

- [23] Cavellani CL, da Silva AC, Ribeiro GK, Oliveira LF, Ferraz ML, Teixeira V de P. Association between cysticercosis and neoplasia: a study based on autopsy findings. *Sci World J* 2013;29:895942.
- [24] Schmieder A, Schledzewski K, Michel J, Schönhaar K, Morias Y, Bosschaerts T, et al. The CD20 homolog Ms4a8a integrates pro- and anti-inflammatory signals in novel M2-like macrophages and is expressed in parasite infection. *Eur J Immunol* 2012;42:2971-82.

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## Both hemorrhagic and ischemic stroke following high doses of cannabis consumption



### Accident vasculaire cérébral hémorragique et ischémique après consommation de doses élevées de cannabis

Cannabis-related strokes have rarely been reported despite its widespread use. In the literature, only limited number of case reports was described to have stroke possibly related to cannabis use; yet there is only one patient described with a hemorrhagic stroke associated with cannabis use [1-3]. To best of our knowledge, here, we report the first case of both hemorrhagic and ischemic stroke following high doses of cannabis consumption.

#### Case report

A 38-year-old man was admitted to the emergency department due to right-sided hemiplegia, motor aphasia and impairment of consciousness. In his cranial computed tomography (CT), a large hematoma (70 × 40 mm) was detected in left basal ganglia. Cranial magnetic resonance imaging (MRI) showed two millimetric foci of restricted diffusion in the right cerebral

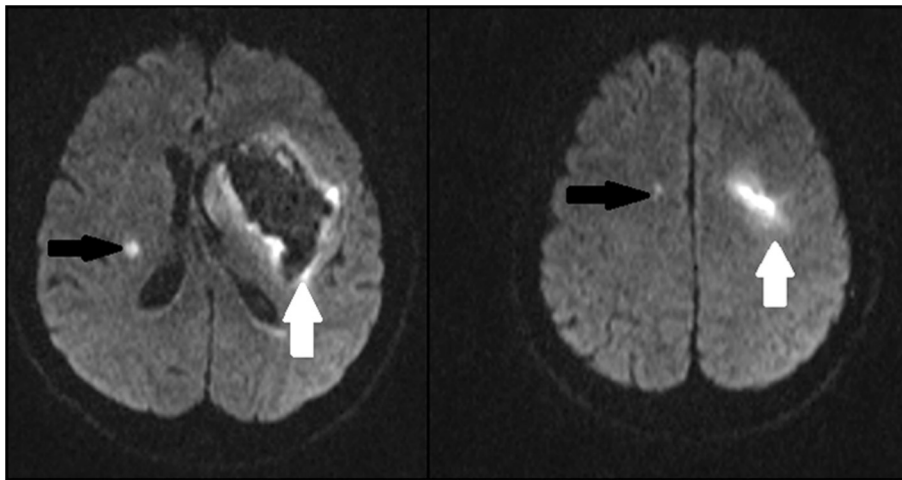
hemisphere, as well as hematoma in the left hemisphere at acute stage (figure 1). There was no other hemorrhagic lesion as cerebral microbleeds on gradient echo sequence. Glasgow Coma Scale (GCS) was 6/15 points; he was intubated and followed-up in intensive care unit. He was transported to stroke unit three weeks later; he was conscious with a GCS score of 12/15 points. The NIHSS (National Institute of Health Stroke Scale) score was 12 points.

His past medical history was unremarkable, except for frequent alcohol consumption, tobacco smoking (18 pack-years), and cannabis use. His family members stated that his cannabis consumption has increased within the last four months, and about two hours before the event, he consumed more than 4 g of cannabis via bong. His blood pressure measurements were stable within normal ranges all through his hospitalization in stroke unit. He had no past medical history of hypertension, or any other cardiac, neurological or vascular disease. Holter electrocardiography, transthoracic and transoesophageal echocardiography were normal. Detailed biochemistry tests, cholesterol profile, markers of vasculitis (ANA, anti-ds DNA, RF, anticardiolipin and antiphospholipid antibodies, antinuclear factor, antineutrophil cytoplasmic antibody), serology for human immunodeficiency virus, syphilis, and hepatitis B and C, and tests for hematological disorder (protein S/C, anti-thrombin III activity, fibrinogen, Factor V Leiden/prothrombin/MTHFR mutations) were all normal. Urine screening was strongly positive for cannabis, but negative for other stimulants as cocaine, heroin, and amphetamines.

Extracranial Doppler ultrasonography of carotid and vertebral arteries and MRI-angiography were normal. The digital subtraction angiography showed vasospasm in the left internal carotid artery. At discharge, GCS of the patient was 15/15 points with a NIHSS of 3 points. At 6th month of follow-up in outpatient stroke clinic, his neurological status was stable. However, his family members stated that he was still on cannabis use with lower doses and frequencies in compared to pre-event period. The patient did not accept to perform control cranial digital subtraction angiography.

#### Discussion

Although case reports and population-based studies have demonstrated a clear temporal relationship between stroke and cannabis use, it is still difficult to establish a causal relationship [1-3]. Most of the reported cases are younger than 50 years without any vascular risk factors; though concomitant alcohol and tobacco consumption were suggested as potential triggering factors [4,5]. The lack of lesions in the cerebrovascular structure supports the hypothesis of altered cerebral autoregulation and regional hypoperfusion in the pathogenesis of cannabis-related ischemic stroke [3-5]. An acute progressive inflammation and thrombosis in cerebral arteries, as in thromboangiitis obliterans, was also suggested in the pathogenesis of



**FIGURE 1**  
Cranial magnetic resonance imaging showing two millimetric foci of restricted diffusion in right cerebral hemisphere (black arrows) and hematoma in left hemisphere at acute stage (white arrows)

ischemic stroke [6]. Hemorrhagic stroke, on the other hand, is most often seen with sympathomimetic drugs, while cannabis-induced transient arterial hypertension in addition with the failure of cerebrovascular autoregulation may play a role in the pathogenesis of cannabis-related hemorrhagic stroke [2]. Another hypothesis in etiopathogenesis of hematoma in the left hemisphere could be hemorrhagic transformation of an undetected ischemic stroke; however, it has been showed that hemorrhagic transformation is especially associated with large, territorial cerebral infarctions of cardioembolic origin, and high blood pressure [7].

In the presented case, symptom onset following unusually high doses of cannabis exposure with the lack of other cardiovascular risk factors supports a causal association of high doses of cannabis consumption in addition to concomitant alcohol and tobacco intake. Though, analytical observational studies are warranted to establish a cause-and-effect relationship between cannabis use and stroke of hemorrhagic or ischemic origin.

**Disclosure of interest:** the authors declare that they have no conflicts of interest concerning this article.

## References

- [1] Barber PA, Pridmore HM, Krishnamurthy V, Roberts S, Spriggs DA, Carter KN, et al. Cannabis, ischemic stroke, and transient ischemic attack: a case-control study. *Stroke* 2013;44:2327–9.

- [2] Singh NN, Pan Y, Muengtaweepansa S, Geller TJ, Cruz-Flores S. Cannabis-related stroke: case series and review of literature. *J Stroke Cerebrovasc Dis* 2012;21:555–60.
- [3] Dervaux A, Laqueille X. Cannabis : usage et dépendance. *Press Med* 2012;41:1233–40.
- [4] Wolff V, Lauer V, Rouyer O, Sellal F, Meyer N, Raul S, et al. Cannabis use, ischemic stroke, and multifocal intracranial vasoconstriction: a prospective study in 48 consecutive young patients. *Stroke* 2011;42:1778–80.
- [5] Peters EN, Budney AJ, Carroll KM. Clinical correlates of co-occurring cannabis and tobacco use: a systematic review. *Addiction* 2012;107:1404–17.
- [6] Karila L, Danel T, Coscas S, Chambon JP, Reynaud M. L'artérite progressive cannabique, un sous-type clinique de la thromboangéite oblitérante? *Press Med* 2004;33:21–3.
- [7] Terruso V, D'Amelio M, Di Benedetto N, Lupo I, Saia V, Famoso G, et al. Frequency and determinants for hemorrhagic transformation of cerebral infarction. *Neuroepidemiology* 2009;33:261–5.

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