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Thyroid Storm with Acute Flaccid Quadriparesis due to Thyrotoxic Myopathy

https://doi.org/10.15605/jafes.034.S69

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INTRODUCTION

Severe thyrotoxicosis is known to cause myopathy, but is rarely associated with acute flaccid quadriparesis. It is imperative to distinguish this from other potentially life-threatening conditions such as Guillain-Barré syndrome, myasthenia gravis and hypokalemic periodic paralysis that may present with similar clinical features.

We report a case of thyroid storm presenting with acute flaccid paralysis.

CASE

A 25-year-old lady was diagnosed with Graves' disease one year ago but was poorly compliant to antithyroid drugs. She presented with 3 days' history of fever and rapidly progressive generalised body and limb weakness rendering her bedridden. Neurologic examination identified flaccid quadriparesis with areflexia and intact sensation. She was agitated, febrile, tachycardic with atrial fibrillation in failure and was diagnosed with thyroid storm with Burch and Wartofsky score of 60. Her thyroid stimulating hormone level was <0.01 mIU/L and free thyroxine was 43.56 pmol/L. Serum electrolytes and creatinine kinase were normal, and type 2 respiratory failure was not demonstrated. Other investigations were unremarkable including viral serology, autoimmune markers and anti-ganglioside antibodies. Her nerve conduction study and electromyography were suggestive of generalized myopathy without neuromuscular junction abnormalities. She was intubated, ventilated and commenced on hydrocortisone, Lugol's iodine, propylthiouracil, cholestyramine and propranolol, resulting in marked clinical improvement and normalisation of thyroid function in 7 days. Total thyroidectomy was done before discharge as definitive treatment. She regained muscle power and function gradually over months following biochemical remission.

CONCLUSION

Acute thyrotoxic-induced myopathy should be considered in uncontrolled thyrotoxicosis presenting with flaccid quadriparesis. Contributing features may include increased cellular metabolism and energy utilisation, increased catabolism and protein degradation, and inefficient energy utilisation. Early definitive therapy with radioactive iodine or thyroidectomy is crucial in achieving rapid biochemical control, preventing future occurrence of acute thyrotoxic induced myopathy, and improving muscle recovery.

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Hypothyroidism: The Great Mimicker

https://doi.org/10.15605/jafes.034.S70

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INTRODUCTION

Hypothyroidism is a common endocrinopathy presenting with an assortment of well-described symptoms and signs. Less commonly, myopathy may be the sole presenting manifestation, making it perplexing to ascertain the diagnosis from other systemic and local aetiologies. This is a case of severe hypothyroidism which had manifested itself in the form of persistently raised creatine kinase (CK) following a presumed episode of non-ST elevation myocardial infarction (NSTEMI).

CASE

A 57-year-old gentleman first presented to a district hospital in December 2016 with atypical chest pain. His electrocardiogram showed T-wave inversion over leads V2 to V6, I and AVL. Along with his raised CK (3,448 U/L), the impression then was NSTEMI and he was treated accordingly. Throughout his admission, his CK showed a declining trend and was 2,481 U/L upon discharge. An echocardiogram revealed good ejection fraction of 65% with no regional wall motion abnormalities.

However, during his subsequent visit, his CK did not normalise. Initial concern of statin-induced myopathy resulted in his statin being withheld. Nevertheless, his CK showed a further rise to 4,328 U/L in May 2017. Further history revealed symptoms of cold intolerance, fatigue and constipation, suggestive of hypothyroidism. He denied muscle aches or weakness and there was no demonstrable proximal myopathy. His subsequent thyroid function test demonstrated extremely high thyroid stimulating hormone (TSH) (>100 μIU/mL) with low free thyroxine (0.8 pmol/L). Following commencement of thyroxine replacement, his TSH (0.67 μIU/mL) and CK normalised.

CONCLUSION

In hypothyroidism, the involvement of skeletal muscles may vary, ranging from an asymptomatic rise in creatine kinase (CK) to overt muscle weakness. Because hypothyroidism can be a great mimicker, a high index of suspicion is imperative.