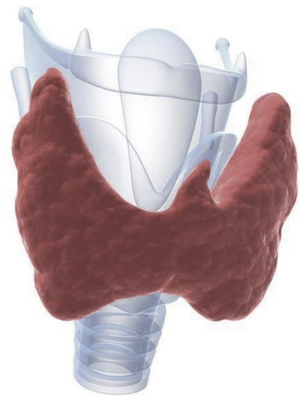

“Off legs” in a 30 year old: a Grave concern?

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Presentation

A 30 year old gentleman of South Asian descent presented with recurrent episodes of sudden onset limb weakness. He had a 9 year history of Graves’ disease and had recently discontinued carbimazole therapy in preparation for radio-iodine treatment.

He reported numerous similar episodes over the past 3 years. Each episode resolved within 2-3 hours and he had fully normal mobility in between episodes. The episodes occurred in the morning and when he was not euthyroid. He denied sensory, visual or bulbar symptoms.

As an outpatient, he had been extensively investigated by the neurology team for these episodes of weakness, with normal head and whole spine Magnetic Resonance Imaging and Nerve Conduction studies.

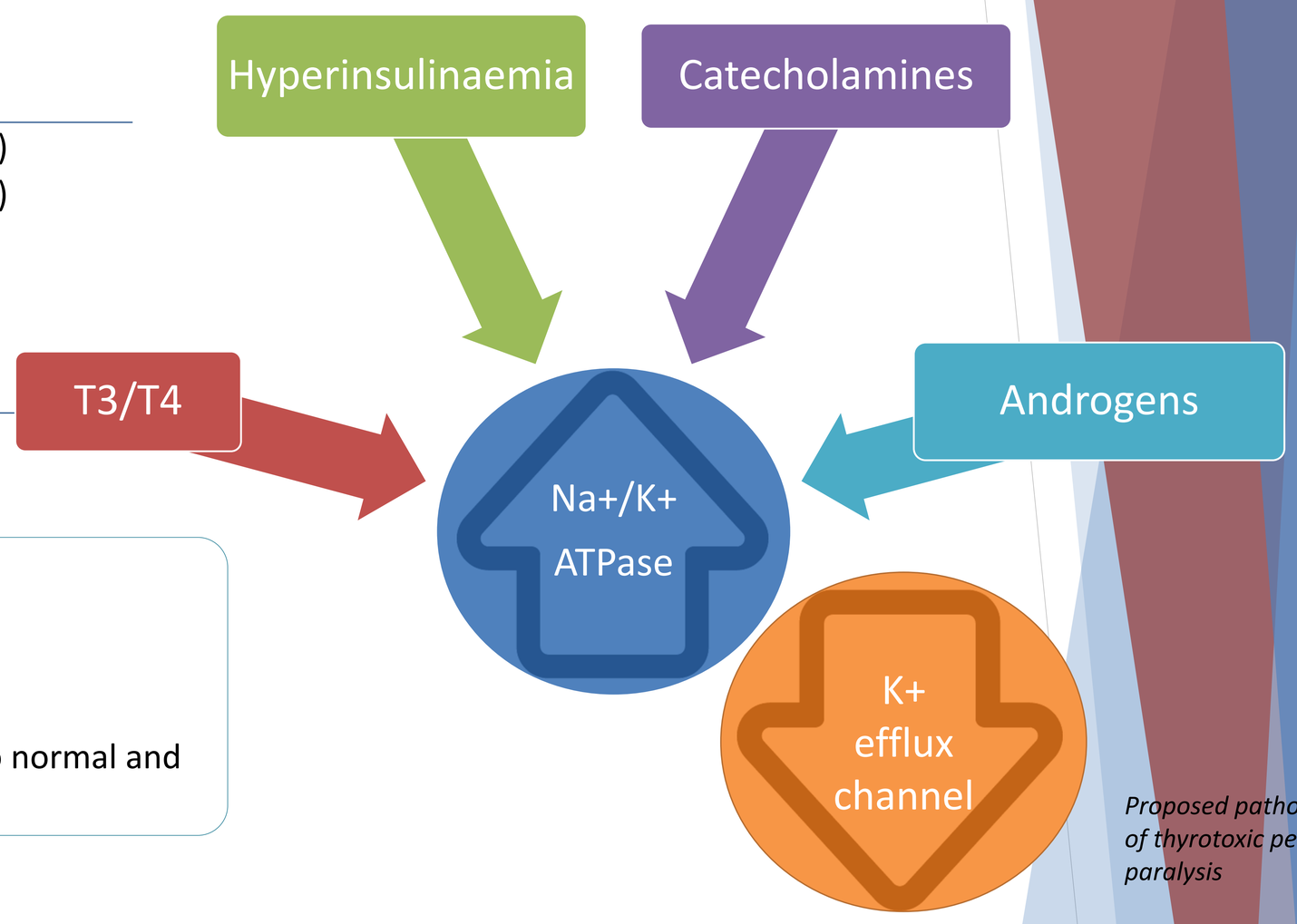
On examination, he had reduced power in all four limbs, most noticeably in the proximal limbs. Reflexes were preserved.

Blood results

• Potassium	2.1	(3.5-5.4 mmol/L)
• Phosphate	0.44	(0.8-1.4 mmol/L)
• Free T3	44	(10-24 pmol/L)
• Free T4	40.4	(4-8.3 pmol/L)

ECG

- Sinus tachycardia



Proposed pathogenesis of thyrotoxic periodic paralysis

Management

- Intravenous and oral potassium replacement
- Propanolol
- Carbimazole
- Discharged when serum potassium returned to normal and symptoms resolved

Discussion

Thyrotoxic periodic paralysis (TPP) is a rare complication of thyrotoxicosis, most commonly seen in Asian populations with an incidence of around 2% in thyrotoxic patients[1]. It is characterised by acute hypokalaemia and muscle paralysis, usually triggered by carbohydrate-laden meals, alcohol or vigorous exertion[2].

The pathogenesis is thought to be a complex interplay between increased Na+/K+ ATPase activity and reduced potassium efflux from cells leading to decreased serum potassium levels without a total body deficit[2]. Thyroid hormones increase Na+/K+ ATPase activity directly and indirectly through sensitising tissues to catecholamine action[3]. Hyperthyroid patients with hyperinsulinaemia have been observed to have a greater incidence of TPP[4] which is likely due to insulin action upon Na+/K+ ATPase receptors. In addition, due to the greater incidence in men and observed increase in Na+/K+ ATPase activity in animal studies, testosterone is considered to play a role in TPP pathogenesis[5].

The aim of treatment in the acute setting is replacement of potassium to prevent potentially fatal complications e.g. cardiac arrhythmias. Ultimately, the aim is to correct the underlying hyperthyroidism.

Conclusions

This case highlights this lesser considered feature of thyrotoxicosis and is a reminder that an atypical presentation can delay diagnosis, which can be frustrating for the patient and the clinician. This patient was seen by neurologists and had multiple admissions before the correct diagnosis was considered. Careful attention to the patient’s description of their symptoms as well as comorbidities may aid earlier diagnosis. It is also important to be aware that in some patients, rapid elevation of thyroid hormones can occur once carbimazole is stopped prior to radioiodine, pre-disposing an episode of paralysis.

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