Letter to the Editor

We read with great interest the article by Behrouz et al.1 In light of recent movements for widespread legalization of recreational marijuana, the adverse effects of cannabis remain a pressing area of research. Behrouz et al2 present a retrospective cohort analysis of 213 patients with aneurysmal subarachnoid hemorrhage (aSAH), of whom 25.9% tested positive for cannabinoids (CB+). The authors noted that CB+ aSAH had an increased risk of delayed cerebral ischemia.

Behrouz et al1 claim to be the first to investigate the incidence of cannabis use in patients with aSAH and its impact on patient outcomes. However, we recently published a similar study2 utilizing the Nationwide Inpatient Sample where we compared the incidence of hospitalized aSAH in cannabis users versus nonusers, determined the effect of confounders on this relationship, and evaluated the effect of cannabis use on in-hospital outcomes in patients with aSAH.2 We reported a relative risk of 1.07 (95% confidence interval, 1.02–1.11) for aSAH hospitalizations comparing cannabis users versus nonusers. The relative risk in cannabis users versus nonusers was 2.13 (confidence interval, 1.95–2.34) in 24 to 35 years age group. We identified 94053 aSAH hospitalizations, with 2104 (2.24%) cannabis users. In patients with aSAH, cannabis use was associated with younger age, men, black, and Medicaid enrollment (P<0.0001). Substance abuse (amphetamine, cocaine, and tobacco) was also independently associated with aSAH hospitalization, supporting the theory that cannabis may be a gateway drug. Cannabis use was independently associated with increased likelihood of aSAH hospitalization (odds ratio, 1.18; 95% confidence interval, 1.12–1.24; P<0.0001) adjusting for various confounders.

Behrouz et al1 attributed the higher prevalence of delayed cerebral ischemia in CB+ to an increased risk of acute ischemic stroke from cannabis.3 This is consistent with our published work where we noted that CB+ patients were more susceptible to acute ischemic stroke and aSAH hospitalization than nonusers.2,4 Our result2 was consistent with the result of Behrouz et al1 with no association between cannabis use and endovascular procedures for vasospasm or in-hospital mortality.

Selection criteria are an important distinction between our study and that of Behrouz et al.1 We excluded cannabis patients coded as in remission to identify only active drug users. Interestingly, Behrouz et al1 excluded CB+ patients using other drugs from their analysis. We explored the relationship between cannabis and other drugs and found that cannabis use alone increased the likelihood of aSAH by 18%; this number increased to 28% with concomitant cannabis and tobacco use, and further increased to 38% with concomitant cannabis, tobacco, and cocaine use (P<0.0001).

The significant association between aSAH, cannabis, and delayed cerebral ischemia made by Behrouz et al1 is a notable contribution to the existing literature on illicit substances. It would be interesting to see whether this association remains significant after adjusting for other substance use (tobacco, cocaine, and amphetamines). We inform the authors that our study has provided much of the information labeled as previously unknown, and we agree that additional data are necessary to establish causality and a relationship between cannabis use and poor outcomes in patients with aSAH.

Disclosures

None.

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Letter by Reddy et al Regarding Article, "Cannabis Use and Outcomes in Patients With Aneurysmal Subarachnoid Hemorrhage"
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