CALN1

To analyze the clinical significance of calneuron 1 (CALN1) expression in glioma and its role in tumor immune cell infiltration by bioinformatics. Methods The expression of CALN1 gene in glioma in the Cancer Genome Atlas (TCGA) database was analyzed by Xiantao Academic Online. Kaplan-Meier survival analysis was used to evaluate its prognostic value, and receiver operating characteristic (ROC) curve was employed to evaluate its clinical diagnostic efficiency. Gene Set Enrichment Analysis (GSEA) was adopted to identify the potential mechanism of CALN1 in glioma. The relationship between CALN1 mRNA and glioma immune cell infiltration was discussed. Results The expression of CALN1 decreased significantly in glioma, and its expression level was negatively correlated with tumor grade. Compared with the control group, the expression level of CALN1 in isocitrate dehydrogenase mutant and 1p/19g co-deletion gliomas increased significantly. Glioma patients with low expression of CALN1 had poor prognosis and significantly reduced overall survival, disease specific survival and progression-free interval. ROC curve analysis showed that CALN1 expression level had good clinical diagnostic value. The results of GSEA gene enrichment suggested that the expression level of CALN1 was negatively correlated with mitosis and neutrophil degranulation. Immunoinfiltration analysis showed that T helper type 2 (Th2) cells, macrophages and neutrophils were significantly increased in the group with low expression of CALN1. Conclusion The expression level of CALN1 in glioma is positively correlated with the prognosis. The abnormal decrease of CALN1 expression may lead to the invasion of tumor-promoting immune cells. CALN1 can be used as a potential prognostic marker and therapeutic target for glioma¹⁾.

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Zhang H, Zhang J, Wang T. [Bioinformatic analysis of the clinical significance of calneuron 1 (CALN1) in glioma and its correlation with immune cell infiltration]. Xi Bao Yu Fen Zi Mian Yi Xue Za Zhi. 2023 Mar;39(3):205-212. Chinese. PMID: 36946344.

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