Dexamethasone suppression test

The dexamethasone suppression test (DST) is used to assess adrenal gland function by measuring how cortisol levels change in response to an injection of dexamethasone. It is typically used to diagnose Cushing's syndrome.

High Dose Dexamethasone suppression test (HDDST) has low sensitivity (65%) and specificity (60%) in predicting Cushing's disease [1)].

The DST was historically used for diagnosing depression, but by 1988 it was considered to be “at best, severely limited in its clinical ability” for this purpose.

Dexamethasone is an exogenous steroid that provides negative feedback to the pituitary gland to suppress the secretion of adrenocorticotropic hormone (ACTH). Specifically, dexamethasone binds to glucocorticoid receptors in the anterior pituitary gland, which lie outside the blood–brain barrier, resulting in regulatory modulation.

Interpretation

Low-dose and high-dose variations of the test exist.

The test is given at low (usually 1–2 mg) and high (8 mg) doses of dexamethasone, and the levels of cortisol are measured to obtain the results.

A low dose of dexamethasone suppresses cortisol in individuals with no pathology in endogenous cortisol production. A high dose of dexamethasone exerts negative feedback on pituitary ACTH-producing cells, but not on ectopic ACTH-producing cells or adrenal adenoma.

Dose

A normal result is a decrease in cortisol levels upon administration of low-dose dexamethasone. Results indicative of Cushing's disease involve no change in cortisol on low-dose dexamethasone, but inhibition of cortisol on high-dose dexamethasone. If the cortisol levels are unchanged by low- and high-dose dexamethasone, then other causes of Cushing's syndrome must be considered with further work-up necessary. After the high-dose dexamethasone, it may be possible to make further interpretations.

1)
Aron DC, Raff H, Findling JW. Effectiveness versus efficacy: The limited value in clinical practice of high dose dexamethasone suppression testing in the differential diagnosis of adrenocorticotropin-dependent Cushing's syndrome. J Clin Endocrinol Metab. 1997;82:1780–5.