JOURNAL ARTICLE

Valproic acid in glioma: Will the anticancer issue ever be solved?

Pim B van der Meer, Johan A F Koekkoek ✉

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Extract

Radiosensitizers are agents that putatively increase the efficacy of radiotherapy, by enhancing the radiosensitivity of tumor cells. Histone deacetylase (HDAC) inhibitors, a diverse class of different pharmaceutical agents, have shown promising results as radiosensitizers in different types of solid tumors including glioblastoma. By inhibiting the removal of the acetyl group from the lysine residues of histone proteins HDAC inhibitors control accessibility of genes within the cell and may therefore induce anticancer effects (eg, cancer cell cycle arrest, differentiation, and cell death). Currently, four HDAC inhibitors (vorinostat, belinostat, panabinostat, and romidepsin) are either European Medicines Agency (EMA) and/or Food and Drug Administration (FDA) approved for treatment of T-Cell lymphoma or multiple myeloma.[1]

Valproic acid (VPA), which has also been classified as an HDAC inhibitor, is commonly prescribed as antiseizure medication (ASM) treatment in glioma patients with epilepsy. It used to be one of the preferred first-line treatment in many neuro-oncology institutions a decade ago, but has nowadays largely been replaced by levetiracetam (LEV). Rationale for VPA in glioma patients with epilepsy is based on its well-known characteristics as a broad spectrum ASM with good efficacy and tolerability in (non-)brain tumor-related epilepsy.[2] Due to its wide prescription as ASM, the general lack of new effective treatment modalities in glioma patients, and a growing list

of studies showing a beneficial effect of VPA on overall survival, VPA has gained considerable interest in the neuro-oncology scientific community as a potential therapeutic agent to prolong overall survival.

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