Case Report

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Unusual presentation of thyrotoxicosis with paraparesis in a young male: A rare case report

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ABSTRACT
Thyrotoxic Periodic Paralysis (TPP) is an uncommon disorder seen primarily in Asian males and caused by excessive thyroid hormones. This is an endocrine emergency that can lead to respiratory failure, dysrhythmia, and death. The mainstay of therapy has been potassium replacement. However, recent evidence suggests propranolol is a more effective therapy. We present a case of TPP in a 19-year male with rapidly progressive paraparesis & hypokalemia.

INTRODUCTION:

Even though it is commonly seen in Graves' disease, TPP is not related to the etiology, severity, and duration of thyrotoxicosis. [1]

The pathogenesis of hypokalaemic periodic paralysis in certain populations with thyrotoxicosis is unclear. Transcellular distribution of potassium is maintained by the Na+/K+–ATPase activity in the cell membrane, and it is mainly influenced by the action of insulin and beta-adrenergic catecholamines.[2] Hypokalemia in TPP results from an intracellular shift of potassium and not total body depletion. It has been shown that the Na+/K+–ATPase activity in platelets and muscles is significantly higher in patients with TPP.[3] Hyperthyroidism may result in a hyperadrenergic state, which may lead to the activation of the Na+/K+–ATPase pump and result in cellular uptake of potassium.[2, 4, 5] Thyroid hormones may also directly stimulate Na+/K+– ATPase activity and increase the number and sensitivity of beta receptors.[2, 6] Patients with TPP have been found to have hyperinsulinemia during episodes of paralysis. This may explain the attacks after high-carbohydrate meals.[7]

CASE REPORT:

A 19 year old male patient presented to our emergency room with sudden onset weakness of lower limbs. He was not able to stand or walk. Power of 0/5 in both lower limbs and 3/5 in upper limbs was noticed on examination. Routine investigations revealed to have severe hypokalemia with a serum potassium of 1.6 meq/l (normal range 3.5-5.0 meq/l), a serum phosphorus level of 3.4 mg/dl (normal range 3-4.5 mg/dl) and mild hypomagnesemia with serum magnesium level of 1.5mg/dl (normal range 1.8-3.0 mg/dl). ECG showed hypokalemic changes with prolonged PR interval, increased P-wave amplitude and widened QRS complexes. He was managed on intravenous as well oral potassium and history revealed weight loss, increased appetite and tremors from past 4 months. He had a multinodular goiter and radioactive iodine uptake scan (Iodine 131) showed a toxic nodule (Toxic nodule shows increased iodine uptake while the rest of the gland is suppressed) with no exophthalmos, sensory or cranial nerve deficits. Thyroid function tests revealed thyrotoxicosis with free T4 of 4.3ng/dl (normal range 0.8-1.8ng/dl), T3 of 279 ng/dl (normal range = 60 - 181 ng/dl) and a TSH level of <0.15milliunits/L (normal range = 0.3 - 4 milliunits/L). He was managed on intravenous potassium & propanolol. The patient showed dramatic improvement of his symptoms. The patient was discharged home on carbamazole with the diagnosis of TPP secondary to toxic nodular goiter.

In this case there was a significant family history as one of his elder brother had a sudden death (cause not known) and his mother was primary hypothyroid on levothyroxin replacement therapy.

DISCUSSION :

TPP is seen most commonly in Asian populations, with an incidence of approximately 2% in patients with thyrotoxicosis of any cause.[1,8,9,10] The attacks of paralysis have a well-marked seasonal incidence, usually occurring during the warmer months.[1] Pathogenesis of hypokalaemia has been explained by some authors to be due to an intracellular shift of body potassium, which is catecholamine mediated.[11,12] Shizume and his group studied total exchangeable potassium which revealed that patients with thyrotoxic periodic paralysis were not significantly different from controls when the value was related

to lean body mass.[11] The paralytic symptoms and signs improve as the potassium returns from the intracellular space back into the extracellular space.[13] The diurnal variation in potassium movement where there is nocturnal potassium influx into skeletal muscle would explain the tendency for thyrotoxic periodic paralysis to occur at night.[14] Hypophosphataemia and hypomagnesaemia are also known to occur in association with thyrotoxic periodic paralysis.[14,15,16,17,18] The correction of hypophosphataemia without phosphate administration supports the possibility of intracellular shift of phosphate.[16] Electrocardiographic findings supportive of a diagnosis of TPP rather than sporadic or familial periodic paralysis are sinus tachycardia, elevated QRS voltage and first-degree AV block (sensitivity 97%, specificity 65%).[20] In addition to ST-segment depression, T-wave flattening or inversion and the presence of U waves are typical of hypokalaemia.

The management is to deal with the acute attack as well as treatment of the underlying condition to prevent future attacks. Rapid administration of oral or intravenous potassium chloride can abort an attack and prevent cardiovascular and respiratory complications.[4] A small dose of potassium is the treatment of choice for facilitating recovery and reducing rebound hyperkalaemia due to release of potassium and phosphate from the cells on recovery.[1,2,3] Rebound hyperkalaemia occurred in approximately 40% of patients with TPP, especially if they received >90 mmol of potassium chloride within the first 24 hours.[4] Another mode of treatment is to give propranolol, a nonselective b-blocker, which prevents the intracellular shift of potassium and phosphate by blunting the hyperadrenergic stimulation of Na+/K+–ATPase.[20] Hence, initial therapy for stable TPP should include propranolol.[21,22,23] The definitive therapy for TPP includes treatment of hyperthyroidism with antithyroid medications, surgical thyroidectomy, or radioiodine therapy.

Competing Interests
None Declared
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