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Case Report

Cannabis and Alcohol Co-Ingestion Causing Non Aneurysmal Subarachnoid Hemorrhage: A Case Report

Dhruva Sharma*, Deven Juneja, Prashant Singh, Omender Singh and Amit Goel

Institution of Critical Care Medicine, Max Super Speciality Hospital, Saket, New Delhi, India

***Corresponding Author:** Dhruva Sharma, Institution of Critical Care Medicine, Max Super Speciality Hospital, Saket, New Delhi, India.

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Abstract

Substance abuse and co-ingestion with alcohol is quite common. Consuming heavy dose could lead to toxicity and co-ingestions may lead to unusual clinical presentations. Co-ingestion of cannabis and alcohol is very common and may have varied effects, especially on neurological system. Our case report highlights an atypical neurological presentation, with non-aneurysmal subarachnoid hemorrhage, after co-ingestion of cannabis and alcohol in a patient with no other associated comorbidities.

Keywords: Subarachnoid Hemorrhage; Tobacco; Hypertension

Introduction

Subarachnoid hemorrhage (SAH) is bleeding in the space between brain and the arachnoid membrane. Its common causes include intracranial aneurysm, arteriovenous malformation, trauma, non-aneurysmal (peri-mesencephalic pattern), vasculitis. Hypertension, tobacco use, excessive alcohol use, sympathomimetic drugs, Black race, Hispanic ethnicity, and aneurysmal size > 10 millimeters (mm) are the risk factors for rupture of aneurysm. Out of all atraumatic SAH patient 15% are non aneurysmal [1].

Delta-9-tetrahydrocannabinol, which is extracted from the flower of *Cannabis indica* and *Cannabis sativa,* is a naturally occurring psychoactive compound which is commonly abused with alcohol [2].

National Household Survey of Drug Abuse (NHSDA) in India evaluated the extend of various drug abuse and found that the prevalence of intake of alcohol was 19.6%, and that of cannabis use, is 3.8% [3]. Co-ingestion is common and according to National alcohol survey among drinkers, 11.4% reported using cannabis and alcohol among where 3.9% reported using separately and 7.5% of drinkers co-ingest simultaneously [4].

Here, we present a case with unconventional clinical presentation after co-ingestion of alcohol and cannabis in toxic doses.

Case Report

A 36 year old male presented with complaints of severe headache since 3 days associated with vomiting followed by seizures which were tonic clonic in nature followed by altered sensorium. Patient was given injection lorazepam 4mg intravenously stat and loaded with antiepileptic levetiracetam 1 gm intravenously followed by 500 mg twice daily.

On admission, his vitals were: blood pressure of 138/88 mmHg, pulse rate of 92/min and nuchal rigidity was absent. Computed tomography (CT) scan brain was done in view of seizures and altered mental status which was suggestive of hyperdense acute SAH in bilateral high frontoparietal cortical sulcal spaces (Figure 1). For further evaluation, CT angiography was also done, which did not show any sign of aneurysm (Figure 2).

There was no history suggestive of any brain trauma, hypertension, infection and vasculitis. On further questioning, a history of alcohol and tetra-hydro-cannabinol (ganja) consumption was elu-

cidated. Since last 6-7 days he was having alcohol in heavy amount (>30gm /day) and smoking cannabis. On investigating for drug toxicity his urine drug assay panel was found to be positive for cannabinoids (THC) and benzodiazepine. His vasculitis panel was negative routine blood investigations were all unremarkable. His electrocardiogram and chest X-ray were also normal.

For further confirmation, digital subtraction angiography (DSA) was planned but patient refused for same as there was significant clinical improvement.

He was managed conservatively in a neurology intensive care unit (ICU) and his altered sensorium improved gradually. He was discharged from ICU after 2 days and from the hospital after day 3. On discharge, his modified Rankin score was 0 and there was no delayed cerebral ischemia on follow up.

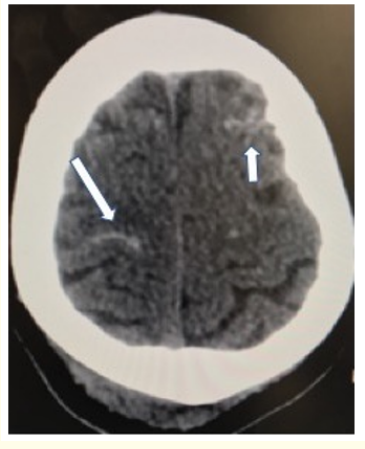

Figure 1: CT scan brain showing subarachnoid hemorrhage (marked by arrow).

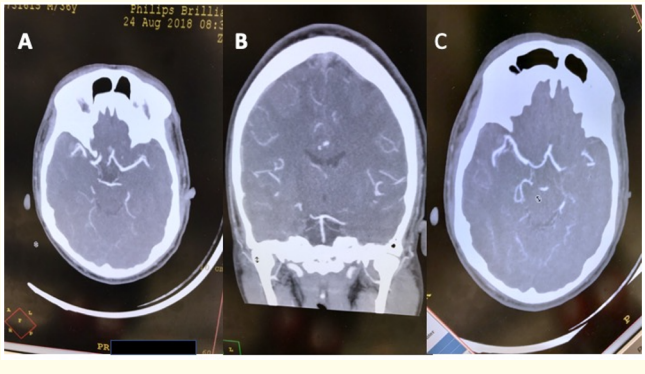

Figure 2: CT angiography (A,B,C) head.

Discussion

Alcohol abuse with co-ingestion of cannabis is common, especially among younger population [4]. Consumption in toxic dose can lead to bizzare clinical presentation and make diagnosis difficult.

Heavy alcohol drinking, more than 30 g/day, is a risk factor for the rupture of aneurysm causing SAH [5,6]. Cannabis with active ingredient tetrahydrocannabinol can have various adverse effects in toxic dose such as tachycardia, hypertension, nystagmus, slurring of speech, impaired concentration, and short term memory loss [2].

Non-aneurysmal SAH is mainly attributed to trauma, vasculitis, and peri mesencephalic pattern. In our case, there was no history of trauma, vasculitis panel was negative and bleeding in CT scan was not suggestive of peri mesencephalic pattern. Additionally, family history was negative for any such incidence.

Since CT angiography is highly accurate technique with sensitivity and specificity of 97.2% and 97.9%, respectively with modern machines and is comparable to DSA which is the gold standard [7].

Abuse of Alcohol and cannabis can cause neurological toxicity. Heavy alcohol intake is an independent risk factor for rupture of aneurysmal SAH. However in our case, co-ingestion of alcohol and cannabis, might have lead to unmediated non-aneurysmal, non mesenchymal SAH.

Subarachnoid hemorrhage causes seizures in up to 26% of patients, which occur most commonly within the first 24 hours [8] however alcohol consumption increases seizure threshold whereas withdrawal of same precipitate it while cannabis has been used as recreational drug in seizure disorder patents [9]. In contrast synthetic cannabis intake can induce seizure [10].

Our case was unique in the aspect as it involve the co-ingestion of cannabis and alcohol in toxic dose which led to direct non-aneurysmal and non-mesenchymal subarachnoid bleeding with early onset seizure.

No such studies have been done to correlate co-ingestion of substance abuse and SAH.

Conclusion

Toxic co-ingestions may lead to atypical clinical presentations and diagnostic dilemmas. Hence, it is prudent to consider drug tox-

icity in such a clinical scenario, to initiate early appropriate management and improve outcomes.

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