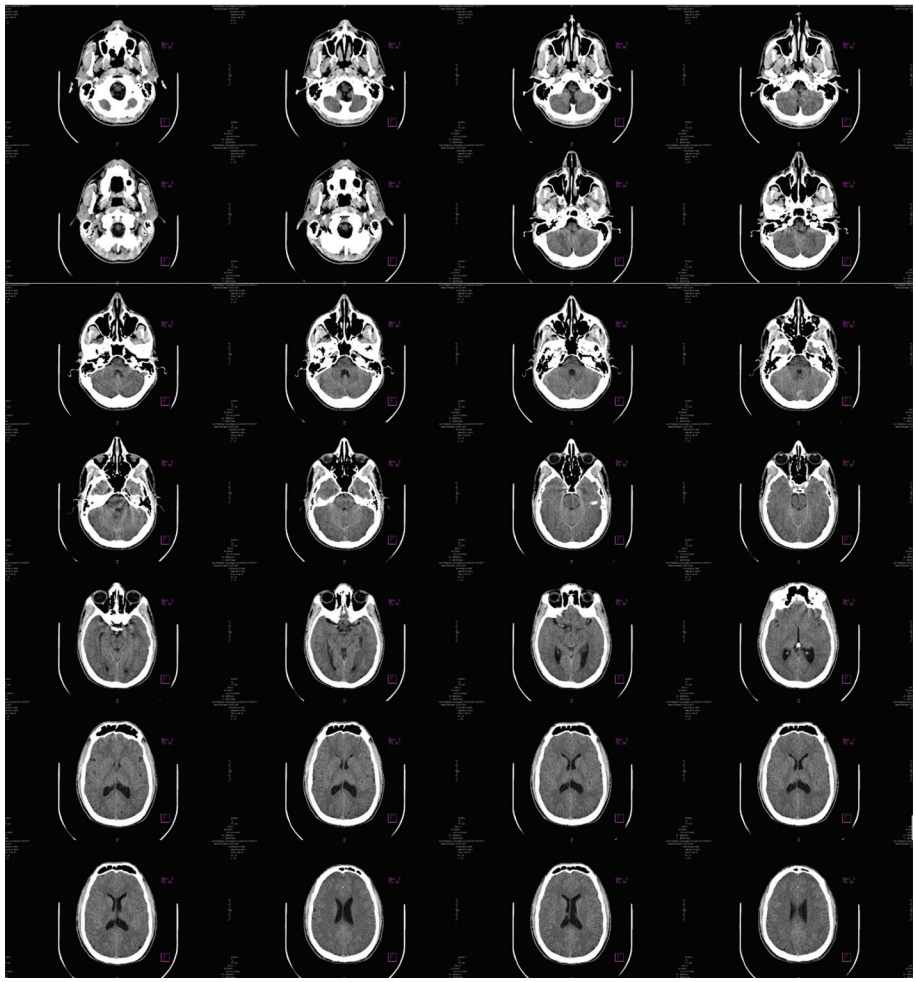


Figure 1. Patients MSCT brain results that shows mild diffuse atrophic brain changes

that found a noticeable neurotoxic effect of cannabis on certain areas of the brain, such as areas abounding in cannabinoid receptors – hippocampus [7] and cingulated cortex [8]. Contrary studies do not find evidence of bad influence of cannabis [17]. They present only subtle morphological differences in the brain between cannabis users and non-users [18].

Possible curative effects of cannabinoids (active parts of cannabis) such as cannabidiol (CBD), cannabigerol (CBG) and tetrahydrocannabivarin (THCV) have been pointed out in the treatment of psychosis because of their antipsychotic effect [5, 6].

We want to emphasize that in the case of our patient the brain of an adolescent is different from the adult brain because of the incomplete maturation of its structures, both anatomically (neuronal connectivity and morphology) and neurochemically [10]. Chronic exposure of endocannabinoid system of adolescents (in this case a year and a half of everyday cannabis consumption in large amounts), may cause dramatic changes in the brain maturation resulting in long lasting neurobiological alterations and consequent changes in cognitive functioning, with increasing risk of developing a psychotic disorder [11–14].

The relation of grey and white matter atrophy to the quantity and frequency of cannabis use has been proven [15]. In heavy cannabis smokers, grey matter of the brain is significantly reduced in the areas abounding in cannabinoid CB1 receptors [14].

No matter how controversial or dubious, the evidence and arguments presented in the above-mentioned studies cannot be denied. We must by all means be cautious in our approach and must individualize treatment of every patient.

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