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Mini-review

From derma to nervous system: Exploring the therapeutic efficacy of azelaic acid in Neuroblastoma and Glioblastoma

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The study explores the potential of a naturally occurring phytochemical, azelaic acid (AZA) as an immunomodulator against two aggressive forms of cancer in the nervous system, such as Neuroblastoma and Glioblastoma. Neuroblastoma affects the peripheral nervous system in children below the age of five. Genetic mutations such as overexpression of gene functions like MYCN, which regulates cell growth and proliferation under normal conditions, or deletion of chromosomal subunits are demonstrated as the predominant cause of neuroblastoma. Glioblastoma, tumorigenesis in glial cells affects the central nervous system. The Isocitrate dehydrogenase (IDH1) enzyme mutation is a critical marker for detecting glioblastoma. As the brain is immunologically inert, prognosis becomes even more difficult, causing death. Available drugs fail to cross the blood-brain barrier, thus lowering the efficacy. Additionally, chemotherapy and radiation therapy come with a lot of side effects, with increased cases of relapse. Hence, the requirement of a new therapeutic option is the need of the hour. Phytochemicals are natural compounds with therapeutic properties. AZA, a dicarboxylic acid naturally found in barley, wheat, and rye, is a known compound used in skin care products and helps cure acne vulgaris. Recently, *in vitro* studies have observed that Azelaic acid exhibits anti-proliferative and immunoregulatory effects on Acute Myeloid leukemia. However, a detailed mechanistic insight into the mode of action of neuroblastoma and glioblastoma is unexplored. In this review, we intend to discuss the known antineoplastic activities of azelaic acid in cancer and thereby design a hypothesis. Furthermore, a wide range of questions on the underlying mechanisms and whether AZA plays any role in the immunostimulatory effects on a tumor microenvironment in the immunologically inert brain will also be discussed and reviewed in this paper.

Keywords: Neurotrophic factors, MYCN alteration, IDH1 mutation, tumor microenvironment, immune escape, immunomodulatory pathways