For insular gliomas its important to determine preoperative seizure characteristics, tumor characteristics, surgical factors, and postoperative seizure outcomes.

Isocitrate dehydrogenase 1 and Isocitrate dehydrogenase 2 mutations (IDH1/2) have an established association with preoperative seizures in patients with grades II-IV diffuse gliomas. Here, we examined if IDH1/2 mutations are a biomarker of postoperative seizure frequency.

In a retrospective study. Patients with grades II-IV supratentorial diffuse glioma, immunohistochemistry results of IDH1-R132H, and antiepileptic drug (AED) prescribed postoperatively were included. The primary outcome was seizure frequency over the first 12 postoperative months: Group A - postoperative seizure freedom; Group B - 1-11 seizures over 12months (less than one seizure per month); and Group C - greater than one seizure per month. Rates of IDH1-R132H mutation were compared between the three outcome groups in univariate and multivariate analyses. Subgroup analysis was performed in 64 patients with IDH1/2 pyrosequencing data.

One hundred cases were included in the analysis: 30.0% grade II, 20.0% grade III, and 50.0% grade IV gliomas. Group B patients averaged 1 seizure over 12months, compared with 2 seizures per month in Group C. Isocitrate dehydrogense 1-R132H mutation was present in 29.3% (17/58) of Group A, 18.2% (14/22) of Group B, and 70.0% (14/20) of Group C patients (p=0.001). On multivariate analysis, after controlling for preoperative seizure, grade, and temporal tumor location, IDH1-R132H was associated with Group C when compared with both Group A (RR 4.75, p=0.032) and Group B (RR 9.70, p=0.012). In the subgroup with IDH1/2 molecular data, an IDH1/2 mutation was present in 64.7% (22/34) of Group A, 28.6% (4/14) of Group C, and 87.5% (14/16) of Group C patients (p=0.004).

In patients with supratentorial diffuse gliomas, IDH1-R132H mutations are associated with a more severe phenotype of postoperative epilepsy. These findings support further research into IDH mutations, and the potential for an antiepileptic therapeutic effect of their inhibitors, in patients with glioma-associated epilepsy ¹⁾.

1)

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