

Thyrotoxic periodic paralysis, and high carbohyrdate diet; an unusual presentation in a Caucasian male Rahat Ali Tauni, Rameez Arif, Shiu-Ching Soo

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The clinical case

A 54 year old Caucasian male presented with progressive muscle weakness leading to complete paralysis in upper and lower limbs evident on examination. Initial venous gas analysis showed potassium of 2.1 mmol/L, and ECG showed U waves with prolonged QTc as shown below:



<u>Clinical manifestations of Thyrotoxicosis</u>





A diagnosis of hypokalaemic periodic paralysis was made, and intravenous potassium administered. The paralysis gradually resolved over the next 3-4 hours, as repeat serum potassium level rose to 4.0 mmol/L.

He described 6 month history of episodic weakness occuring almost daily after exercise, resolving spontaneously with rest. He was a former professional bodybuilder and maintained a very active lifestyle through weight lifting at the gym and regular aerobic exercise. He consumed unusually high calorific and highcarbohydrate diet, often with high glycaemic index foods. Further assessment by an endocrinologist 3 days later revealed a history of tremor, weight loss despite high appetite, sweating, and family history of thyroid disease. On examination, he was found to have tremors and lid lag. Biochemistry confirmed Graves' thyrotoxicosis with following results:

Investigations	Results	Normal Limits
TSH	<0.01	0.27-4.20

fT4	59	12-22
TSH receptor antibody	19.3	<1.4
Anti TPO antibody	348	<34

He was advised to consume low carbohydrate and low glycaemic index diet, and was commenced on propranolol and carbimazole. He noticed instant improvement in weakness, and had no further paralysis for a week in hospital. He is now under endocrine clinic follow up and has not had any episodes of weakness for more than a year.

Pathogenesis

TPP is a complex disorder, leading to muscle paralysis through variety of mechanisms. Blood sugar rises in response to food intake and pancreas produces insulin to move glucose into the tissues. It is postulated that an exaggerated insulin response is triggered in such patients causing glucose and potassium (K+) to enter the muscle tissues. This reduction in serum potassium (K+) is suspected of causing the muscle fatigue and weakness as experienced by patients with hypokalaemic periodic paralysis.



Discussion and learning points

Thyrotoxic periodic paralysis (TPP) is most commonly seen in Asian men. Although familial hypokalaemic periodic paralysis can be seen in Caucasians, TPP is exeptionally rare in Caucasian men and this case is an example of the latter. Treatment is with antithyroid drugs, beta blockers and dietary modifications, and leads to immediate relief of symptoms. Treatment with IV potassium in acute setting is essential to prevent potentially life threatening cardiac arrythmias. Raising awareness among general physicians about early detection of thyrotoxicosis in all patients of periodic paralysis may improve patient outcomes. This case also highlights that unusually high carbohydrate diet can often precipitate paralysis, and physicians should evaluate dietary habits while establishing the diagnosis and formulating management plan.

<u>References</u>

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