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Correlation between Platelet Count, TSH and Thyroxine in a Patient Presenting with Myxedema

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ABSTRACT

Myxedema is a very rare and life threatening endocrine emergency which results from severe hypo-thyroidism and effects almost every organ system. We-hereby-present a 64-year-old-woman with PMH of hypothyroidism, liver cirrhosis, DM, HLD who presented to our ED because of altered mental status and dizziness. Patient reported feeling ill for the last two days, appeared lethargic, and responded slowly to questions. She denied headache, fever, changes in vision/smell, neck pain, chest pain, cough/wheezing, abdominal pain, bowel or urinary symptoms. On arrival to the ED, patient was hypothermic with temperature of 93.2 F, hypotensive with blood pressure of 99/49 mm Hg, heart rate of 53 beats per minute and respiratory rate of 15 breaths per minute. On examination, the patient was lethargic, confused at times, heart sounds regular, no murmurs, soft, non-tender ab-domen, no significant findings on neck exam. Laboratory work-up was significant for white cell count of 2.6 L x 10^3/uL, Hb 11.6 g/dL, HCT 33.8%, platelet count 53 x 10^3/uL, TSH at presentation 73.30 mIU/L, free T4 was 0.12 ng/dL. Patient was treated with 200 mcg of IV levothyroxine, 10 mcg of leothyronine, 100 mg of hydrocortisone. In the ED, patient was given broad spectrum anti-biotics for the possibility of an infection triggering her myxedema. Over the course of days, it was observed that as the TSH trended down, the thyroid hormone level increased and the platelets trended up.

Trend in TSH (mIU/L), Free thyroxine (ng/dL) and platelets (x 10^3/uL) in our patient.

Day 1 73.30 0.12 53

Day 3 34.50 0.33 58

Day 5 27.40 0.35 70

Day 7 24.20 0.60 77

Day 10 7.85 0.88 103

There seemed to be an association with elevated TSH, low thyroxine and low platelets. The low platelets were very concerning to us. But we found that as the myxedematous state of our patient improved with a decreasing TSH and increasing T4, the platelet count got better. It's not the throm-bocytopenia that we need to treat. It's the Myxedematous state that we need to address and the he-matological abnormality corrects itself. As the TSH and the thyroid hormones normalize, the plate-lets correct themselves. One possible mechanism through which thyroid hormones may increase the number of megakaryocytes is the modulation of bone marrow matrix proteins, such as fibronectin [1, 2]. Thyroid hormones increase the expression of fibronectin gene. Individuals with hyperthy-roidism have elevated blood levels of fibronectin [1, 2]. Fibronectin appears to affect megakaryo-cyte maturation and thrombopoiesis through interaction with integrin α4β1 [3]. Apoptosis is the major mechanism through which platelets die and thyroid hormones have been shown to inhibit apoptosis in several cell lines [4].

Thrombocytopenia can be part of a multisystem complication of myxedema which corrects as the thyroid functions normalizes.

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