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Drugs and its Pharmacokinetics of Adrenal Hormones

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Opinion Article

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DESCRIPTION

Glucocorticosteroids

Glucocorticosteroids influence carbohydrate and protein metabolism, and play a vital role in the response to stress. In order to facilitate their transportation to the liver, glucocorticosteroids increase the mobilisation of amino acids from skeletal muscle, bone and skin where they are converted into glucose (gluconeogenesis) and stored as glycogen. Fat mobilization by catecholamines is potentiated by glucocorticosteroids. The major therapeutic uses of the glucocorticosteroids exploit their powerful anti-inflammatory and immunosuppressive properties. They reduce circulating eosinophils, basophils and T-lymphocytes, while increasing neutrophils. Applied topically to skin or mucous membranes, potent steroids can cause local vasoconstriction and massive doses administered systemically can cause hypertension due to generalized vasoconstriction.

Mechanism of action: Glucocorticosteroids combine with a cytoplasmic glucocorticosteroid receptor causing its dissociation from a phosphorylated heat shock protein complex. The receptor–glucocorticosteroid complex translocate to the nucleus, where it binds to Glucocorticosteroid Response Elements (GREs) in DNA and acts as a transcription factor. This increases the transcription of various signal transduction proteins. In addition, the glucocorticosteroid receptor interacts with both NF-κB and AP-1 and inhibits them from enhancing transcription of many pro-inflammatory proteins. Thus glucocorticosteroids produce a delayed but profound anti-inflammatory effect.

Adverse effects: Cushingoid physical appearance, impaired resistance to infection, salt and water retention, hypokalemia, hypertension, hyperglycemia, osteoporosis.

Drugs of glucocorticosteroids

Hydrocortisone: Hydrocortisone has predominantly glucocorticoid effects, but also has significant mineralocorticoid activity. Hydrocortisone is quickly absorbed from the gastrointestinal system, but due to varying presystolic metabolism, there is significant variation in bioavailability. It is metabolized in the liver (by CYP3A) and other tissues

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to tetrahydrometabolites that are conjugated with glucuronide before being excreted in the urine. The plasma $t_{1/2}$ is approximately 90 minutes, but the biological $t_{1/2}$ is longer (six to eight hours).

Prednisolone: Prednisolone is an analogue of hydrocortisone that is approximately four times more potent than the natural hormone with regard to anti-inflammatory metabolic actions, and involution of lymphoid tissue, but slightly less active as a mineralocorticoid. The anti-inflammatory effect of prednisolone can improve inflammatory symptoms of connective tissue and vasculitic diseases, but whether this benefits the underlying course of the disease is often unclear. Treatment must therefore be re-evaluated regularly and if long-term use is deemed essential, the dose reduced to the lowest effective maintenance dose. Alternate-day dosing produces less suppression of the pituitary–adrenal axis, but not all diseases are adequately treated in this way (e.g. giant cell arteritis).

Mineralocorticoids

Mineralocorticoids mimic aldosterone's effects on the distal nephron, causing sodium retention and potassium excretion. The synthetic mineralocorticoid fludrocortisone is effective orally. Fludrocortisone is used when mineralocorticoid replacement is needed in patients with adrenal insufficiency. Occasionally, fludrocortisone is used to treat severe postural hypotension. Mineralocorticoid antagonists (e.g. spironolactone) are used to treat mineralocorticoid excess (e.g. Conn's syndrome).

Drugs of mineralocorticoids

Aldosterone: Aldosterone is the main mineralocorticoid secreted by the zona glomerulosa of the adrenal cortex. It has no glucocorticoid activity, but is about 1000 times more active than hydrocortisone as a mineralocorticoid. The main factors that control its release are plasma sodium, plasma potassium and angiotensin- II. Pituitary failure, which results in a total absence of ACTH and of cortisol secretion, allows aldosterone production to continue.

Aldosterone acts on the distal nephron, promoting Na^+/K^+ exchange, causing sodium retention and urinary loss of potassium and hydrogen ions. Primary hyperaldosteronism (Conn's syndrome) is due to either a tumour or hyperplasia of the zona glomerulosa of the adrenal cortex. Clinical features include nocturia, hypokalaemia, hypomagnesaemia, weakness, tetany, and hypertension and sodium retention.

Fludrocortisone: Fludrocortisone (9-α-fluorohydrocortisone) is a potent synthetic mineralocorticoid, being approximately 500 times more powerful than hydrocortisone. It binds to the mineralocorticoid steroid receptor and mimics the action of aldosterone. It undergoes significant presystolic metabolism, but unlike aldosterone is active by mouth. It is used as replacement therapy in patients with adrenocortical insufficiency. Although sometimes it causes symptoms of Conn's syndrome, it can also be used to treat people with symptomatic postural hypotension.

CONCLUSION

The endocrine glands known as the adrenal glands (sometimes referred to as suprarenal glands) generate a number of hormones, including adrenaline and the steroids cortisol and aldosterone. Over the kidneys, they are located. Each gland has an inner medulla and an outer cortex that makes steroid hormones. The zona glomerulosa, the zona fasciculata, and the zona reticularis are the three primary zones that make up the adrenal cortex.