Adrenal Hyperplasia Due to 21-Hydroxylase Deficiency. NEJM 2020;383:1248–61.

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ADRENAL CASE REPORTS

Testosterone Use and Adrenal Hemorrhage

Moin U. Sattar, MD[1], Priyanka L. Mahali, MD[1], Natalia Chamorro-Pareja, MD[2], Nisha Suda, MD MHA[3], Noah Bloomgarden, MD[4].

[1]Montefiore Medical Center/Albert Einstein College of Medicine (Moses and Weiler Campuses) Endocrine, Bronx, NY, USA, [2]Jacobi Medical Center, New York, NY, USA, [3]Montefiore Medical Center / Albert Einstein College of Medicine, New York, NY, USA, [4]Montefiore Hospital & Medical Center, Scarsdale, NY, USA.

Background: Testosterone supplementation has been associated with a variety of side effects, such as polycythemia, and can potentially increase the risk of cardiovascular disease. Testosterone use has also been associated with increased thrombotic events, especially in patients with underlying hypercoagulable state. **Clinical Case:** A 57-year-old man presented with abdominal pain and distention. He had history of previous intramuscular (IM) and oral testosterone use for ten years. For 8 weeks prior to initial presentation, he reported using weekly IM 500mg Testosterone injections. Computed Tomography (CT) of the abdomen and pelvis revealed multiple thrombi of the portal, splenic, superior mesenteric and inferior mesenteric veins. He was started on Warfarin and discharged home. A few weeks later, he presented with similar symptoms with labs now showing an International Normalized Ratio (INR) of 10.2. Repeat CT was significant for presence of bilateral adrenal hemorrhage, measuring 2.9cm on the right and 2.4cm on the left, which were not seen on previous imaging done one week prior. During the hospital course, he was found to be hypotensive with low platelet count so Intravenous Immunoglobulin therapy was initiated for suspected catastrophic antiphospholipid syndrome (CAPS). Morning cortisol was 5.82 ug/dL (6.2–29.0) so this critically-ill patient was started on stress dose hydrocortisone, which was subsequently tapered to physiological dose after clinical improvement. Cosyntropin stimulation test was performed after withholding the prior dose of hydrocortisone. The baseline cortisol was 0.88 ug/dL (6.20–29.00ug/dL), after administration of 250mcg of Cosyntropin 30- and 60-minute cortisol levels were 1.5 ug/dL (4.3–22.4ug/dL) and 1.6 ug/dL (4.3–22.4ug/dL) respectively. Baseline ACTH of 121.0 pg/mL (7.2–63.3pg/mL), consistent with primary adrenal insufficiency. Dehydroepiandrosterone Sulfate (DHEA-S) level was 15.7 ug/dL (80.0–560.0ug/dL). Hypercoagulability workup was significant for the presence of lupus anticoagulant and antibodies positive for heparin induced thrombocytopenia, so patient was diagnosed with Antiphospholipid syndrome. **Conclusion:** This is a case of hypercoagulability in a patient with history of anabolic steroid misuse who developed extensive intraabdominal venous thrombosis, adrenal hemorrhage, and primary adrenal insufficiency. Adrenal vein thrombosis and hemorrhage can be life threatening sequalae of testosterone misuse and should be considered in the differential for patients with history of testosterone misuse and adrenal insufficiency.

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The Case of a Glucocorticoid-Induced Myopathy & Myasthenia Gravis Combination: Is There a Border?

Irina Dzherieva, PhD[1], Snezhana Brovkina, MD[1], Natalia Volkova, MD,PhD[1], Ilia Davidenko, MD[1], Igor Reshetnikov, MD[2], Maxim Yudin, MD[3], Maria Plyashkevich, MD[1], Maria Merenkova, MD[1].

[1]Rostov State Medical University, Rostov on Don, Russian Federation, [2]Rostov State Medical University, Rostov-on-Don, Russian Federation, [3]Hospital, [4]Rostov on Don, Russian Federation.

Introduction: Glucocorticoid-induced myopathy is the sign of Cushing's syndrome in 83%, but can also be consequence of exogenous administration of glucocorticoids. The patients with the compromised neuromuscular system (for example, because of myasthenia gravis) have a higher risk of this complication. Differential diagnosis of glucocorticoid-induced myopathy is challenging. Clinical case. A 77-year woman complained of difficulty in the act of breathing, swallowing, weakness in the limbs, inability to self-service, ptosis. Due to the growing complaints the patient was hospitalized to ICU. From anamnesis: She was diagnosed with myasthenia gravis 2 months ago. The presence of autoantibodies to the nicotine acetylcholine receptor was confirmed. Pyridostigmine bromide 60 mg/day, Methylprednisolone 20 mg/day were prescribed for 5 days. The patient noted some improvement. After the next neurologist's examination the dose of Methylprednisolone was doubled. (It is not known for certain whether the doctor wanted to reduce the dose and the patient misunderstood.) She noted some worsening: weakness and speech difficulties increased. She returned to the previous dose after 3 days, but there was no improvement. Objectively: hypersthenic body type, BMI 39 kg/m2, predominant deposition of adipose tissue in the abdomen and face. Breathing without a ventilator. BP 250/100 mm Hg, heart rate 87 BPM. Laboratory tests revealed hyperkalemia of 8.76 mmol/l (3.5–5), creatinine of 481 mmol/l (44–124), hyperglycemia of 16 mmol/l. Glucocorticoid-induced myopathy was suspected, the administration of methylprednisolone was cancelled. The improvement of the condition was noted in 3 days. Blood pressure, glycaemia, levels of potassium and creatinine returned to normal. A biopsy of muscle revealed: atrophy of type I and II muscle fibers. There were no signs of necrosis, regeneration or inflammation. Thereby the diagnosis of glucocorticoid-induced myopathy was confirmed. Conclusion. Glucocorticoid-induced myopathy may look like a decompensation of neuromuscular disease. Since there are no accurate diagnostic tests nowadays the main argument of diagnosis is the paradoxical reaction after increasing the dose of glucocorticoids as well in this case. Even a short period of use of these drugs can lead to the development of side effects. It is necessary to regularly monitor the dynamics of the condition of such patients.

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The Development of Hypogonadotropic Hypogonadism After Starting Low Dose Mifepristone for Cushing's Syndrome