

Clinical Note

Stroke after cannabis consumption

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ABSTRACT

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Cannabis use is a widespread habit among the youth from well developed societies, however only 20 cases dealing with cannabis and stroke have been reported. Due to diagnostic difficulties linked to concomitant drug abuses and the lack of specific symptoms new cases should be studied. A 19-year-old male came to the Emergency Service reporting severe cephalgia and impairment in the left hemibody strength after cannabis use. Brain CT was normal and the MRI revealed an ischaemic infarct in the posterior cerebral artery. Cannabis was detected in the blood and in the urine analysis. After 14 days no neurologic deficits were found in the examination and the patient left the hospital. The most relevant issues in this case are: 1) clear aetiological diagnosis related to cannabis use, 2) initial migraine presentation with normal findings in the CT and 3) complete recovery.

Key Words: Cannabis. Cerebrovascular disease. Arteritis. Stroke.

RESUMEN

Ictus isquémico tras consumo de cannabis

El uso del cannabis es un hábito muy extendido entre los jóvenes de las sociedades desarrolladas, y sin embargo sólo se han documentado 20 casos de ictus por consumo de cannabis. Las dificultades diagnósticas derivadas del uso de otras drogas y de la falta de especificidad clínica justifican el estudio de nuevos casos control. Un varón de 19 años acude a Urgencias por cefalea holocraneal y pérdida de fuerza en el hemicuerpo izquierdo después del consumo de cannabis. El TAC cerebral fue normal, y la RMN cerebral evidenció un infarto isquémico en la arteria cerebral posterior. En sangre y orina se detectó cannabis. A los catorce días se le dio el alta sin déficits neurológicos. Las características más relevantes de este caso incluyen: 1) diagnóstico etiológico nítido, por el consumo exclusivo de cannabis, 2) clínica inicial de migraña acompañada con TAC cerebral normal y 3) recuperación clínica total.

Palabras clave: Cannabis. ACV. Arteritis. Infarto.

INTRODUCTION

Despite the fact that cannabis use is a widespread habit among young people from well developed societies, there is still a great deal of uncertainty regarding the psychosocial damage that it can cause¹. Recently, 48 relevant cases were studied and a clear link between cannabis consumption, a reduction in intellectual capacity and an increased addiction to other drugs were identified. However, it was finally concluded that new evidence is necessary to attribute the significant deterioration of public health to cannabis². Although it is clear that cannabis use can be beneficial in certain circumstances because of its anti-emetic effects or its therapeutic use in cases of neurological diseases, the fact of the matter is that the adverse effects

identified in the literature⁵ include depression, schizophrenia, dementia and a higher risk of cancer of the airways.

The first case of cannabis-induced neurological symptoms was published in 1964⁶ and CT scan images were not used until 1991 to identify the presence of cerebrovascular disease⁷. Since then this link between cannabis use and the neurological symptoms that affect young people has been documented⁸⁻¹⁰ and at times it includes the use of other drugs¹¹ or congenital conditions related to clotting. There are usually many symptoms in the initial examination including hemiparesis, aphasia or hemianopia and therefore the emergency department provides the first level of care for these patients¹³.

The present clinical case began with a migraine caused by a single aetiological factor which was cannabis consumption.

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CLINICAL CASE

The patient was a 19-year-old male with no relevant medical history, a non smoker who does not drink. He consumed cannabis on a daily basis and came to the emergency department complaining of left hemiplegia and preceding headache. These symptoms were triggered two hours after consuming cannabis.

He was conscious and oriented; his pupils were dilated and reactive and no vision or sensation to light on the left side. He had facial paralysis on the left side and left hemiparesis 3/5 with extensor plantar reflexes and an ataxic component. Sensation was not affected and the results of the rest of the examination including the cardiorespiratory examination were normal.

No abnormalities were detected in the blood and urine tests, the ECG or the chest x-ray. Cerebral CT did not detect any bleeding or ischaemic complications.

The patient was admitted to hospital with the diagnosis of classic migraine/ischaemic infarction with a marked improvement being observed in the motor deficit in the following 48 hours. Rehabilitation treatment began with physio and occupational therapy.

Different imaging studies were carried out 120 hours after the symptoms began. The MRI scan showed an acute ischaemic infarction in the territory of the anterior right cerebral artery. The angioMRI showed occlusion of collateral branches of the P3 segment. The transcranial and supra-aortic trunk Doppler, the bubble test and the transthoracic echocardiogram showed no abnormalities. Serology for neurotropic viruses, vitamin B12, folic acid and the test results for genetic and plas-matic thrombophilia were negative or normal.

The results of the toxicology report and the EMIT were as follows: Cannabis 0.062 µg/ml (blood), and >0.2 µg/ml (urine). No opiates, cocaine, benzodiazepine, amphetamines or methadone was detected. After two weeks the patient was discharged from hospital without neurological deficits and with antiplatelet treatment (Clopidogrel).

DISCUSSION

It is difficult to hold drug consumption responsible for being the cause of cerebrovascular disease in young patients because extensive epidemiological research¹⁴ shows that a history of drug use in 12.1% of cases and this could only be cited as a possible cause of stroke in 4.7% of cases. Cannabis is no exception to this and even though its use is widespread throughout the population, only 20 cases of cerebrovascular di-

sease have been described and the most recent two cases are from 2006 since neurological deficits were documented using brain scan images¹⁵. The discrepancy involving very low incidence rates of stroke in relation to the high level of cannabis consumption could be put down to coincidence, however the cause-effect relationship is so evident in some cases that it renders this hypothesis invalid.

The main difficulty involved in identifying a clear causal relationship between cannabis and cerebrovascular disease is the simultaneous use of other toxic substances. In fact, in the twelve cases studied in the Mousouttas revision¹⁶, there was no link to tobacco, alcohol or other drugs in only five of the cases. Furthermore, in some cases no drug tests were carried out^{7,11} even though there was significant evidence supporting the link given that the episode occurred within 15 minutes of consumption¹⁷. All authors insist on the need to study control cases in order to adequately define this link.

The initial clinical manifestation does not reveal any specific symptoms that would suggest a toxic cause. In the cases published, ischaemic infarction occurred during consumption⁸ or, more commonly, after a period of time which can vary greatly between 10 minutes and several days later¹¹. The epi-

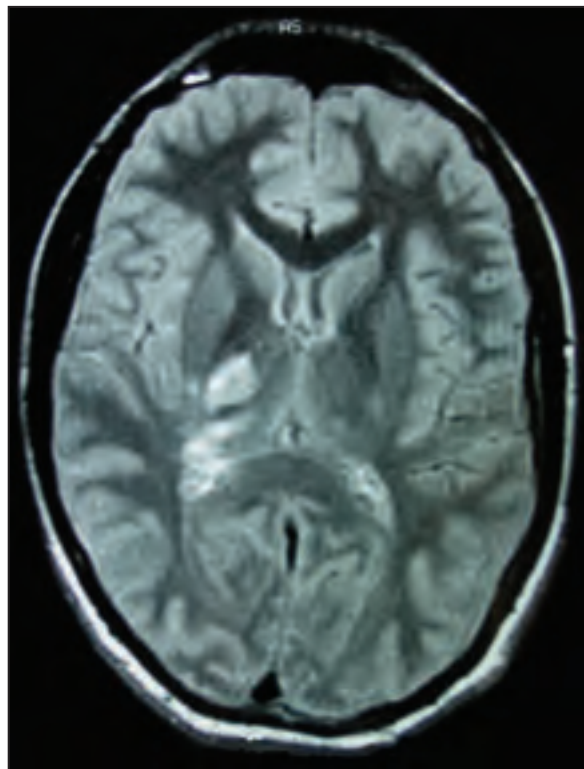


Figure 1. MRI scan: Acute ischaemic infarction in the territory of the right anterior cerebral artery (anterolateral choroidal arteries and striated thalamus) in sequence T2.

sode occur only once and in exceptional circumstances recurrent¹⁶. It has been reported in young people aged between 15¹⁰ and 34⁷ years with no relevant medical history, although transient ischaemic attacks have been recorded in half of cases⁹.

Hemiparesis is the main symptom along with hemianopia, dysarthria and aphasia which are less common^{18,19}. In this case, given the nature of the headache which was the initial symptom, the diagnosis of a classic migraine was considered, and although cephalgia is not mentioned in cerebrovascular accidents by cannabis there is still a highly contentious link with ischaemic accidents^{20,21}.

The differential diagnosis of strokes in young patients involves excluding other more common causes (cardiac embolism, atherothrombosis, non-sclerotic arterial vasculopathies, lacunar infarctions and migraines)²² in cases where there may be a link with cannabis²³, or when thrombophilia is present. Even though the role of thrombophilia is still unclear, its link with strokes caused by cannabis consumption has already been recorded¹². It is obvious that there is a temporal link between

consumption and examination and the presence of cannabis in blood/urine are key factors when diagnosing the patient. The CT scan images and the MRA scan are not specific and it seems that the Doppler can identify significant differences, for example in cases of chronic use; the brain perfusion of young consumers aged between 18 and 30 is comparable to normal subjects aged 60^{24,25}. However, this is not very useful for the diagnosis. In the case of this patient, the initial CT scan was normal and the Doppler did not record any abnormalities but the MRI identified the ischaemic infarction in the territory of the anterior cerebral artery.

Patient progress is usually good and when the patient rests¹⁸ there is full recovery, as in this case, although small deficits may remain¹⁷.

The most relevant aspects of this case include the following: 1) clear aetiological diagnosis related to cannabis use, 2) initial migraine presentation with normal findings in the CT, and 3) complete recovery.

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