most common cause of death, but conventional therapies can be limited depending on the clinical presentation. We present a case of a patient in thyroid storm who rapidly developed multi-organ failure, preventing her from obtaining potentially life-saving treatment. **Case Presentation:** A 68-year-old female with a past medical history of hypertension, hyperlipidemia, and Grave's disease, who was non-compliant with medications, presented to a facility for shortness of breath after the unexpected death of her husband. She was diagnosed with a non-ST elevation myocardial infarction and new onset heart failure. At that time, her TSH level was <0.010 uIU/mL and Free T4 was 1.80 ng/dL. Imaging revealed a significantly enlarged thyroid gland measuring 8cm by 6.6cm. She was started on methimazole and discharged home. A few days after discharge, she underwent a cardiac catheterization and was found to have Takotsubo cardiomyopathy. On presentation to our facility 2 weeks later, the patient was experiencing worsening shortness of breath and anxiety. She was found to have new-onset uncontrolled atrial fibrillation with rapid ventricular response and a blood pressure of 77/38 mmHg. The Burch-Wartofsky Point Scale was calculated to be 55 points, highly suggestive of thyroid storm. TSH was < 0.010 uIU/mL, total T4 was 16.63 ug/dL, and free T4 was 3.28 ng/dL. She was initiated on propylthiouracil, cholestyramine, hydrocortisone, and esmolol. Within 12 hours, she developed fulminant multi-organ failure requiring ventilatory support and vasopressors. She also developed ischemic hepatitis and propylthiouracil was discontinued. Urgent therapeutic plasma exchange (TPE) and continuous renal replacement therapy (CRRT) were later attempted but both therapies were not initiated due to severe hemodynamic instability. A bedside echocardiogram revealed an estimated ejection fraction of 20-25%. Due to worsening cardiogenic shock, she was evaluated for extracorporeal membrane oxygenation (ECMO) but was not a candidate. She instead underwent an emergent Impella device implantation. Despite this intervention, the patient's clinical condition did not improve after multiple vasopressors, and the patient's family opted for comfort-focused measures. The patient died after 1 day of hospitalization. **Conclusion:** A multimodality approach to treatment is recommended for patients with thyroid storm but underlying conditions such as Takotsubo cardiomyopathy and fulminant multi-organ failure may complicate the treatment plan. The complexity of this case highlights the need to understand relative contraindications to salvage therapies, such as TPE, and the role for other treatment options when patients present with co-existing multi-organ failure.

Thyroid

THYROID DISORDERS CASE REPORT

A Case of Thyrotoxic Periodic Paralysis

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Introduction: Thyrotoxic periodic paralysis (TPP) is an uncommon disorder characterized by simultaneous thyrotoxicosis, hypokalemia, and paralysis. It is a rare complication of hyperthyroidism with a prevalence of 1 in 100,000. It has a higher prevalence in young Asian males, with much fewer cases reported in Hispanic patients. The majority of cases are seen in hyperthyroidism due to Grave's disease, however other causes of thyrotoxicosis have been associated with TPP. Hypokalemia occurs when thyroid hormone stimulates the sodium-potassium-ATP pump by binding to the thyroid response elements upstream of the genes for this pump, increasing its activity and thereby causing a transcellular shift of potassium into the intracellular fluid. Attacks usually begin with proximal muscle weakness of the lower extremities and may progress to tetraplegia, with the degree of muscle weakness corresponding to serum potassium levels. Interestingly, no correlations with serum T3 or T4 levels have been found.
Clinical Case: A 38-year-old Hispanic man with a history of GERD presented to the emergency department with an inability to move his extremities. Initial labs were significant for profound hypokalemia to 2.0 mEq/L (3.5-5) and hyperthyroidism, with TSH<0.05 uU/mL (0.3-4.2) and free T4 2.4 ng/dL (0.6-1.5). Additional workup revealed a positive thyroid stimulating immunoglobulin and a positive thyrotropin binding inhibitor, and thyroid ultrasound demonstrated a hyperemic thyroid with numerous subcentimeter hypoechoic nodules, all of which was suggestive of Grave's disease. His symptoms resolved with potassium repletion, suggesting thyrotoxic periodic paralysis secondary to hyperthyroidism. Upon further evaluation, the patient endorsed several months of hyperthyroid symptoms such as tremors, heat intolerance, and weight loss. He also endorsed alcohol use, a high carbohydrate diet, and recent life stressors, a combination of which likely precipitated his thyrotoxic periodic paralysis. He was discharged on methimazole 10 mg daily and propranolol 40 mg BID, with a decrease in his free T4 and improvement in his symptoms. He has been educated to avoid alcohol consumption and high-carbohydrate meals to avoid precipitating another episode of periodic paralysis, and once his hyperthyroidism is under better control he plans to undergo radioactive iodine ablation (RAI) for definitive management.
Conclusions: Thyrotoxic periodic paralysis is a rare but dangerous complication of hyperthyroidism, and should be considered in the differential diagnosis when young individuals present with otherwise unexplained hypokalemia and paralysis. Acute management is potassium repletion, long term management involves treatment of thyrotoxicosis and avoiding precipitating factors, and definitive therapy is RAI or thyroidectomy.

Thyroid

THYROID DISORDERS CASE REPORT

A Case of TSH-Secreting Pituitary Adenoma With Unusual Positive Staining for SF1

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Background: TSH-secreting pituitary adenomas are the rarest functional pituitary tumors. While they are often plurihormonal, SF1 positivity (indicating gonadotroph lineage) is unusual. Here we present the rare case of a patient with TSH-secreting pituitary adenoma with staining positive for TSH, GH, prolactin, SSTR2A, PIT-1, and SF1.