RESEARCH PAPER | Medical Science | Volume : 5 | Issue : 12 | December 2015 | ISSN - 2249-555X

Hyperthyroid Hypokalemic Periodic Paralysis in an Indian Male

KEYWORDS

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ABSTRACT *A case of hyperthyroid periodic paralysis in an Indian male is reported. He presented with complete paralysis below the waist and his admission potassium of 1.8 mEq/L. Correction of the hypokalemia resolved his symptoms. Thyroid function tests revealed the presence of hyperthyroidism which was managed medically. The pathophysiology of hyperthyroid hypokalemic periodic paralysis is discussed. Hypokalemic periodic paralysis is a condition occasionally associated with thyrotoxicosis, predominantly in Japanese and Chinese but sporadically found in other races. We report the diagnosis of hyperthyroid periodic paralysis in an Indian male who had a very low serum potassium level due to this disorder.*

CASE REPORT:

A 42-year-old male awoke with diffuse, total paralysis below the level of the waist with bilateral finger tingling. He had one episode of emesis before being transported to our center. He acknowledged that 2 weeks prior to admission he had an episode of weakness much less severe and only lasting 30 minutes, and admitted to feeling tired over the past several days. The evening prior to admission the patient had eaten a meal consisting mainly of khichdi and vegetables. The patient's past medical history was otherwise unremarkable. He denied medication use, smoking or ethanol abuse, and denied any family history of weakness, paralysis, low potassium, or thyroid disorders. There was no history of heart or lung disorders. The man's vital signs were pulse 68 per minute, blood pressure 130/60 mmHg and respirations 24 per minute. He was afebrile. He was only able to raise his extremities slightly against gravity, which was actually improved from earlier. The right lower lobe of the thyroid was enlarged compared to the remainder of the gland and had a bruit on auscultation. There was no opthalmopathy. The lung fields were clear. The heart rate was irregularly irregular. There was no third or fourth heart sound. The abdomen was benign. He was areflexic. The cranial nerve and sensory exams were normal.

The admission potassium was 1.8 mEq/L. His other electrolytes, calcium and complete blood count were normal. His urine potassium was 17 mEq/L. The electrocardiogram showed atrial fibrillation with prominent U-waves and a prolonged QT interval of 0.48 to 0.52 sec. The patient was given intravenous potassium boluses followed by oral potassium in the intensive care unit. Within a few hours the patient had regained nearly complete recovery of his strength and reflexes in all extremities and converted to a normal sinus rhythm. His potassium level improved and remained normal. Thyroid function tests were triiodothyronine 306 ng/dL (normal 80 to 160), thyroxine 19.4 ng/dL (normal 5 to 12), and thyroid-stimulating hormone 2.4 [tU/mL (normal 0 to 6). An iodide 123 thyroid uptake nuclear medicine scan showed a 2 hour thyroid uptake of 21% (normal less than 8%) and a 24 hour uptake of 49% (normal less than 33%) consistent with hyperthyroidism. Propranolol and methimazole were begun in the hospital with propylthiouracil successfully used on an outpatient basis to manage the hyperthyroidism. No further episodes of hypokalemia or paralysis have been noted.

DISCUSSION:

Hyperthyroid hypokalemic periodic paralysis and familial hypokalemic periodic paralysis are separate disease entities. The familial variety is passed as a dominant trait with its sex dependent variable penetrance accounting for its increased prevalence in males compared to females (3:1). The attacks in the familial and non -familial varieties can be precipitated by rest, carbohydrate loading, cold, and various medications including insulin. Several distinctions make hyperthyroid periodic paralysis an entirely different disease from familial periodic paralysis with concurrent hyperthyroidism:

1. Patients with hyperthyroid periodic paralysis tend to be older at the time of presentation (3rd decade) compared with familial periodic paralysis.
2. Hyperthyroid periodic paralysis is cured by treatment of the hyperthyroidism.
3. A positive family history is rare in hyperthyroid periodic paralysis.
4. Of 500 cases reported in the literature over 90% of patients with hyperthyroid periodic paralysis are Oriental.
5. The male-female ratio is remarkably high in hyperthyroid periodic paralysis (over 6:1 compared to 3:1 in periodic paralysis).
6. Acetazolamide is effective in the prophylaxis of familial periodic paralysis but is ineffective for, and may even worsen hyperthyroid periodic paralysis.

PATHOPHYSIOLOGY

The diffusion potential for potassium (EK +) across the cell membrane is approximately -97 mV, which approximates the normal resting skeletal muscle membrane potential (Em) of -87 mV. During an attack of hypokalemic periodic paralysis, there is an intracellular flux of potassium. If this were the complete mechanism, one would expect Em to be more negative, ie, hyperpolarized. However, during an attack of hypokalemic periodic paralysis the Em is actually depolarized to -50 mV making it unable to sustain an action potential. An additional pathophysiological response based on the sodium-potassium (Na+-K+) pump is pro-

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posed. Hyperthyroidism induces adrenergic hypersensitivity to catecholamines, probably by increasing the number of B receptors in skeletal muscle cell membranes. Adrenergic activity stimulates the Na+-K+ pump activity and increases the number of the Na+-K+ pump sites. The first proposed step in pathophysiologic process is an elevated Na+-K+ permeability ratio. That Na+ permeability is elevated in thyrotoxicosis is supported by human skeletal muscle biopsies in hyperthyroidism (irrespective of periodic paralysis) showing twice normal intracellular sodium concentrations. The elevated Na+-K+ permeability depolarizes the cell membrane but the hyperpolarizing effect of increased Na+-K+ pump activity, augmented by the hyperthyroidism, stabilizes Em. Hypokalemia results from the intracellular potassium shift. Na+-K+ pump activity and its hyperpolarizing effect is then reduced and the skeletal muscle cell Em depolarized to non excitable levels. That carbohydrate loading precipitates attacks and mild exercise prevents attacks fits the model well. Insulin production stimulated by carbohydrate loading stimulates the Na+-K+ pump. The action potentials of exercised skeletal muscle raise intracellular sodium and extracellular potassium, delaying or averting shutdown of the Na+-K+ pump. The lack of activity during sleep, as well as the diurnal variation in potassium flux accounts for the usual onset of attacks at night. Propranolol is effective in treatment of hyperthyroid hypokalemic periodic paralysis probably due to its inhibition of the Na+-K+ pump more so than its inhibition of insulin secretion. That over 90% of cases are found among Orientals implies that genetic factors may be involved in hyperthyroid periodic paralysis. The disorder is suggested to be a latent genetic disease unmasked by hyperthyroidisml6'19 and human leukocyte antigen haplotypes A2Bw22, Awl9B17, and DRw8 have been associated with thyrotoxic periodic paralysis. Our patient is most unusual in that he has no Oriental background.

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