Thyrotoxic Periodic Paralysis

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Case Presentation

- A 23 year old Caucasian man without significant history presented with whole body weakness and paralysis.
- Laboratory revealed a potassium of 1.1 and a suppressed thyroid stimulating hormone (TSH) level.
- Paralysis improved with potassium replacement.
- Propylthiouracil (PTU) as well as methimazole were attempted for treatment of thyrotoxicosis; however, due to development of a skin rash, both were quickly discontinued.
- A subsequent endocrinology workup revealed a large left thyroid mass on ultrasound with thyroid scintigraphy illustrating an increased uptake in this thyroid mass consistent with a hyperfunctioning adenoma.
- Propranolol was initiated for management of thyrotoxic periodic paralysis.
- However, the patient was unable to tolerate propranolol secondary to hypotension and dizziness.
- Because of the patient's inability to tolerate oral anti-thyroid medication as well as beta-blocker therapy with severe intermittent paralysis symptoms associated with hyperthyroidism, he underwent surgical thyroidectomy.
- He tolerated this procedure well with resolution of of paralysis following thyroidectomy.

Background

- Hypokalemic Periodic Paralysis is a rare, usually hereditary form of electrolyte channelopathy resulting in episodes of muscle weakness that last from minutes to hours. Even more uncommonly, this can also be acquired in association with hyperthyroidism.

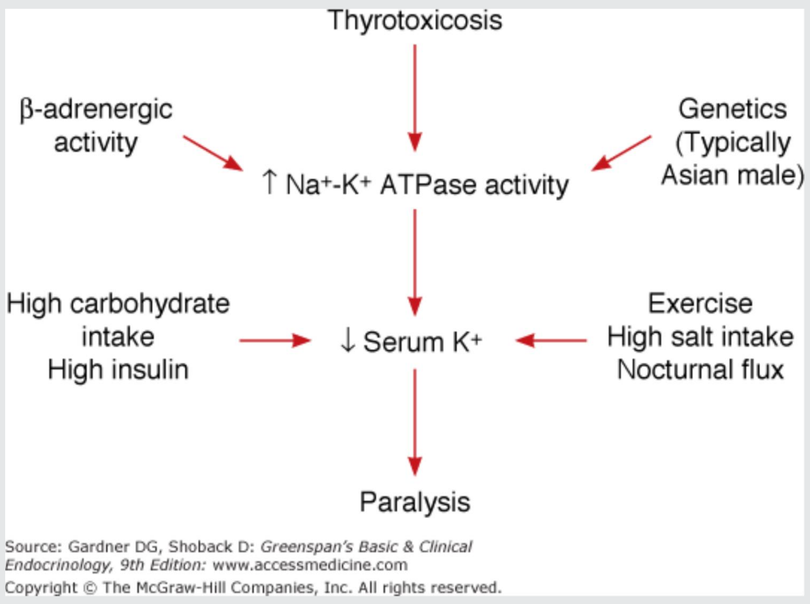

Figure 1: Proposed pathogenesis of thyrotoxic periodic paralysis.

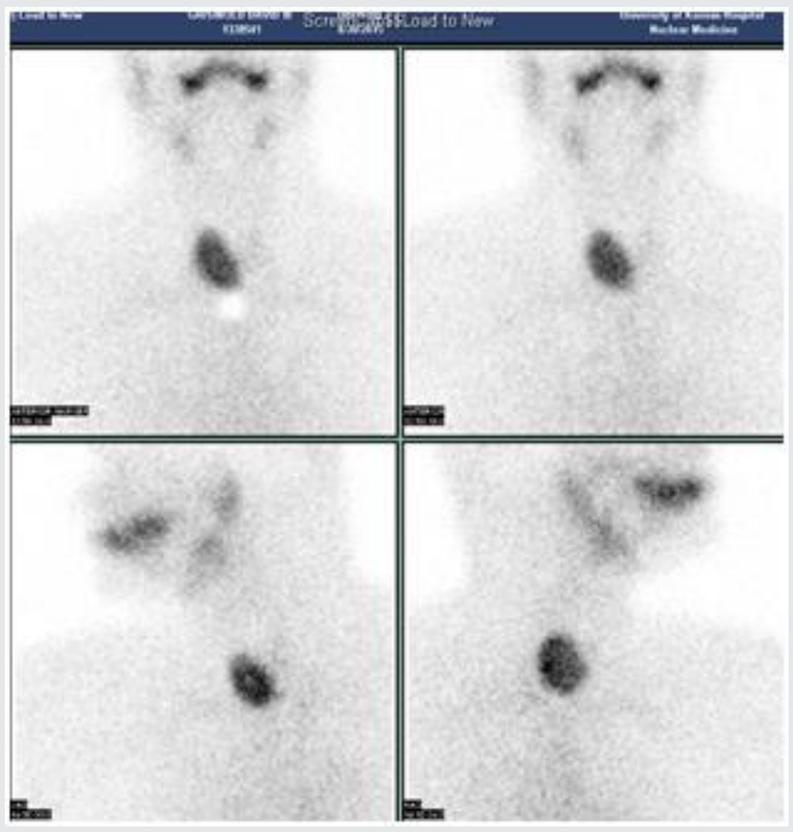

Figure 2: Images from thyroid uptake scan illustrating solitary hyperactive nodule.

References

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Discussion

- Thyrotoxic periodic paralysis (PP) represents an acquired form of hypokalemic PP.
- It is more prevalent in Asians than in non-Asians; it is more common in males than females.
- The mechanism by which hyperthyroidism can produce hypokalemic PP is not well understood.
- It is thought that thyroid hormone increases tissue responsiveness to beta-adrenergic stimulation, which, along with thyroid hormone, increases sodium-potassium ATPase activity on the skeletal muscle membrane.
- This tends to drive potassium into cells, perhaps leading to hyperpolarization of the muscle membrane and relative inexcitability of the muscle fibers.
- Thyrotoxic patients with periodic paralysis have been found to have higher sodium pump activity than those without paralytic episodes.
- Any cause of hyperthyroidism can be associated with thyrotoxic periodic paralysis.
- Treatment of an acute attack includes potassium replacement, beta-blocker therapy, and management of underlying thyrotoxicosis.