

The night when the floppy Chinese lad almost died because of his thyroid

Introduction

- Thyrotoxic period paralysis (TPP) is a rare endocrine emergency associated with hyperthyroidism that needs urgent treatment.
- Prompt correction of hypokalaemia and definitive treatment of thyrotoxicosis are the mainstay of treatment of such cases. We present a case of young Chinese lad with similar presentation.

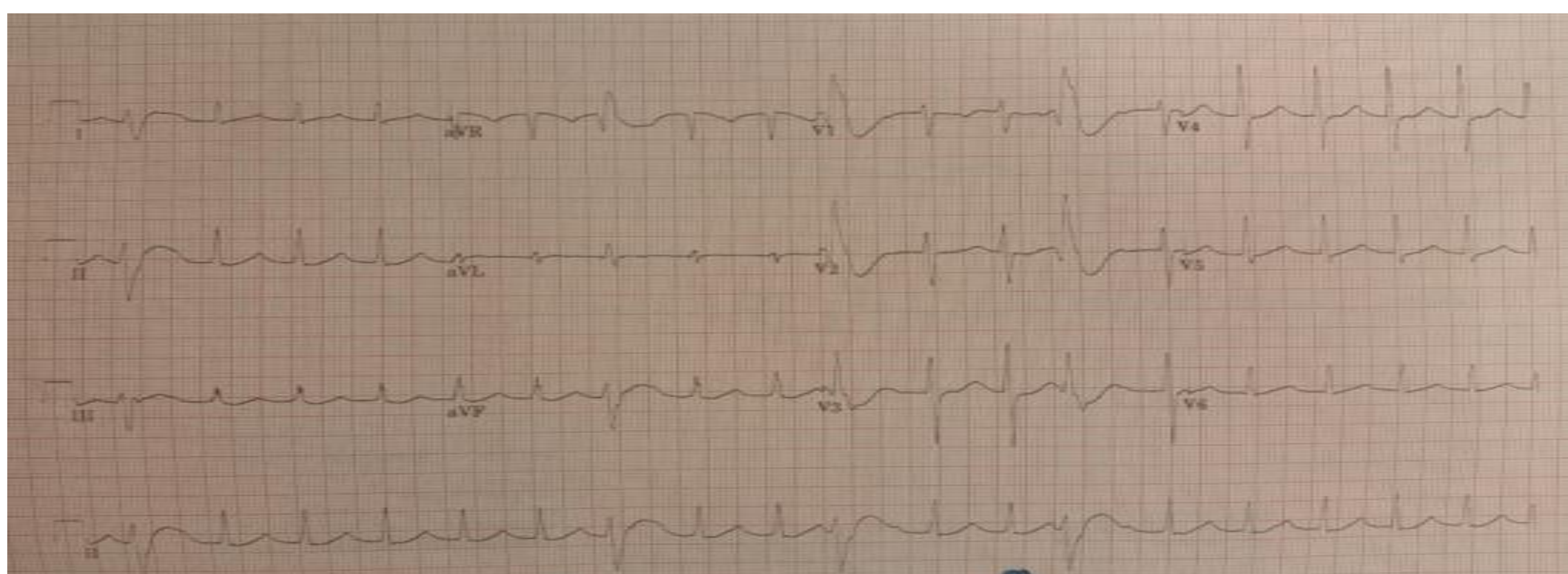
Case Report

A 19 year old healthy Chinese student presented with rapidly progressing lower limb weakness developing overnight. He was resident at the students' hostel and went to sleep at night after a Chinese meal. He couldn't get up from his bed in the morning. Luckily his friend called the ambulance and brought him in hospital.

He was completely flaccid with a power of 1 out of 5 (on MRC scale) in all four limbs. There was no family history of paralysis but the patient had noticed a similar milder episode of self resolving leg weakness 2 months ago. Clinically he was euthyroid.

Relevant investigations

- Serum potassium (K^+) 1.7 mmol/L
- ECG showed junctional tachycardia with long QTc (630ms)
- Thyroid functions test
 - TSH < 0.01 mU/L,
 - FT4 > 100 pmol/L,
 - FT3 = 35.8 pmol/L



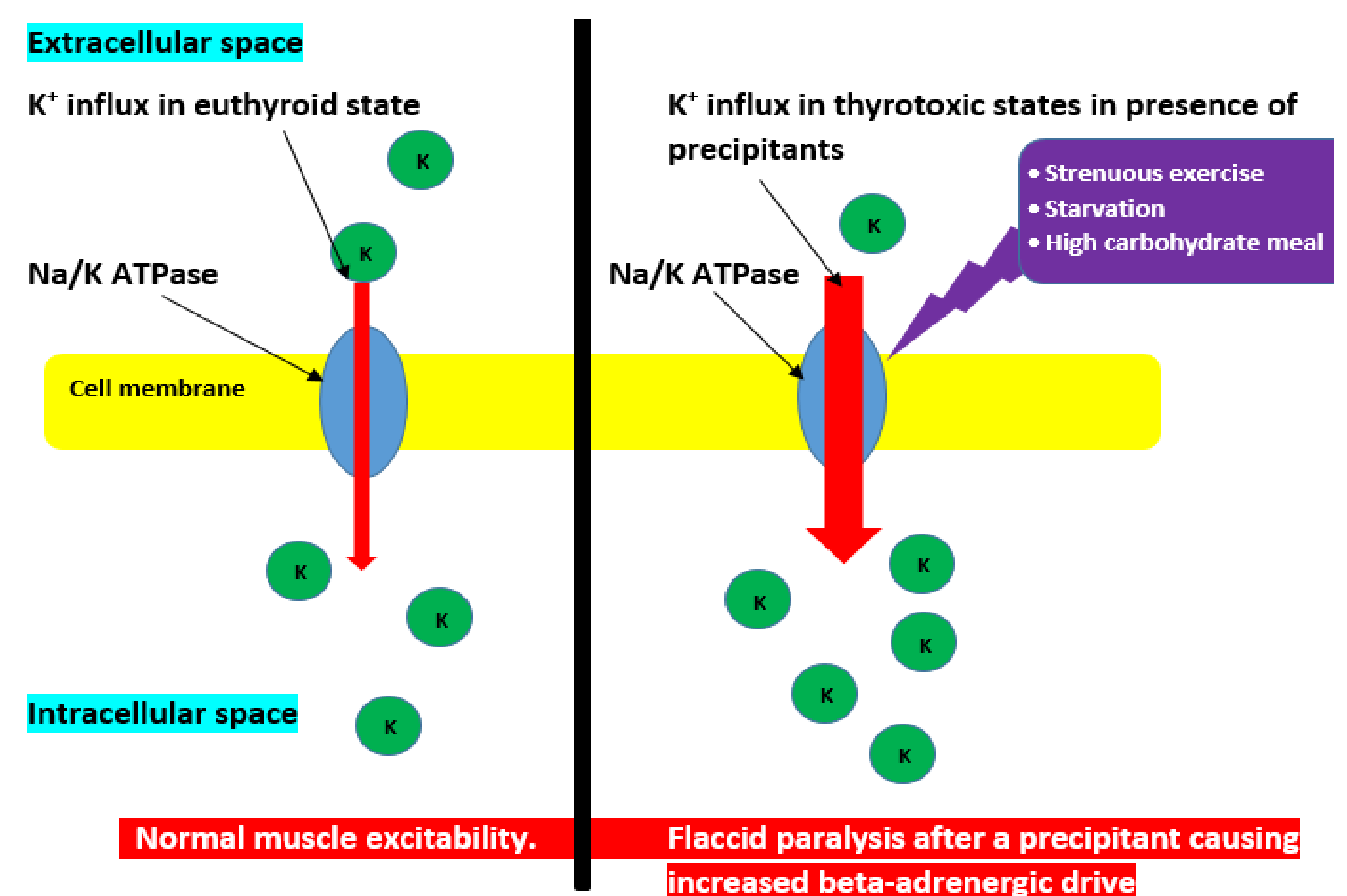
Patient received the following treatment

- Patient was kept on cardiac monitoring
- Hypokalaemia was corrected with intravenous KCL given @ 10mmol/hour. There was complete recovery of the flaccid paralysis within 4 hours of correction of hypokalaemia.
- He was also commenced on propranolol (40mg TDS) and carbimazole (20mg BD). Definitive treatment with radioiodine is planned after achieving euthyroidism with carbimazole.

Case Discussion

Thyrotoxic periodic paralysis is rare (0.2 %) in non Asian population but can present in 8-13 % of Asian men with thyrotoxicosis. It is a sporadic form of hypokalaemic periodic paralysis belonging to a group of muscle diseases called channelopathies.

The exact mechanism is not fully understood. Certain genetic mutations (Kir2.6) in the sodium potassium ATPase channels have been identified making them extra responsive. The channel functions normally in euthyroid states but in presence of thyrotoxicosis the intracellular potassium shift is enhanced by presence of beta-adrenergic or insulin stimulus. This leads to profound hypokalaemia and inexcitable muscle fibres causing flaccid paralysis. So any stimulus increasing beta-adrenergic drive or insulin levels will cause hypokalaemic paralysis in these cases like excessive exercise, starvation or high carbohydrate meal.



Conclusion

Both thyrotoxicosis and hypokalaemia have arrhythmogenic potential. Individuals can be incapacitated in acute setting as a result of rapidly progressive flaccid paralysis due to profound hypokalaemia. Prompt identification in high risk individuals (**young Asian men**) is important to ensure definitive treatment is offered.

Acute flaccid paralysis resolves rapidly after correction of hypokalaemia. Precipitants (**strenuous exercise, high carbohydrate meal and starvation**) should be avoided until rendered euthyroid with carbimazole. Due to risk of recurrence all cases of TPP must be offered curative treatment of thyrotoxicosis with either **thyroidectomy or radioiodine**.