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Case Report

A rare cause of acute hippocampal encephalopathy ☆

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ABSTRACT

Hippocampal encephalitis (HE), or more generally limbic encephalitis, can be secondary to various etiologies. Through this case, we report a very rare toxic cause: acute hippocampal encephalopathy secondary to cannabis use in a heavy cannabis user (>20 joints/day). A 39-year-old male presented with a feverless disturbance of consciousness. The MRI revealed signal abnormalities in the hippocampal regions. Further investigations ruled out other causes of encephalitis. No renal function impairment or rhabdomyolysis was found. The objective of our study is to describe the radiological features of this severe neurological complication of cannabis

Case report

We report the case of a 39-year-old man from a low socioeconomic background, living in an urban environment. He had no significant medical or surgical history, with a history of smoking, though the extent was not quantified. He presented with a feverless disturbance of consciousness. His family reported excessive cannabis consumption, with a daily intake of over 20 joints. Clinical evaluation revealed a patient with a Glasgow Coma Scale (GCS) of 10/15, without focal neurological signs. No cardio-respiratory abnormalities were noted. Blood glucose testing showed no glycemic abnormalities. The patient was afebrile. Biological tests revealed a mild inflammatory syndrome with white blood cells at 17,000/mm³. Cerebrospinal fluid analysis was normal, with no signs of meningitis or antibodies linked to autoimmune encephalitis. Toxicological screening confirmed the presence of cannabis in the bloodstream, with no other toxic substances found. An urgent brain MRI revealed bilateral and symmetric signal abnormalities in the hippocampi on T2 and FLAIR sequences, with restricted diffusion (Figs. 1–3). No pathological contrast enhancement was observed (Fig. 4). Hippocampal volume was slightly reduced. Based on all these findings, a diagnosis of acute hippocampal encephalopathy secondary to cannabis was established. After a stay in medical intensive care and

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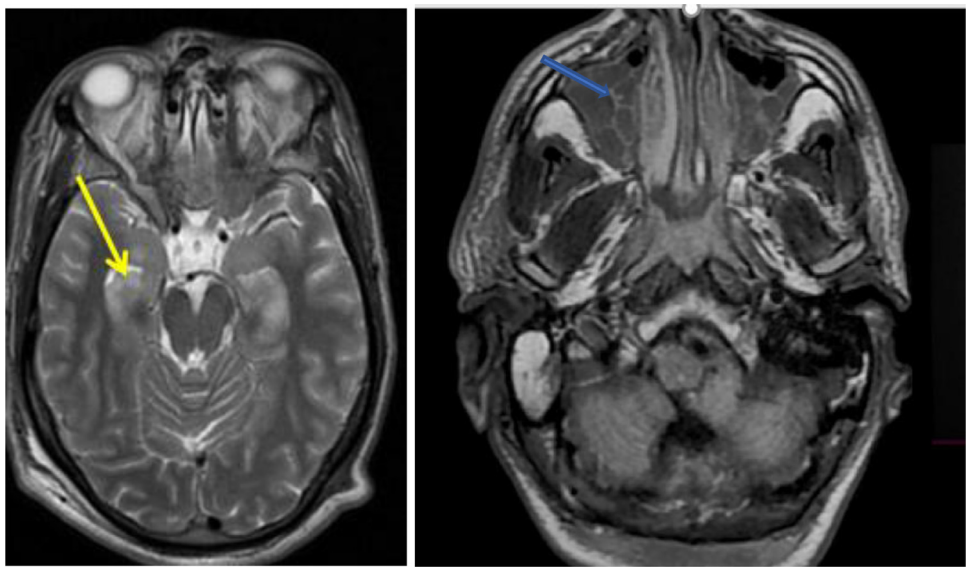

Fig. 1 – Brain MRI in axial T2 sequences showing bilateral and symmetrical T2 hyperintensity of the hippocampi. Also noted is the almost complete opacification of the maxillary and sphenoid sinuses.

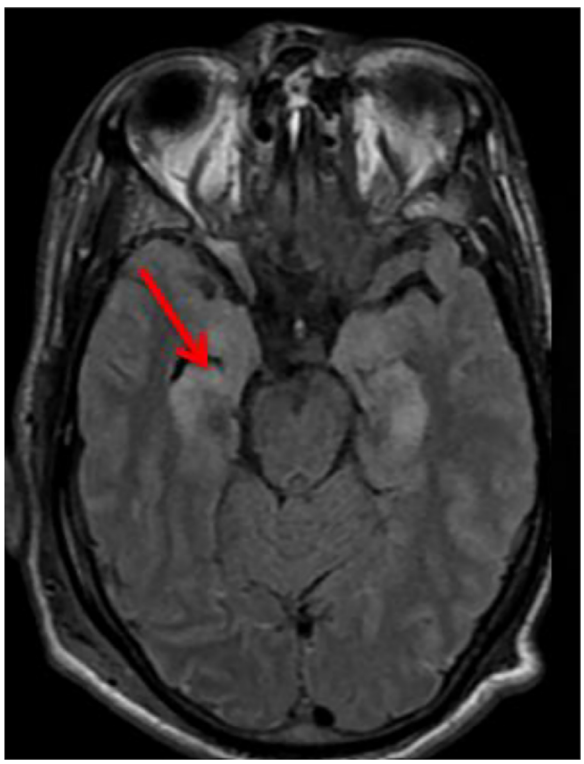

Fig. 2 – Axial FLAIR sequence showing bilateral and symmetrical FLAIR hyperintensity of the hippocampi.

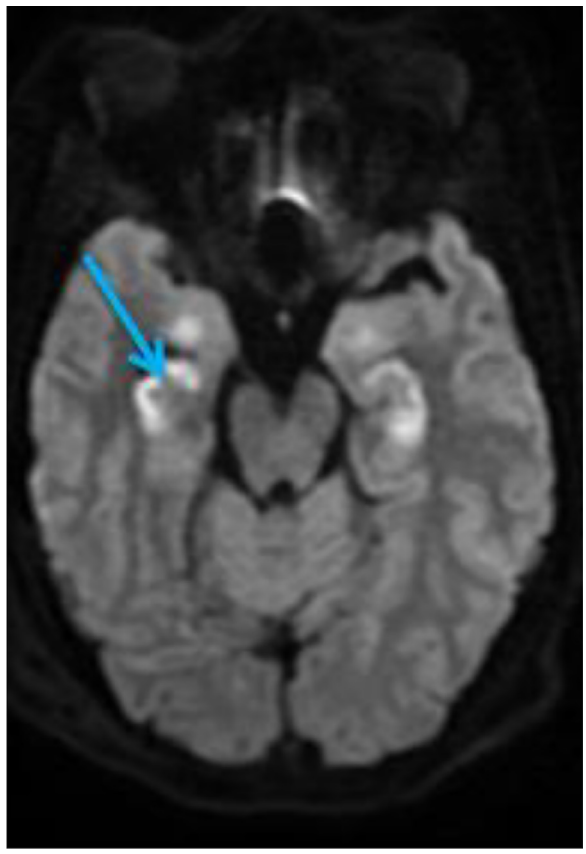

Fig. 3 – Diffusion sequence showing restriction in both hippocampi.

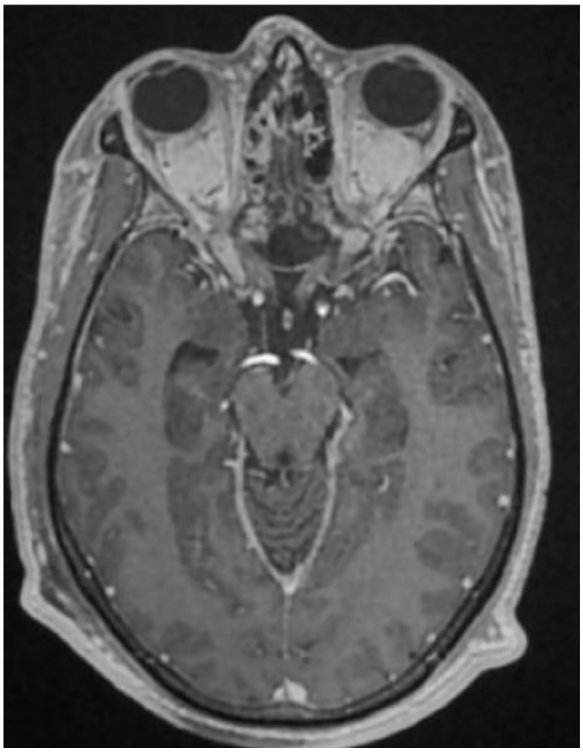

Fig. 4 – T1 MRI sequence with gadolinium injection showing the absence of contrast enhancement in the pathological area.

stabilization, the patient's condition improved, with noticeable improvement in his consciousness disturbance a few days after diagnosis.

Introduction

Acute hippocampal encephalopathy is a very rare complication of cannabis use. To our knowledge, very few cases have been described in the literature, 2 of which have been diagnosed in our hospital in recent years [1,2]. The recent surge in the number of cases can be attributed to the widespread and excessive consumption of cannabis across the world.

Discussion

The active compounds of cannabis are delta(9)-tetrahydrocannabinol (Δ-9-THC) and cannabidiol (CBD). These molecules exert central effects through the cannabinoid receptors CB1 and CB2 [1]. These receptors are particularly present in GABAergic interneurons of the hippocampus, as well as in the amygdala and the cerebral cortex. The activation of CB1 receptors by Δ-9-THC leads to the inhibition of the release of neurotransmitters, including amino acids and monoamines. The central effects of cannabinoids include disruption of psychomotor behavior, short-term memory impairment, intoxication, appetite stimulation, antinociceptive actions, and antiemetic effects [3]. Cannabidiol is primarily an anticonvulsant, whereas Δ-9-THC is proconvulsant [1]. According to studies, CBD plays a role in mitigating the harmful effects of Δ-9-THC, as well as in certain pathologies such as Alzheimer's, schizophrenia, and major depressive disorders [4].

Clinically, cannabis intoxication can manifest as cannabinoid hyperemesis syndrome, which is characterized by cyclic nausea, vomiting, and abdominal pain that is relieved by hot showers [5], Depression, anxiety, and cognitive impairments, including memory and concentration difficulties, are also associated with cannabis use [5]. In addition to neurological complications, the presence of CB1 receptors in the striated muscles of the myocardium, skeletal muscles, and kidneys may contribute to bronchiolitis and respiratory tract infections, as well as cardiac arrhythmias and blood pressure disturbances [6].

Other complications include rhabdomyolysis and acute tubular necrosis of the kidneys, frequently of multifactorial origin [2]. The inflammatory syndrome commonly associated with cannabis encephalopathy is explained by the presence of CB2 receptors on immune cells.

On magnetic resonance imaging, acute encephalitis typically presents as bilateral signal abnormalities, either symmetrical or asymmetrical, with hyperintensity on T2 and T2 FLAIR sequences, without enhancement after Gadolinium injection [1]. The same pattern is observed in our case. Chronic encephalitis, secondary to prolonged cannabis use, primarily Δ-9-THC, is characterized by hippocampal atrophy. In contrast, in cases of combined use of Δ-9-THC with CBD, the hippocampal volume remains normal [7].

The differential diagnosis is made with autoimmune and paraneoplastic limbic encephalitis [8]. also includes herpes encephalitis and metabolic encephalopathies secondary to anoxia, hypoglycemia, or carbon monoxide poisoning [9]. Autoimmune limbic encephalitis most commonly presents with bilateral and asymmetric limbic signal abnormalities, often involving the basal ganglia, in contrast to herpes encephalitis, which typically shows asymmetric involvement sparing the temporal lobes. Carbon monoxide poisoning is characterized by signal abnormalities, typically in the pallidum or in the white matter.

Confronting the anamnetic, clinical, and biological data is crucial in identifying the cause of these hippocampal signal abnormalities.

Chronic cannabis consumption manifests in various ways, taking into account the potential for genetic predisposition [10].

Currently, there are medical applications of cannabis undergoing clinical trials for the treatment of painful muscle spasms and other symptoms of multiple sclerosis. Medications based on the enhancement of endocannabinoid function could offer new therapeutic approaches in the future.

Conclusion

The medical and neurological repercussions of cannabis remain poorly understood. Given the increase in its consump-

tion, understanding the link between its use and hippocampal damage must be clearly elucidated for healthcare professionals in order to ensure early diagnosis and proper management.

Patient consent

1. Ethics approval

Our institution does not require ethical approval for reporting individual cases.

2. Informed consent

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

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