Cerebral atrophy and cognitive impairment in a young patient with chronic cannabis use – a case report

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Cannabis is known to mankind for ages. It was used as a medicine, an aphrodisiac, and a euphoriant. The psychotropic and neuromodulatory effects associated with cannabis use are extensively studied topics in the recent past. The authors report a case of cognitive impairment and cerebral atrophy in a young male patient with chronic cannabis use.

Keywords:
cannabis dependence, cerebral atrophy, cognitive decline, cognitive impairment

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Introduction

Cannabis is the most commonly used illicit substance in India. Cannabis has been used in India from ancient times to overcome fatigue, worry, and for its euphoric effect. Cannabis preparations are used in three main forms – Charas (resin), Bhang (seeds and leaves), and Ganja (flowering tops). According to a study conducted by the National Drug Dependence Treatment Centre (NDDTC), New Delhi in 2019, 7.2 million Indians had consumed cannabis within the past year and, 0.25% of Indians were using cannabis in a dependency pattern (Ambekar *et al.*, 2019). Roughly 200 million people used cannabis in 2019 representing 4% of the global population and the number of cannabis users increased by 18% over the past decade (World Drug Report, 2021).

Health benefits and the harmful effects of cannabis use are a topic of controversy. There is a decreased risk perception and increased use among adolescents in the past decade (World Drug Report, 2021). Many studies have suggested significant detrimental effects on cognition in cannabis users, particularly long-term users. Here we report a patient with chronic cannabis use who presented with behavioral issues and was later found to be having significant cognitive decline and brain atrophy. He had shown improvement in cognitive functions with complete abstinence.

Case report

A 22-year male with a history of smoking cannabis in the form of joints (a rolled cannabis cigarette) from the last 5 years was brought to our hospital with complaints of irritability, aggression toward family members, decreased appetite, and disturbed sleep for 1 year. There were no symptoms of psychotic, manic, or depressive illness. There was a recent heavy intake (100 g) of cannabis. He was the class topper till his high school and secured a 90% score in intermediate examinations. During his graduation, he was introduced to cannabis by his classmates. Initially, he used to smoke one joint per day and the quantity gradually increased. He used to be absent from college and spent most of his time procuring and smoking cannabis. His use increased to five joints (equaling 50 g of cannabis) per day by the end of graduation. He was able to finish his graduation on the third attempt. He joined a private job but was removed from it due to his frequent absenteeism and poor performance at work. He used to spend most of the time in a single room smoking cannabis. The patient occasionally smoked tobacco when he could not get cannabis. He denied the use of other substances. His personal care deteriorated significantly. There was a police case filed on him after involving in a fight with his friends under cannabis intoxication. There was no history of road-traffic accidents, head injury, or seizure disorder. No history of promiscuous sexual behavior was reported by the patient. On mental-status examination, the patient was unkempt. Eye-to-eye contact was not maintained. No behavior was suggestive of hallucinations. The patient denied any delusions and no other abnormal thought content was found. On cognitive-function assessment, his attention was very

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poor, so the detailed evaluation was not done initially. The patient had very poor motivation to quit the substance and started arguing about the positive effects and the happiness he gets with cannabis use. Routine blood investigations revealed no abnormality and urine examination for drugs was positive for tetrahydrocannabinol. Viral markers for HIV, hepatitis-B virus, and coronavirus disease-2019 were negative. Computed tomography of the brain showed diffuse cerebral atrophy. The patient was admitted to our rehabilitation center. He was started on tablet olanzapine 5 mg, which was later increased to 10 mg for his aggression, insomnia, and decreased appetite. Tablet *N*-Acetylcysteine (NAC) 2400 mg in divided doses and tablet Memantine 10 mg were added later. For his craving for cigarettes, nicotine gums were started. After 3 weeks of confirmed abstinence, Addenbrookes cognitive examination (ACE-III) was performed, which showed a total score of 63 and significantly lower scores on attention, memory, verbal fluency, and language domains. The cutoff score is 82 with 84% sensitivity and 100% specificity for cognitive impairment. The patient was given cognitive remediation in the form of paper–pencil exercises for 2 months. The repeated cognitive assessment had shown improvement in the areas of attention and memory, but significant deficits in the areas of verbal and language domains. Cognitive remediation was continued. The patient stayed for a period of 6 months and got discharged recently from our center. His ACE score came as 92 at the time of discharge with significant improvement in all the domains. Complete abstinence and long-term follow-up was advised.

Discussion

Cannabinoid receptors are distributed throughout the brain and concentrated more in the prefrontal cortex and hippocampus. Their major role in the brain is to regulate the release of neurotransmitters such as serotonin, dopamine, and glutamate. These receptors increase during adolescence and any alteration of the endocannabinoid system during adolescence may result in a cascade of neuronal abnormalities, which leads to poor cognitive and emotional outcomes in adulthood (Rubino and Parolaro, 2008). Cannabis use in adolescence is associated with abnormal synaptic pruning and aberrant maturational brain changes. These abnormalities in brain-remodeling processes result in cognitive impairment and psychosis-like illness in adolescents. Abnormal pruning of synapses in the prefrontal cortex leads to impaired judgment, difficulty in social conformity, and high impulsivity (Iversen, 2003). Apart from its action on neurochemicals and genetic expression of neural development, cannabis has a direct neurotoxic effect on brain tissue (Jacobus and Taper, 2014).

Schwartz *et al.* (1989) reported short-term memory impairment persisting after 6 weeks of monitored abstinence from cannabis. Lane *et al.* (2007) found that adolescents with histories of heavy cannabis use performed worse on preservative responding and flexible thinking compared with controls with limited histories of use. Harvey *et al.* (2007) found that adolescents with cannabis use performed poorly on tests of attention and executive functioning. Although the degree of cognitive impairment in cannabis users remains inconclusive, a pattern of decline in attention, learning, and memory was described in the literature (Jacobus and Taper, 2014).

Conclusion

Cannabis use in adolescence has implications on academic functioning, social adjustment, and occupational functioning extending into later life. Healthcare professionals, teachers, and parents need to be sensitized about the ill effects of cannabis on adolescent health, particularly brain growth. Using fact-based information to raise awareness, increasing the research into harmful cannabis use, and implementing integrated and people-centered approaches will give sustainable solutions to drug abuse.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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