Thyroid

THYROID DISORDERS CASE REPORT

Severe Rhabdomyolysis and Acute Renal Failure Due to Noncompliance to Levothyroxine Therapy

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Background: Noncompliance to levothyroxine (LT4) is common however only rarely it leads to severe side effects. We report a case of rhabdomyolysis leading to acute kidney injury (AKI) requiring hemodialysis (HD) due to noncompliance to LT4 therapy for one month. Clinical Case: A 68-year-old Caucasian male presented with a 2-week history of worsening fatigue and generalized weakness, accompanied by pain in bilateral lower extremities. Medical history included coronary artery disease, heart failure with reduced ejection fraction, hypertension, dyslipidemia, hypothyroidism, type 2 diabetes mellitus, and CKD. Home medications included LT4 200 mg daily, metoprolol 25 mg daily, doxazosin 5 mg daily, fluoxetine 40 mg daily, fosinopril 40 mg twice daily, amlodipine 5 mg daily, povidone-iodine solution to care for her surgical site post-operatively. Upon further inquiry, she recalled using amiodarone use. Upon further inquiry, she recalled using amiodarone use. She was taking it 3 to 4 days before coming to the hospital. On examination, he had proximal muscle weakness with power 3/5 in bilateral lower extremities and mild tenderness on thigh muscles. Labs revealed creatinine 13.1 mg/dL (0.60-1.10 mg/dL), BUN 101 mg/dL (0-30 mg/dL), eGFR 4.0 mL/min/1.73m2, CK 69,500 U/L (22-198 U/L), TSH 55.8 uIU/L (0.8-1.8 uIU/L), and FT4 0.61 ng/dL (0.8-1.8 ng/dL). ABG showed metabolic acidosis. Routine labs three months prior revealed normal thyroid function tests (TSH 1.6 uIU/ml and FT4 1.3 ug/dL) on LT4 200 mcg and baseline CKD stage 3b (eGFR 51 mL/min/1.73m2 with baseline creatinine 1.4 mg/dL). The patient was diagnosed with severe rhabdomyolysis secondary to noncompliance with LT4 therapy in presence of concurrent statin use, leading to AKI. Rosuvastatin was stopped and he was treated with aggressive intravenous hydration, sodium bicarbonate, and LT4 200 mcg daily. Despite two days of aggressive treatment, CK remains elevated and hence HD was initiated. The patient underwent three sessions of HD during the course of his hospitalization. Due to lack of renal recovery, outpatient HD was arranged. At 4 weeks of outpatient follow-up, the patient was oliguric and HD dependent. At 8 weeks outpatient follow-up, CK, TSH, and FT4 was normal on LT4 200 mcg daily and became dialysis independent. Conclusion: Noncompliance to LT4 therapy along with concomitant use of statin can result in severe rhabdomyolysis induced AKI in patients with CKD.

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THYROID DISORDERS CASE REPORT

Severe Thyrotoxicosis Following Topical Iodine Application

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Background: Exposure to iodine can lead to iodine-induced hyperthyroidism in patients with underlying thyroid disease. Clinical Case: A 67-year-old woman with a history of nontoxic multinodular goiter and atrial fibrillation presented with fatigue, palpitations, weight loss, and tremor. Laboratory evaluation demonstrated new-onset profound biochemical hyperthyroidism (FT4 > 7.77 ng/dL, n 0.8 – 1.8 ng/dL; FT3 > 27.0 pg/mL, n 2.0-4.4 pg/mL). She was treated with beta-blocker, high doses of methimazole, and cholestyramine while further evaluation was pursued. She declined SSKI due to reported iodine allergy and steroids due to concerns about impact on wound healing following recent hip arthroplasty. TSI and TRAbs were negative, and thyroid ultrasound showed stable nodules at 1.7cm. Pelvic ultrasound and MRI were obtained due to concern for non-thyroidal etiology, and revealed a 3.7cm septated cystic ovarian lesion, raising suspicion for struma ovarii. Whole body scan to localize site of thyroid hormone production could not be obtained due to high risk of clinical deterioration after methimazole, as she had persistent clinical and biochemical thyrotoxicosis on high doses (up to 90mg/day). She ultimately required 3 sessions of plasma exchange to lower her thyroid hormone levels, and then underwent bilateral salpingo-oophorectomy. Final pathology revealed mucinous cystadenoma without ectopic thyroid tissue. Post-operatively, her thyroid hormone levels were persistently elevated but improved compared to pre-operative levels, allowing for brief cessation of methimazole and completion of whole body scan. Imaging demonstrated a single focus of radioactive iodine uptake in the lower right thyroid lobe, correlating with the dominant 1.7 cm nodule on prior ultrasound, consistent with a toxic adenoma. Additionally, she was found to have an elevated urine iodine level (1200 mcg/24 hours, n 75 – 851 mcg/24 hours). Patient endorsed low iodine diet due to allergy history, and denied recent contrasted imaging study, dietary supplements, or amiodarone use. Upon further inquiry, she recalled using povidone-iodine solution to care for her surgical site post-arthroplasty, approximately a week before the onset of her initial symptoms. Her clinical presentation was ultimately attributed to toxic adenoma, with severe thyrotoxicosis exacerbated by iodine load. She underwent total thyroidectomy and is doing well on levothyroxine post-operatively.
Conclusions: Topical iodine administration can contribute to iodine-induced hyperthyroidism in patients with underlying thyroid disease, and its use should be carefully considered in these patients. When evaluating a patient with new thyrotoxicosis, a detailed history of oral, IV, and topical iodine use should be obtained.

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THYROID DISORDERS CASE REPORT
Skin Deep: A Rare Case of Thyrotoxic Periodic Paralysis in an African American Patient
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Background: Thyrotoxic Periodic Paralysis (PP) is a rare form of hypokalemic PP that occurs in association with hyperthyroidism, especially Grave’s disease. This disease is frequently seen in males and is particularly prevalent among Asians with an incidence rate of 2%. In non-Asian populations, the incidence among those with hyperthyroidism is even lower at 0.1 - 0.2% and therefore significantly rare in African populations. Inability to recognize this emergency in the non-Asian population can therefore result in potentially fatal outcomes. Case Presentation: A 27 year old African American male with a history of Grave’s disease presented to the emergency department (ED) with the inability to move his muscles. Patient was initially diagnosed with Grave’s disease in 2017 when he was found to have suppressed TSH with elevated TSI and started on methimazole 40mg daily. The patient ran out of methimazole about 2 weeks prior to presentation and woke up on the day of admission with extreme muscle weakness. At the outside hospital, he was found to have suppressed TSH with elevated TSI and started on methimazole 40mg daily. The patient was restarted on Methimazole 40mg daily.

Discussion: Thyrotoxic PP is seen in a male-to-female ratio ranging from 17:1 to 70:1 and occurs at an average age of 20-40 years. Thyrotoxic PP is especially rare in the non-Asian population at an incidence rate of 0.1 - 0.2%. Nevertheless, in setting of ever-growing diversity due to immigration and inter-race relationships, it is difficult to predict one’s genetics based on the color of their skin. It is possible that our African American patient may have an Asian ancestor unbeknownst to him. Therefore, we must keep a broad differential regardless of one’s race so as to not miss timely diagnosis of medical emergencies which can result in reduced muscle strength, flaccid paralysis, respiratory failure, cardiac arrhythmias and eventual death.

Thyroid
THYROID DISORDERS CASE REPORT
Stopping Levothyroxine Therapy in Subclinical Hypothyroidism, Perhaps We Could Start a Trend
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Introduction: Subclinical hypothyroidism (SCH) is diagnosed based on elevated thyrotropin (TSH) and normal thyroxine (FT4) levels. Previous guidelines recommended treatment of SCH with levothyroxine (LT4) when TSH is > 10 uIU/mL or if TSH <10 uIU/mL with symptoms suggestive of hypothyroidism, positive thyroperoxidase antibodies (TPO Ab) or evidence/risk factors of cardiovascular disease. There has been an increasing practice of LT4 prescription for SCH, which contributes to making LT4 the second most prescribed drug in the US. Case: This case reviews the course of a patient with SCH treated with LT4. A 68 year old woman with medical history of hyperlipidemia, osteoporosis, and non-toxic multinodular goiter was diagnosed with SCH due to Hashimoto’s thyroiditis based on TSH 9.59 uIU/mL (0.34 - 5.60), FT4 0.66 ng/dL (0.58 - 1.64), and +TPO Ab. The patient had similar thyroid function tests (TFT) 1 year ago. She reported symptoms of hair loss, dry skin, and fatigue. She decided to undergo a trial of LT4 50 mcg daily. Two months later, her TSH had normalized and she reported slight improvement of fatigue. After one year on LT4, the patient reported symptoms of anxiety and heat intolerance and decided to stop LT4. Laboratory work up revealed TSH 0.22 uIU/mL and FT4 1.03 ng/dL consistent with exogenous hyperthyroidism. At the time of her follow up, the patient had been off LT4 for about 3 weeks. She continued to have ongoing fatigue, but reported resolution of the hyperthyroid symptoms. She was advised to stay off LT4 and to have yearly TFT. Discussion This case illustrates that not only LT4 treatment for SCH did not result in apparent improvement of hypothyroid-related symptoms, but also caused iatrogenic hyperthyroidism. A recent meta-analysis of 21 randomized clinical trials including 2192 patients with SCH found that LT4 therapy is not significantly associated with improvement in general quality of life or thyroid-related symptoms. Additionally, there is evidence of potential harm associated with LT4 and added burden of lifelong management. In 2019, a new guideline panel issued a strong recommendation against thyroid hormones in adults with SCH. Patients who are already on LT4 therapy for SCH may benefit from LT4 discontinuation. Clinicians need to discuss with their patients if LT4 discontinuation is a reasonable consideration. Studies at low risk of bias assessing patient important outcomes after LT4 discontinuation are required imminently.