Case Report

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Internal Medicine Section

Cannabis-associated Spontaneous Bilateral Basal Ganglia Haemorrhage: A Case Report

MRIDULA SINGH[1], RASHMI MISHRA[2], AABHASHA PAROTRA[3], SIDDHARTH MAHESHWARI[4], RAJINDER K DHAMIJA[5]

ABSTRACT

Spontaneous Bilateral Basal Ganglia Haemorrhage (SBBGH) is an exceptionally rare entity, with fewer than sixty cases reported worldwide. Although cannabis use is a recognised risk factor for ischemic stroke, its association with Intracerebral Haemorrhage (ICH) remains uncommon and poorly characterised. A 23-year-old male with no vascular risk factors presented with acute onset right-sided hemiparesis, dysarthria, and dysphonia. Symptoms developed a few hours after cannabis ingestion. Neurological examination revealed right facial nerve palsy, vocal cord palsy, and reduced motor strength in the right upper and lower limbs. Non-contrast computed tomography of the brain demonstrated acute bilateral basal ganglia haematomas. Comprehensive evaluation, including autoimmune, infectious, coagulation, and genetic work-up, was unremarkable. Magnetic resonance angiography and venography excluded vascular malformations, aneurysms, and cerebral venous thrombosis. Urine toxicology was positive for cannabis, identifying it as the probable aetiological factor. This case represents a rare presentation of SBBGH associated with cannabis use. It expands the spectrum of cannabinoid-related cerebrovascular complications and highlights the importance of considering substance-related aetiologies in young patients with atypical ICH.

Keywords: Cannabinoids, Cerebrovascular disorders, Haemorrhagic stroke, Substance-related disorders, Young stroke

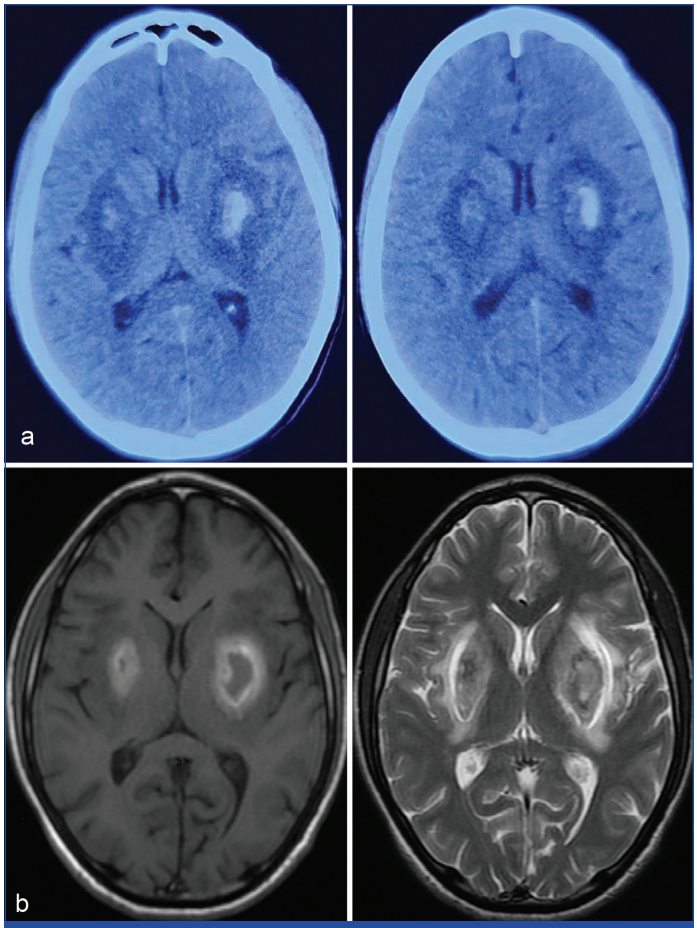
CASE REPORT

A 23-year-old right-handed male presented with sudden onset right-sided weakness, dysarthria, and dysphonia developing over 1-2 hours. He had no symptoms to suggest sensory, cerebellar or cortical involvement. There was no preceding headache, seizure, trauma, or loss of consciousness. There was no known history of hypertension, diabetes mellitus, dyslipidaemia, or cardiovascular disease. His personal and family history was unremarkable, except for regular cannabis consumption (1-2 times per month). Notably, he had ingested approximately 4-5 grams of cannabis in the form of bhang 6-8 hours prior to the onset of neurological symptoms.

On admission, the patient was alert and oriented, with a Glasgow Coma Scale (GCS) score of 15. He was normotensive, and systemic examination was unremarkable. Neurological examination revealed right-sided facial weakness consistent with seventh cranial nerve involvement, vocal cord palsy resulting in mild dysarthria, and mild right hemiparesis with Medical Research Council grade 4/5 power in both the upper and lower limbs {National Institutes of Health Stroke Scale score of 4: mild facial palsy (1), mild dysarthria (1) and drift in right upper and lower limb (1+1)}. Sensory examination and cerebellar testing were normal.

Non-contrast computed tomography of the brain demonstrated acute hyperdense haemorrhages involving the bilateral basal ganglia, with a larger lesion on the left-side measuring approximately 4.1 cubic centimetres and a smaller right-sided haemorrhage measuring 0.8 cubic centimetres. There was no intraventricular extension, mass effect, or midline shift [Table/Fig-1].

An extensive diagnostic evaluation was undertaken to identify potential secondary causes. Routine haematological and biochemical investigations, including complete blood count, renal and liver function tests, lipid profile, erythrocyte sedimentation rate, and coagulation parameters, were within normal limits. Autoimmune screening and viral serologies were negative. Evaluation for secondary hypertension, including assessment for renal, thyroid, and adrenal disorders, did not reveal any abnormalities. Electrocardiography showed normal sinus rhythm, and transthoracic echocardiography was unremarkable.



[Table/Fig-1]: a) NCCT head showing acute hyperdense bilateral basal ganglia haematomas (4.1 cc left, 0.8 cc right); b) Axial T1WI showing a central isointense core with a hyperintense rim, while T2WI showing a central iso- to hyperintense signal with hyperintense perifocal oedema, in bilateral basal ganglia, consistent with early subacute ICH. GRE shows no microbleeds.

Magnetic Resonance Imaging (MRI) of the brain with gradient echo sequences did not demonstrate cerebral microbleeds suggestive of an underlying small-vessel vasculopathy. Magnetic resonance

angiography and venography excluded intracranial aneurysms, arteriovenous malformations, diffuse atherosclerotic disease, and cerebral venous thrombosis [Table/Fig-1]. Digital subtraction angiography could not be performed due to financial constraints. Genetic testing did not reveal pathogenic variants associated with hereditary cerebral haemorrhage syndromes (APP, COL4A1/2, NOTCH3, HTRA1).

Urine toxicology screening was strongly positive for cannabis and negative for cocaine, heroin, amphetamines, and other commonly abused substances. There was absence of conventional risk factors like hypertension, while normal vascular imaging ruled out any aneurysm, arteriovenous malformations, and vasculitis. There was no history of fever at the onset, or any history of anticoagulant use which excluded infectious aetiology and coagulopathy. Given the temporal relationship between cannabis ingestion and symptom onset, a diagnosis of cannabis-associated SBBGH was considered most likely.

The patient was managed conservatively with close neurological monitoring (blood pressure monitoring, intravenous fluids and pantoprazole). There were no signs of raised ICP precluding the need for osmotherapy and seizure prophylaxis. His hospital course was uneventful, with gradual improvement in motor strength and speech over 4-5 days. The patient was discharged on day six with enrolment into substance cessation counselling session. He was followed-up after six weeks, had significant improvement in strength and was compliant with substance cessation. As the patient showed clinical improvement, no follow-up imaging was done.

DISCUSSION

Spontaneous ICH involving the basal ganglia is most commonly unilateral and typically associated with long-standing hypertension or underlying small-vessel disease. SBBGH is an exceedingly rare clinical entity, with fewer than sixty cases reported in the literature. Cannabis use is a well-documented risk factor for ischemic stroke, but its association with ICH is uncommon [1]. The aetiological spectrum of SBBGH includes hypertension, vascular malformations, infections, coagulopathies, and substance abuse. Hypertension is the commonest cause in elderly, while vascular anomalies are more relevant in the younger age group. Infectious causes like cerebral toxoplasmosis and fungal vasculitis occur in immunocompromised states, whereas coagulopathies also contribute. Though rare, substance abuse-primarily alcohol, cocaine and amphetamines-has been implicated as a trigger [1,2].

Cannabis is predominantly linked to ischemic strokes, potentially due to mechanisms such as vasoconstriction, oxidative stress, vasculopathy, and cerebral autoregulation impairment, which are attributed to its psychoactive component, delta-9-Tetrahydrocannabinol (THC). Cannabis-related ICH is less common, with a prevalence of 0.3% in descriptive studies, and mainly documented through case reports [3]. The probable pathophysiological hypotheses underlying cannabis-associated ICH are multifactorial and remain incompletely understood. Cannabis-induced cerebral vasospasm causes transient ischemia followed by reperfusion injury, which can damage the vascular endothelium, increasing the risk of vessel rupture and subsequent haemorrhage. Secondly RCVS-like activity is a proposed mechanism, characterised by transient, multifocal narrowing of cerebral arteries. This phenomenon, often observed in imaging studies post-cannabis use, may lead to abrupt changes in cerebral perfusion pressure [4-6].

Cannabinoids may interfere with platelet aggregation and thrombin activity, especially in the context of pre-existing microvascular stress. Transient arterial hypertension is a well-recognised acute effect of cannabis, possibly due to its sympathomimetic properties. Sudden elevations in systemic blood pressure overwhelm the cerebrovascular autoregulation, particularly in the small penetrating arteries of deep brain structures such as the basal ganglia [2,7,8]. Despite an extensive literature search, only a limited number of case reports exists describing ICH attributed to cannabis use. Renard D et al., reported a 34-year-old Female presenting with headache after smoking four cannabis cigarettes. Imaging demonstrated a right temporal lobe haemorrhage, while angiography revealed diffuse multifocal arterial narrowing, suggesting a vasospastic mechanism [9]. Similarly, Rose DZ et al., described a 31-year-old male who developed seizures, left homonymous hemianopsia, and lower limb paralysis after synthetic cannabis ("Spice") use. Neuroimaging showed bifrontal subarachnoid haemorrhage with associated intraparenchymal haemorrhages, and DSA demonstrated multifocal vasospasm, supporting a RCVS-like process [4].

Aydin S et al., reported a 23-year-old chronic synthetic cannabis user presenting with altered consciousness (GCS 12/15). CT revealed a large right frontal haematoma, while angiography showed multiple intracranial arterial stenoses, indicating probable cannabinoid-induced vascular injury [7]. Tandon R et al., described a 23-year-old male who consumed 5-10 g of "bhang" and subsequently developed altered sensorium, hypertonia, hyperreflexia, and diplopia. MRI demonstrated left thalamic and brainstem haemorrhage, with normal vasculitic and coagulation profiles, similar to our patient [6].

Atchaneeyasakul K et al., reported a 27-year-old male with sudden left hemiparesis following ingestion of a large quantity of raw cannabis. Imaging revealed a right basal ganglia haemorrhage with normal DSA findings, suggesting transient hypertension or autoregulatory dysfunction rather than structural vascular pathology [8]. Ince B et al., described a 38-year-old male with right hemiplegia and aphasia after cannabis consumption with alcohol intake. Imaging revealed a large left basal ganglia bleed, while DSA showed vasospasm involving the left internal carotid artery [10]. [Table/Fig-2] highlights salient features of previously reported cannabis related intracranial haemorrhage.

Author	Age of patient	Clinical features	Quantity and type of cannabis taken	CT/MRI imaging findings	Any other associated factors
Aydin S et al., [7]	23	Altered consciousness (GCS 12/15)	Chronic use of synthetic cannabis ("Spice")	Large right frontal haematoma	Multiple intracranial arterial stenoses
Rose DZ et al., [4]	31	Seizure, left homonymous hemianopsia, lower limb paralysis	Synthetic cannabis ("Spice")	Bifrontal SAH, left frontal & right parieto-occipital ICH	Vasospasm on DSA
Tandon R et al., [6]	23	Altered (GCS 7), hypertonia, hyperreflexia, diplopia	5-10 g of "bhang"	Left thalamic & brainstem bleed	Normotension, normal vasculitic & coagulation
Atchanee-yasakul K et al., [8]	27	Sudden left-sided weakness, decreased mentation	Large oral ingestion of raw cannabis	Right basal ganglia haemorrhage (32 × 24 mm)	No vascular abnormalities on DSA
Ince B et al., [10]	38	Right hemiplegia, motor aphasia and impaired consciousness (GCS 6)	4 g of cannabis via bong, smoking and alcohol	Left basal ganglia bleed (70 X 40 mm)	DSA showed vasospasm of left ICA
Renard D et al., [9]	34	Headache	4 cigarettes of cannabis	Right temporal lobe bleed	Diffuse multifocal arterial narrowing

[Table/Fig-2]: Clinical characteristics of previously reported cases of cannabis related intracranial haemorrhage.
GCS: Glasgow coma scale; SAH: Subarachnoid haemorrhage; ICH: Intracranial haemorrhage; DSA: Digital subtraction angiography

CONCLUSION(S)

In this patient, the absence of conventional risk factors, normal vascular imaging, negative infectious and coagulation work-up, and the close temporal relationship to cannabis ingestion strongly implicate it as the causative agent. To our knowledge, this represents the first documented case of SBBGH associated with cannabis use, thereby expanding the spectrum of its recognised cerebrovascular complications. This novel association highlights the importance of a thorough substance use history in patients presenting with ICH and underscores the need for further research into the haemorrhagic potential and underlying pathophysiological mechanisms of cannabis. Clinicians should maintain a high index of suspicion for cannabis-related vascular events, especially in young patients with atypical haemorrhagic presentations and no identifiable risk factors.

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PARTICULARS OF CONTRIBUTORS:

1. Assistant Professor, Department of Neurology, Institute of Human Behaviour and Allied Sciences, New Delhi, India.
2. Senior Resident, Department of Neurology, Institute of Human Behaviour and Allied Sciences, New Delhi, India.
3. Senior Resident, Department of Neurology, Institute of Human Behaviour and Allied Sciences, New Delhi, India.
4. Assistant Professor, Department of Neurology, Institute of Human Behaviour and Allied Sciences, New Delhi, India.
5. Professor, Department of Neurology, Institute of Human Behaviour and Allied Sciences, New Delhi, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Rashmi Mishra,
19/804, 8th Floor, East End Apartments, Mayur Vihar Phase 1 Extension,
New Delhi, India.
E-mail: rashmi.virgo02@gmail.com

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