ug/dL (500 nmol/L) or greater (1). Patients with subnormal cortisol levels remain on glucocorticoid until retested in 3-6 months. The goal of this study was to determine whether a baseline cortisol value predicts a normal response to the ACTH stimulation test.

Methods: We reviewed 235 ACTH stimulation (stim) tests conducted on 76 patients with secondary adrenal insufficiency following remission of CS. Patients had resection of a single adrenal gland (n=7), pituitary adenoma [with (n=3) or without (n = 47) subsequent radiation], 70% of pituitary tissue (n=5), or ACTH secreting intrathoracic tumor (n=13). One had an ectopic ACTH secreting tumor in spontaneous remission (n=1). ACTH stim tests were conducted between 0800h and 0900h, 24 hours after the last dose of glucocorticoid, using 250 mcg of cosyntropin intravenously. Cortisol levels were measured just before administration of cosyntropin, and 30 and 60 minutes afterwards. Patients were considered to have passed the test if baseline or peak cortisol values reached > 18mcg/dL. Baseline cortisol values were compared to the 'pass' rate.

Results: Baseline F values (ug/dL) and passing rates (# pass/total) were:

<4: 1/91;
4-4.9: 2/27;
5-5.9: 8/31;
6-6.9: 2/21;
7-7.9: 7/25;
8-8.9: 4/12;
9-9.9: 8/12;
>10 - < 15: 6/11
15 – 19.5: 5/5

Thus, Am cortisol values >9 ug/dl were significantly more likely to predict a normal response to ACTH stim than lower values (p<0.0001). ACTH values (n=184) did not predict peak F levels. However, no patient with ACTH value <5 pg/ml passed the test; all had peak F values of 0-10.5.

Conclusion: Baseline cortisol can be a guide as to whether the more costly stimulation test is needed. In the small cohort with baseline 0800h – 0900h cortisol >15 ug/dL, all passed the test, suggesting that it is not needed in such patients. We recommend use of an ACTH stimulation test to assess recovery of the HPA axis when a morning cortisol reaches 9 mcg/dL, with an expected pass rate of about 66%.

Reference: 1. Nieman LK, Biller BM, Findling JW, Murad MH, Newell-Price J, Savage MO et al. Treatment of Cushing's Syndrome: An Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab 100:2807, 2015.

Steroid Hormones and Receptors

STEROID BIOLOGY AND ACTION

HSD3B1 Expression in the Human Immune System

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3β-hydroxysteroid dehydrogenase-1 (3βHSD1), catalyzing conversion of dehydroepiandrosterone (DHEA) to Δ^4-androstenedione, is an essential enzyme in the pathway toward production of biologically active androgens such as dihydrotestosterone from the adrenally produced precursor DHEA sulfate, the most predominant steroid hormone in circulation. We previously identified, in the gene (*HSD3B1*) that encodes 3βHSD1, a germline gain-of-function missense-encoding variant that has now been validated in several studies as predicting more rapid progression in prostate cancer patients treated with gonadal testosterone deprivation. Production of androgens from adrenal precursors is important not just in the context of prostate cancer but in other physiologic and pathophysiologic processes, which could include asthma. Androgens are associated with better lung function in both asthma and healthy cohorts, and increasing circulating androgen levels in males help explain the switchover in asthma being more common in boys than girls but then more common in women than men. A main treatment for asthma, as well as other inflammatory processes, is administration of glucocorticoids, yet unresponsiveness to glucocorticoids in a subset of patients remains a major problem. Systemic glucocorticoid administration suppresses adrenally produced DHEA and DHEA-S, suggesting a depleted pool for biologically active androgen production as a mechanism for glucocorticoid resistance. Our surprising preliminary data support a link between glucocorticoid responsiveness and the more active *HSD3B1* allele: patients homozygous for the adrenal-permissive *HSD3B1*(1245C) allele exhibit better response to oral glucocorticoids than those homozygous for the adrenal-restrictive *HSD3B1*(1245A), with heterozygous patients falling in the middle. This suggests a model in which patients with the more active (adrenal-permissive) form of 3βHSD1 produce sufficient androgens despite the depleted pool of precursor hormones whereas patients with the less active (adrenal-restrictive) form cannot. To further elucidate the link between 3βHSD1 activity and immune response, we assayed *HSD3B1* expression in different types of white blood cells. Leukocyte subsets from asthma patients and healthy controls were purified using fluorescence-activated cell sorting, and *HSD3B1* expression was analyzed using qPCR. White blood cells of several types expressed *HSD3B1* at levels comparable to or greater than both prostate cancer and placental choriocarcinoma cell lines with very robust 3βHSD1 activity. Further determining the cell type
specific expression and activity of this key enzyme is an important step in unraveling the link between the *HSD3B1* polymorphism and asthma along with potentially many other immune processes.

Adrenal

ADRENAL CASE REPORTS I

Pheochromocytoma Presenting Rare Skin Findings: Livedoid Vasculopathy and Raynaud's Phenomenon.

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