Glucocorticoid

In neurosurgery practice glucocorticoids are commonly used.

Glucocorticoids (GCs) are a class of steroid hormones that bind to the glucocorticoid receptor (GR), which is present in almost every vertebrate animal cell. The name glucocorticoid (glucose + cortex + steroid) derives from its role in the regulation of the metabolism of glucose, its synthesis in the adrenal cortex, and its steroidal structure.

A less common synonym is glucocorticosteroid.

Glucocorticoid replacement therapy is a medical treatment used to replace or supplement the body's natural production of glucocorticoid hormones, particularly cortisol. This therapy is typically prescribed for individuals with adrenal insufficiency, a condition characterized by inadequate production of cortisol by the adrenal glands.

Adrenal insufficiency can result from various causes, including autoimmune diseases such as Addison's disease, congenital adrenal hyperplasia, adrenal gland disorders, or as a side effect of long-term corticosteroid use for other medical conditions.

Glucocorticoid replacement therapy aims to restore cortisol levels to normal, helping to alleviate symptoms and prevent complications associated with adrenal insufficiency. The dose and regimen of glucocorticoid replacement therapy are tailored to the individual's needs based on factors such as the underlying cause of adrenal insufficiency, the severity of symptoms, and any other medical conditions they may have.

Common glucocorticoid medications used for replacement therapy include hydrocortisone (cortisol), prednisone, and dexamethasone. The goal of treatment is to mimic the body's natural cortisol rhythm, with higher doses taken in the morning to coincide with the body's peak cortisol production and lower doses in the afternoon and evening.

Regular monitoring of cortisol levels and clinical symptoms is essential to adjust the dose of glucocorticoid replacement therapy as needed and to prevent over- or under-replacement. Adherence to the prescribed treatment regimen and close communication with healthcare providers are crucial for the effective management of adrenal insufficiency with glucocorticoid replacement therapy.

The administration of glucocorticoids (GCs) after traumatic brain injury (TBI) is controversial. Clinical evidence reveals the deleterious effects of GCs, but the mechanism remains unclear. Previous studies indicate that GCs impair wound healing by affecting endothelial progenitor cell (EPC) function and inhibiting angiogenesis after skin injury. Thus, we hypothesize that the central deleterious effect of GCs is associated with reduced EPCs and angiogenesis after TBI. Using a controlled cortical impact model, we examined the dynamic changes in circulating EPCs and in the regional microcirculation within 14 days of TBI by flow cytometry analysis and contrast-enhanced ultrasound, respectively. The modified neurological severity score (mNSS) and Morris water maze assay were used to assess neurological recovery. Angiogenesis and hippocampal neuron counts were assessed using immunohistochemistry analysis and hematoxylin and eosin staining 14 days after TBI. Compared with

the TBI control group, dexamethasone treatment significantly reduced the number of circulating EPCs on days 1, 3, 7 and 14 (P<0.05);decreased the number of CD31+ cells, the peak intensity and the number of hippocampal neurons on day 14 (P<0.05);increased the latency on days 12 and 13 (P<0.05);and reduced the percentage of time spent in the goal quadrant (P<0.05) on day 14. Similarly, dexamethasone increased the mNSS on days 7 and 14 (P<0.05). A strong correlation was observed between these results at 14 days after TBI (r=0.815-0.892, P<0.05). These data indicate that DEX inhibits the mobilization of EPC levels and angiogenesis around the lesion after TBI, which may contribute to neuronal cell loss and impaired neurofunction [1].

A study demonstrates a significant decrease in the rate of scoliosis surgery for Duchenne muscular dystrophy (DMD) from 2001 to 2012. It appears that the decline in surgical treatment could be related to the publication and landmark study demonstrating decreased progression of scoliosis with glucocorticoid treatment [2].

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Zhang B, Zhu X, Wang L, Hao S, Xu X, Niu F, He W, Liu B. Dexamethasone Impairs Neurofunctional Recovery in Rats Following Traumatic Brain Injury by Reducing Circulating Endothelial Progenitor Cells and Angiogenesis. Brain Res. 2019 Sep 18:146469. doi: 10.1016/j.brainres.2019.146469. [Epub ahead of print] PubMed PMID: 31541641.

2)
Raudenbush BL, Thirukumaran CP, Li Y, Sanders JO, Rubery PT, Mesfin A. Impact of a Comparative Study on the Management of Scoliosis in Duchenne Muscular Dystrophy: Are Corticosteroids Decreasing the Rate of Scoliosis Surgery in the United States? Spine (Phila Pa 1976). 2016 Sep;41(17):E1030-8. doi: 10.1097/BRS.0000000000001534. PubMed PMID: 26926354.