Thyrotoxic periodic paralysis and Migration: An Emerging Diagnosis in Western Countries

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Objectives

Thyrotoxic periodic paralysis (TPP) is a rare neurological emergency that mostly occurs in Asian male patients with thyrotoxicosis and is characterized by episodes of acute flaccid paralysis and hypokalaemia. The aim of this report is to describe a clinical case of TPP, analysing its clinical, diagnostic, and therapeutic approach, with the goal of raising awareness about early diagnosis and optimal treatment of this reversible but potentially lethal condition in Western Countries.

Materials and Methods

Clinical, laboratory, and instrumental data were collected from a male Chinese patient admitted to the emergency department for acute flaccid paralysis. Blood tests, thyroid imaging and thyroid function tests, HLA typing, and genetic tests were performed to confirm the diagnosis of TPP. Through a detailed medical history, neurological examination, and biochemical confirmation (severe hypokalaemia and thyrotoxicosis), a diagnosis of TPP was made. Treatment included intravenous potassium administration and introduction of antithyroid therapy with methimazole. Follow-up was aimed at functional recovery and relapse prevention.

Test	Result
Serum potassium	1.9 mmol/L (3.5 – 5.0
T4	9.85 ng/dL (0.89 – 1.76
TSH	<0.01 mIU/L 0.55 – 4.78
TSH receptor antibodies	Positive
Antithyroglobulin antibodies	Positive
Electrocardiogram	Normal
Echocardiogram	Normal
CT brain	Normal

Results

A 56-year-old Chinese man with unremarkable neurological personal history presented to emergency department with acute lower limb weakness and serum potassium level of 1.9 mmol/L (normal range 3.5- 5.0). Thyroid tests showed elevated FT4 (9.85 ng/dL -n.r. 0.89-1.76), suppressed TSH (<0.01 mUI/L- n.r. 0.55-4.78), and positive TSH receptor antibodies and antithyroglobulin antibodies. No thyroid medication was ongoing. Electrocardiogram and echocardiogram were normal. Neuroimaging and electromyography were unremarkable. Following intravenous potassium infusion, the patient's strength was fully recovered within 24 hours. Treatment with methimazole was started at the same time. HLA typing showed **HLA-A2 haplotype**, typically associated with TPP in Asian patients, while genetic testing for familial hypokalemic periodic paralysis (SCN and CACNA1S gene mutations) was negative. The patient was discharged in good conditions.

Discussion

TPP remains underdiagnosed in Western countries, leading to a higher risk of improper hypokalemia management and potential rebound hyperkalemia. Treatment includes careful electrolyte correction and management of the thyrotoxic state. The role of genetics, particularly HLA haplotypes, confirms a significant ethnic predisposition.

Conclusion

Due to the severe progression of TPP, timely diagnosis both on clinical signs and biochemical data enables effective management and prevents neurological and cardiac complications. Normalization of thyroid function is fundamental to avoid relapses. Moreover, the increased migration from the Asian continent have made TPP more frequently observed in Western countries, so that this clinical emergency must be considered in the differential diagnosis of neuromuscular disorders and acute muscle weakness.

References

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