classic features of hypothyroidism with muscle stiffness, weakness, and pseudohypertrophy.

**Clinical Case:** A 62-year-old man was admitted to our institution due to dyspnea. He also had progressive muscular weakness and recurrent cramps four months prior. On admission, he was seen in respiratory distress and was eventually intubated and hooked to mechanical ventilation for acute respiratory failure brought about by COVID-19 pneumonia (confirmed by a reverse transcriptase-polymerase chain reaction of a nasopharyngeal swab on admission). He had an unremarkable head and neck examination; both of his calf muscles were hypotrophied. On neurological examination, he had mild proximal lower limb muscle weakness (power 4/5) and deep tendon reflexes were sluggish (+1). The patient was subsequently admitted into the intensive care unit. Laboratory data showed inflammatory markers associated with COVID-19 were elevated: Lactate Dehydrogenase (857 u/L; NV: 120-246 u/L) and Interleukin-6 (137 pg/mL; NV: 0-50 pg/mL). He was given tocilizumab, convalescent plasma therapy, and underwent hemoperfusion. His serum chemistry was unremarkable. Thyroid function tests were consistent with primary hypothyroidism from Hashimoto’s thyroiditis: TSH (22.6712 IU/mL; NV: 0.35-4.94 IU/mL), fT3 (<2.30 PMOL/L; NV: 2.89-4.88 PMOL/L), fT4 (<5.15 PMOL/L; NV: 9.01-19.05 PMOL/L), anti-Tg Ab (1810.3 IU/mL; NV: <50 IU/mL) and Anti-TPO Ab (2610.4 IU/mL; NV: <100 IU/mL). There was a consideration of Hoffman’s syndrome; muscle enzymes were noted to be elevated also: CK-Total (2589 u/L; NV: 120-246 u/L) and Lactate Dehydrogenase (857 u/L; NV: 120-246 u/L). Upon initiation of hormone replacement, his symptoms improved. Thus, we were able to shift to oral levothyroxine route at 300 mcg once daily (approximately twice the recommended dose). Upon the addition of liothyronine 5 mcg BID, his symptoms were noted to be improved significantly and his CK levels were normalized. Despite an initial roadblock, levothyroxine was given as an enema via the rectal route at 300 mcg once daily (approximately twice the recommended dose). Upon the addition of liothyronine 5 mcg BID, his symptoms were noted to be improved significantly and his CK levels were normalized. Despite an initial roadblock, levothyroxine was given as an enema via the rectal route at 300 mcg once daily (approximately twice the recommended dose). The patient was subsequently discharged to the rehabilitation ward and expired after the 11th hospital day.

**Conclusion:** COVID-19 infection could lead to exacerbations of pre-existing conditions such as Hashimoto’s Thyroiditis (in this case, manifesting with Hoffman’s syndrome). Hormone replacement is still the treatment of choice. Concomitant COVID 19 infection and treatment is not a roadblock, levothyroxine was given as an enema via the rectal route at 300 mcg once daily (approximately twice the recommended dose). The patient was subsequently discharged to the rehabilitation ward and expired after the 11th hospital day.

**Reference:** (1) Senanayake, Hemal Ms et al. “Hoffmann syndrome: a case report.” *International archives of medicine* vol. 7, 1 2. 6 Jan. 2014, doi:10.1186/1755-7682-7-2.

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**Thyroid**

**Thyroid Disorders Case Report**

**High-Dose Thyroid Hormone Replacement in Bexarotene-Induced Central Hypothyroidism.**

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**Background:** Central hypothyroidism is a rare disorder characterized by a defect in thyroid hormone production by an otherwise normal thyroid gland due to decreased stimulation by TSH. Medications are an uncommon cause. **Case Presentation:** A 72-year-old man was referred for evaluation of a low TSH. He had a long history of hypothyroidism, euthyroid on levothyroxine, and was diagnosed with cutaneous T cell lymphoma (CTCL). Due to disease progression on dapsone, PUVA and TAR baths, he was started on bexarotene. Soon after, he developed recurrent symptoms of hypothyroidism including fatigue, cold intolerance, dry skin, and myalgias. Workup revealed a TSH of <0.01 ulU/ml (0.27-4.20) with a free T4 of 0.6 ng/dl (0.9-1.7). After evaluation, his levothyroxine dose was increased. Repeat labs 3 months later showed a TSH of <0.01 with a free T4 0.8 ng/dl and T3 43 ng/dl (80-200). Over several months, levothyroxine titration to a supraphysiologic dose of 800 mcg daily was required, despite optimal administration, to normalize FT4. Given persistent hypothyroid symptoms and a low T3 level, liothyronine 5 mcg BID was added and resulted in clinical and biochemical euthyroidism. **Clinical Lessons:** Unlike in primary thyroid disorders, the TSH assay is unreliable in central hypothyroidism since values can be low, normal, or even mildly elevated; regardless, TSH has subnormal bioactivity. Ineffectual TSH leads to clinical and biochemical euthyroidism. **Lessons:** Unlike in primary thyroid disorders, the TSH assay is unreliable in central hypothyroidism since values can be low, normal, or even mildly elevated; regardless, TSH has subnormal bioactivity. Ineffectual TSH leads to clinical and biochemical euthyroidism. **Conclusion:** Ineffectual TSH leads to clinical and biochemical euthyroidism. **Introduction:** Patients with thyroid storm and resistance to conventional medications may receive plasmapheresis until they have the definitive therapy. **Case Presentation:** A 42 years old lady with no past medical history was brought by the EMS with palpitations, shortness of breath, vomiting, and profuse diarrhea. She was found to have an atrial flutter with low blood pressure, received synchronized cardioversion, but unfortunately,
she developed ventricular tachycardia, tonic-clonic seizure and went to pulseless electrical activity (PEA). Upon examination, the patient was intubated, heart rate of 200 beats/min, blood pressure of 80/60 on vasopressors. She had exophthalmos and icteric eyes. Neck examination revealed palpable goiter. There was bibasal fine cracked and mild lower limb edema. Laboratory showed FT4 39 (11.6-21.9 pmol/L), FT3 5 (3.7- 6.4 pmol/L), and TSH <0.01 (0.3-4.2 mIU/L). Burch- Wartofsky’s score was 55/140. Her presentation was suggestive of Graves’ disease with thyroid storm. Further labs showed high liver enzymes, high INR, ammonia as well as high creatinine.

She was started on IV hydrocortisone and cholestyramine. Thionamides were contraindicated due to liver impairment. Extracorporeal membrane oxygenation (ECMO) was initiated for cardiopulmonary support and continued for 6 days. TSH receptor antibodies result was pending, thus a thyroid uptake scan was done while the patient connected to ECMO to confirm the diagnosis. Thyroid scan showed increased uptake suggestive of grave’s disease despite iodine contrast received for CT scan chest two days back. After 5 sessions of plasmapheresis, FT3 2.8, and FT4 30, Lugol’s iodine started and she underwent total thyroidectomy. She was successfully extubated and thyroxine replacement was started after normalization of thyroid hormones

Discussion: The raised liver enzymes (shock liver) were a barrier to thioamides. With the contraindication to antithyroid medications, plasmapheresis was a rapid and safe option before thyroidectomy. The mechanism of plasmapheresis is to eliminate thyroid hormones, TSH-receptor antibodies, and cytokines. The current guidelines lack clear indications, the timing of initiation, and patient selection for plasmapheresis.

Conclusion: Plasmapheresis should be considered as a stabilising measure, especially when patients cannot tolerate conventional medications. Plasmapheresis leads to rapid decline in thyroid hormone levels, providing a window to treat definitively with thyroidectomy.

Thyroid

THYROID DISORDERS CASE REPORT

Hyperthyroid Manifestation in Beta HCG Secreting Tumor

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Background: Serum β-Human Chorionic Gonadotropin (HCG) levels can be increased not only in pregnancy but also in various malignancies such as germ cell malignancy, lung cancer, ovarian cancer, and breast cancer. The alpha subunit of HCG and TSH are homologous, so β-HCG can cross-react with the TSH receptors and induce hyperthyroidism. High level of β-HCG serum can lead to hyperthyroidism, which can aggravate the patient’s malignant manifestations.

Case Presentation: A 33-year-old woman admitted to our hospital with chief complaint general weakness one week before admission. The patient was 8 week-pregnant and the transabdominal ultrasound showed a gestational sac and she had also a positive urine β-HCG test. The patient had slight vaginal bleeding 1 month ago. The physical examination revealed tachycardia, pale conjunctiva, and multiple nodules in both breasts. On laboratory examination, we found low level of Hb 6.7 (n 11.7 - 15.5 g/dL), serum iron 18 (n 65 - 175 mg/dL), TIBC 164 (n 253 - 435 mg/dL), and ferritin 1971 (n 4.63-204 mg/dL), positive urine β-HCG, potassium 2.5 (n 3.1 - 5.1 mmol/L). On transvaginal and transabdominal ultrasound examination, there was no gestational sac in the uterine cavity and there are no abnormalities in other gynecology organs. The chest X-ray showed a nodule in the upper right lung suggestive of metastasis with thickening of the soft tissue of the left mammary region suggestive of a left breast mass. We also found a consistent positive result of urine β-HCG, though it had passed four weeks after the occurrence of vaginal bleeding. Finally, the serum β-HCG examination was carried out and showed an increased result, 290,398 (n <5 mIU/ml). The chest CT showed a heterogeny mass (mixed iso-dense and hypodense) that enhanced contrast on the inferior lobe of left lung (4.1 cm x 12.1 cm x 14.1 cm), a mass on apical segment of right lung, subpleural nodule, a mass on the right kidney, spleen, and mediastinal lymphadenopathy. The patient had packed red cell transfusion and after the Hb level reached 11 mg/dl, she still had tachycardia, so we examined the TSHs level. TSHs was found to be low at 0.014 (n 0.48-4.17 mg/dL), and FT4 increased to 2.82 (n 0.89-1.76 mg/ dL). Thyroid ultrasound showed small simple cysts in both inferior thyroid pools, no solid mass and no increased vascular flow to the thyroid parenchyma. The patient was then thought to have hyperthyroid manifestation due to β-HCG secreting tumor. She was had methimazole and propranolol therapy, and a lung biopsy was planned.

Conclusion: Trophoblastic and non-trophoblastic tumors that secrete high level of β-HCG can induce hyperthyroid manifestations, particularly if the level was more than 20,000 mIU/mL.

Thyroid

THYROID DISORDERS CASE REPORT

Hyperthyroidism and Immune Thrombocytopenia (ITP) Overlap Presenting as Hypoxic Ischemic Encephalopathy in a 38-Year-Old Filipino Female: A Case Report

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Background: The coexistence of these two conditions can mimic severe sepsis in critically ill patients which may leave severe thyrotoxicosis unrecognized. Its higher incidence in Asian population suggests a possible genetic propensity.

Clinical Case: A 38-year-old Filipino female sought consultation due to changes in behavior. Two months prior, patient had persistent heavy menstrual bleeding but refused work-up. There was progressive weakness and pallor in the...