Although relatively small in size, the thyroid gland produces many hormones that have important and significant impact on the function of our organs. The most studied thyroid hormones are triiodothyronine (T3) and thyroxine (T4) which are regulated by thyroid-stimulating-hormone (TSH) produced in the anterior pituitary. When these chemicals become unbalanced in the serum, the regulation of energy is affected and clinical manifestations such as fatigue, slowed speech and movements, cool skin, bradycardia, and neurologic changes develop. Our patient displayed many of these conditions with the most alarming being the cognitive changes that characterize myxedema. Up to 90% of myxedema patients will display these changes. The term “Myxedema Coma” is somewhat misleading as patients do not have to be comatose to fit this disorder. More commonly, they are in “Myxedema Madness” which is the onset of psychosis or confusion due to a lack of thyroid hormones.

Case Report

The patient is a 43-year-old female with a past medical history of thyroid cancer (unknown type), status post complete thyroidectomy, history of myocardial infarction with stent placement, and hypertension who presented to the emergency department because of 1 week of sinus pressure and generalized fatigue. Because of her thyroidectomy, the patient had been prescribed Levothyroxine in the past, which she had not taken in 6 months. The patient had visited an urgent care center 1 week before for the sinus pressure and was prescribed an unknown corticosteroid. She only took this for one day. Throughout the interval week, she developed intermittent double and blurry vision. Occasionally, objects she would focus on would seem to “hover” over the surface of a table and two-dimensional objects on the television would appear to be three-dimensional. The most alarming change occurred when she suddenly awoke from sleep and had a strong urge to run out in the middle of traffic. She had never had any thoughts of suicide or homicide before. When she presented to our hospital, a TSH was ordered and found to be 153μIU/mL. Her Free T4 was <0.15ng/dL at that same time. High-doses of Levothyroxine were immediately given. She clinically improved after 3 days in the hospital with no more thoughts of suicide or visual changes. She was sent home with thyroid replacement therapy and close follow-up with her primary care physician and an endocrinologist.

Discussion

The exact pathophysiology causing the cognitive changes is not completely understood. Poor cerebral blood flow and thus poor glucose delivery to the brain have been suggested however no studies have been able to confirm this. When this delirium and its etiology are recognized, the standard therapy is immediate hormone replacement to a euthyroid state. Although the laboratory values might lag behind, there is generally quick clinical resolution of the symptoms once the replacement therapy gets to therapeutic levels. The degree of monitoring in the hospital must be decided on a case by case basis. Some patients present with severe symptoms despite mild-moderate hormone disturbances while others have mild symptoms in the face of very high TSH levels.

References