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Interplay of Neuroinflammation and Epilepsy in Glioblastoma Multiforme: Mechanisms and Therapeutic Implications

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Abstract

Glioblastoma multiforme (GBM) is the most aggressive form of primary brain cancer in adults and is characterized by poor prognosis and a high incidence of seizures due to tumor-induced alterations in cerebral physiology. This review explores the complex interactions between GBM-induced neuroinflammation and epilepsy, emphasizing the mechanisms of epileptogenesis influenced by blood-brain barrier dysfunction, ion homeostasis, and neurotransmitter dynamics. We discuss the roles of pro-inflammatory mediators such as interleukin-1 β and tumor necrosis factor-alpha in exacerbating excitatory synaptic activity while inhibiting inhibitory signaling, thus creating a milieu conducive to seizure activity. Furthermore, we evaluated the efficacy of current anti-seizure medications and emerging therapeutic strategies, including the reprogramming of tumor-associated macrophages, in managing GBM-related epilepsy and tumor growth. This study aimed to elucidate the critical pathways connecting GBM and epilepsy, thereby advancing our understanding of potential interventional approaches to improve patient outcomes.

Keywords: BBB dysfunction; GBM; Ion homeostasis; Neuroinflammation; Seizure; Tumor-associated macrophages.

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