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%.

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Steroid Hormones and Receptors

STEROID BIOLOGY AND ACTION

HSD3B1 Expression in the Human Immune System

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SAT-734

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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SAT-232

Background: Oversecreted catecholamines from pheochromocytoma (PCC) is responsible for a large variety of clinical signs and symptoms, including skin lesions, due to their effects on hemodynamics and metabolism, as well as the putative effects on the blood coagulation system. Livedo reticularis had been reported as the skin complications of PCC, but there was no pathological evidence. Livedoid vasculopathy, a subtype of livedo reticularis is a noninflammatory thrombotic condition. Here we firstly showed that livedo reticularis accompanying PCC.

Case: A 36-year-old female with hypertension, hyperglycemia, and right adrenal mass had referred to our hospital. She had violaceous reticular lesions on bilateral lower limbs, a three-year history of recurrent ulcers around her right ankle with severe pain. She also had frequent Raynaud's phenomenon. On admission, the blood examination showed that her hemoglobin concentration was 16.3g/dl and her platelet count was 408,000/ml. Plasma catecholamines and the metabolite levels were elevated. The abdominal CT scan showed the right adrenal mass, approximately 6.5cm in diameter, and MRI showed a heterogeneous mass with high signal intensity on T2-weighted images. MIBG scintigraphy showed uptake in the mass. We diagnosed her as PCC. A skin biopsy revealed occlusions of the lumen of small vessels in the mid dermis without vasculitis. Administration of doxazosin revealed the improvement of the clinical parameters including the pain and the ulcer of the right ankle. The laparoscopic right adrenalectomy was performed. Histopathological study of the adrenal tumor was consistent with PCC. Genetic testing and MLPA analysis for *RET, VHL, SDHx, SDH-AF2, MAX*, and *TMEM127* showed no mutations or copy number alterations. Postoperatively, hypertension and hyperglycemia improved, and Raynaud's phenomenon and reticular macules disappeared.

Discussion: Among the various clinical signs of PCC, skin disorders are relatively rare. Limited reports showed that the livedo reticularis was a complication of PCC, but its mechanism had not been elucidated. Livedoid vasculopathy is a rare cutaneous disease manifesting as recurrent ulcers on the lower extremities, which is a noninflammatory thrombotic condition associated with hypercoagulation. The catecholamine excess could induce arterial vasospasm in addition to relative hypercoagulation tendency, which could result in livedoid vasculopathy.

Conclusion: The present study showed first pathological evidence of that the livedo reticularis accompanying PCC was livedoid vasculopathy.

Thyroid

HPT-AXIS AND THYROID HORMONE ACTION

Long-Term Effect of Neutering on Plasma Luteinizing Hormone Concentrations in Cats: A Potential Role in the Pathogenesis of Feline Hyperthyroidism

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SAT-444

Abstract: Feline hyperthyroidism remains a major area of interest within the veterinary field given its high prevalence, affecting nearly 10% of geriatric cats, and that causal factors leading to this disease are not completely understood.[1,2] Feline hyperthyroidism shares clinical and histopathological similarities with Toxic Multinodular Goitre in humans and therefore the discovery of driving cellular mechanisms behind the pathogenesis of this feline disease may bring translational insight into the pathogenesis of the later.[3,4] Gonadotropin hormones such as LH are structurally related to other glycoproteins including TSH and cross-reactivity between these and their receptors has been demonstrated.[5] It is hypothesized that increased concentrations of gonadotropins in neutered cats might be implicated in the pathogenesis of hyperthyroidism. This study aimed to determine the long-term effect of neutering on plasma LH concentrations in cats. Stored plasma samples from client-owned cats were used for measurement of LH and TSH concentrations. Clinical data, including age, sex, neutering age and medical history were reviewed. Two study populations were included in this study: (1) a geriatric cat population (≥ 9 years old): 18 entire and 18 neutered cats matched for age, sex and date of sample collection; (2) an adult cat population (2-8 years old): 45 neutered cats. LH concentrations were measured using a feline ELISA and TSH concentrations were measured by the Immulite canine TSH assay. Geriatric neutered cats have higher plasma LH concentrations (median, 0.25 ng/ml [25th percentile, 0.25; 75th percentile, 2.1]) than age matched entire cats (0.25 ng/ml [0.25, 0.25], P < 0 .001). Cats neutered between 6-9 months of age have higher LH concentrations than cats neutered before or after that period (P = 0.004). No correlation was found between plasma LH and TSH concentrations (P = 0.422). In conclusion, neutering causes significant long-term increase in LH concentrations in cats. Further research to determine whether this results in activation of the TSH receptor and ultimately in thyrocyte hyperplasia and/or hyperfunction is warranted.

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Diabetes Mellitus and Glucose Metabolism

GESTATIONAL DIABETES, DIABETES IN PREGNANCY, AND IN UTERO EXPOSURES

The Impact of Vitamin D Deficiency on Pregnancy in Type 1 Diabetes

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