cortisol/DHEA-S ratio were statistically lower in patients [12.6 µg/dl (4.5, 3.5-24.5) and 5.3 (3.6, 1.3-19.5), respectively] compared to controls [15.5 µg/dl (4.9, 4.2-30.1) and 8.0 (4.7, 1.1-25.5), respectively] (p=0.007 and 0.001, respectively). Sub-analysis, revealed that in men, serum DHEA-S was similar between patients and controls [303.7 µg/dl (149.0, 85.0-744.0) versus 275.0 µg/dl (117.4, 89.0-597.0), respectively, p=0.271) whereas in women serum DHEA-S was higher in patients compared to controls [311.4 µg/dl (194.8, 70.0-790.0) versus 179.2 µg/dl (75.9, 46.0-314.0), respectively, p=0.005]. Serum cortisol and ACTH levels were not different in the above subgroups except serum cortisol in men which was lower in patients compared to controls [12.8 µg/dl (4.4, 3.5-21.6) and 15.9 µg/dl (5.4, 4.2-30.1), respectively, p=0.027]. **Conclusions:** Serum DHEA-S levels were higher in drug-naïve, first episode female patients, with psychosis compared to controls. DHEA-S levels in male patients and controls were similar.

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Diagnostic Threshold for Postoperative Secondary Adrenal Insufficiency After Transsphenoidal Resection of Pituitary Adenomas

Massiell German, MD[1], Anu Sharma, MBBS[2].
[1]University of Utah, Salt Lake City, UT, USA, [2]The University of Utah School of Medicine, Salt Lake City, UT, USA.

Transsphenoidal surgery (TSS) is the first line treatment for pituitary adenomas. A well-known complication of TSS is secondary adrenal insufficiency with a reported risk of 4-9% after TSS. Currently, glucocorticoid replacement is recommended if postoperative AM cortisol is < 3 ug/dL. Postoperative adrenal insufficiency is ruled out if AM cortisol is > 15 ug/dL. However, further evaluation of the adrenal axis with ACTH stimulation test is recommended for intermediate cortisol levels 3-15 ug/dL. Other studies have proposed postoperative cortisol threshold of < 4-14 ug/dL for glucocorticoid replacement. Retrospective analysis of all patients undergoing TSS at a tertiary center from January 2013 through April 2016 was performed. ACTH producing adenomas (Cushing's disease) were excluded. Of the 97 patients included, 17.5% (n=17) had secondary adrenal insufficiency requiring glucocorticoid replacement at 1 year post operatively. Mean age at presentation was 56 ± 16 years and 52% were female. Mean adenoma size was 25.3 ± 11.3 mm. Factors associated with adrenal insufficiency at 1 year post operatively were preoperative secondary adrenal insufficiency (AM cortisol 4.5 ± 1.9 vs 11.0 ± 1.0 µg/dL; $p = 0.03$), and preoperative adenoma contact with optic chiasm (15.7% vs 2.1%; $p = 0.01$). Day 1-7 postoperative cortisol was lower in the group with adrenal insufficiency at 1 year (5.6 µg/dL (IQR 1.9-11.5) vs 19.8 µg/dL (IQR 12.75-43.2); p=0.02). Age, gender, adenoma size, and cavernous sinus involvement were not associated with adrenal insufficiency at 1 year. A day 1-7 postoperative cortisol concentration of ≥8.0 µg/dL had a sensitivity of 75% and specificity of 92% in predicting adrenal insufficiency at 1 year. In patients with secondary adrenal insufficiency at 1 year (n=17), there was a higher frequency of concomitant loss of other pituitary hormone function at 1 year: secondary hypothyroidism 82% (n=14), secondary hypogonadism 70.6% (n=12) and diabetes insipidus 17.7% (n=3). A lower postoperative cortisol threshold of 8 µg/dL can be adopted for glucocorticoid replacement on discharge after TSS.

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Drug Induced Hyperprolactinemia

ASHOK VENKATANARASU, DM ENDOCRINOLOGY[1], Raman Boddula, DM Endocrinology[2], SANTOSH BASAVARAJU, Senior resident, Endocrinology[3], CHIMUTAI CHINTE, Senior resident, Endocrinology[4], Vidya tickoo, Senior resident, Endocrinology[3].
[1]SANJEEVANI THYROID, SUGAR AND HORMONES CLINIC, HYDERABAD, India, [2]Yashoda Hospital, Secunderabad, India, [3]Yashoda Hospital, Secunderabad, India, [4]Yashoda hospital, Secunderabad, India.

Introduction: Drugs are a common cause of hyperprolactinemia. It is essential to differentiate this cause from other pathological causes which avoids unnecessary investigations. Detailed history will help us in identifying the responsible drug and withdrawing the same will be rewarding. Here we are reporting a case series of drug induced hyperprolactinemia. **Methods:** It was a cross-sectional observational study. Subjects were recruited from our outpatient department. Hyperprolactinemia was defined as blood prolactin levels >30 ng/mL in females and >24 ng/mL in males, regardless of the presence of symptoms. Serum prolactin was repeated one week after holding the suspected drug(s). Drug induced hyperprolactinemia is defined as normalisation of serum prolactin on discontinuation of the offending drug. Demographics, clinical presentation and offending drug name were entered in pre designed proforma. **Results:** Total of 32 subjects were studied in this study with age of 35.5±10.8years. Predominantly female subjects were present with female to male ratio 5.4. Basal prolactin was 132±68.7ng/mL and after holding the drug prolactin value was 16.9±8.2. Proton pump inhibitors in combination with prokinetics were the leading cause (71.8%) and followed by multiple drug combinations (15.6%), anti-psychiatric drugs(9.3%) and oral contraceptives(3.1%). Overall 86.75% of subjects were having symptomatic presentation. Commonest clinical presentation among the women was galactorrhea (88.9% of female subjects) followed by irregular menstrual cycles (59% of female subjects) and breast heaviness in 29.6%. Among the men erectile dysfunction was common presentation, noted among 80% of them. Asymptomatic presentation was there in 6.25% of subjects. All subjects were improved clinically after withdrawal of the the offending drug(s).
Conclusions: Most of the subjects were clinically symptomatic. Most common symptom in female was galactorrhea followed by irregular menses and breast heaviness, and erectile dysfunction in males. All subjects were improved clinically after withdrawal of the offending drug(s). A detailed drug history is rewarding and avoids unnecessary investigations for hyperprolactinemia