Steroid hormones help control metabolism, inflammation, immune functions, salt and water balance, development of sexual characteristics, and the ability to withstand illness and injury. The term steroid describes both hormones produced by the body and artificially produced medications that duplicate the action for the naturally occurring steroids.

Patients with pituitary neuroendocrine tumors usually receive “stress dose” steroids in the peri-operative periods.

Though randomized controlled trials(RCT) have not been performed to assess the necessity of steroid coverage, there are several studies that explained the changes of adrenal function during peri-operative peroids.

Steroids are very commonly administered concurrently with temozolomide and radiotherapy after the initial surgical resection of glioblastoma (GBM) to control neurological morbidity. Although the potent anti-inflammatory effect of steroids is well documented to ameliorate vasogenic edema in these tumors, the deleterious effects of steroids on the efficacy of alkylating agents or radiotherapy have been a matter of debate [1) 2) 3) 4)].

Pitter et al., performed a retrospective analysis of glioblastoma patient cohorts to determine the prognostic role of steroid administration. A disease-relevant mouse model of glioblastoma was used to characterize the effects of dexamethasone on tumour cell proliferation and death, and to identify gene signatures associated with these effects. A murine anti-VEGFA antibody was used in parallel as an alternative for oedema control.

They applied the dexamethasone-induced gene signature to The Cancer Genome Atlas glioblastoma dataset to explore the association of dexamethasone exposure with outcome. Mouse experiments were used to validate the effects of dexamethasone on survival in vivo Retrospective clinical analyses identified corticosteroid use during radiotherapy as an independent indicator of shorter survival in three independent patient cohorts. A dexamethasone-associated gene expression signature correlated with shorter survival in The Cancer Genome Atlas patient dataset. In glioma-bearing mice, dexamethasone pretreatment decreased tumour cell proliferation without affecting tumour cell viability, but reduced survival when combined with radiotherapy. Conversely, anti-VEGFA antibody decreased proliferation and increased tumour cell death, but did not affect survival when combined with radiotherapy. Clinical and mouse experimental data suggest that corticosteroids may decrease the effectiveness of treatment and shorten survival in glioblastoma. Dexamethasone-induced anti-proliferative effects may confer protection from radiotherapy- and chemotherapy-induced genotoxic stress. This study highlights the importance of identifying alternative agents such as vascular endothelial growth factor antagonists for managing oedema in glioblastoma patients. Beyond the established adverse effect profile of protracted corticosteroid use, this analysis substantiates the request for prudent and restricted use of corticosteroids in glioblastoma [5)].

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