

Thyrototoxic periodic paralysis with bone metabolism disruption: A case report

Longying Wang¹, Dong Luo², Dalian Wang³, Ping Li⁴, Yan Liu⁵, Zhilong Liu⁶

Abstract

Thyrototoxic Periodic Paralysis (TPP) is a rare condition associated with hyperthyroidism, particularly in young males. This report presents the case of a 20-year-old male with TPP complicated by decreased bone mineral density (BMD). While TPP is primarily a neuromuscular disorder characterised by hypokalaemia-induced periodic paralysis, this case highlights the less-recognised impact of prolonged hyperthyroidism on bone metabolism, resulting in significant bone loss. The patient was treated with potassium supplementation, antithyroid therapy, and alendronate sodium to address bone metabolism. This case underscores the importance of comprehensive management in TPP, emphasising early diagnosis and incorporation of bone health monitoring to mitigate long-term complications such as osteoporosis.

Keywords: Thyrototoxic Periodic Paralysis; Graves' disease; Hypokalaemia; Bone metabolism disorder.

DOI: <https://doi.org/10.47391/JPM.A.30194>

Introduction

Thyrototoxic Periodic Paralysis (TPP) is an uncommon yet potentially life-threatening condition associated with hyperthyroidism, particularly in young males.¹ Acute episodes of TPP are typically triggered by poorly controlled hyperthyroidism and manifest as hypokalaemia-induced muscle weakness or paralysis, often following physical exertion, alcohol consumption, or excessive carbohydrate intake.² Early diagnosis can be challenging due to symptom overlap with conditions such as familial periodic paralysis and Guillain-Barré syndrome.^{3,4}

^{1,5,6}Department of Laboratory Medicine, Dazhou Integrated Traditional Chinese Medicine and Western Medicine Hospital, Dazhou Second People's Hospital, Dazhou, China; ²Department of Emergency, Dazhou Integrated Traditional Chinese Medicine and Western Medicine Hospital, Dazhou Second People's Hospital, Dazhou, China; ³Department of Ultrasound, Dazhou Integrated Traditional Chinese Medicine and Western Medicine Hospital, Dazhou Second People's Hospital, Dazhou, China; ⁴Center for Medical Imaging, Dazhou Integrated Traditional Chinese Medicine and Western Medicine Hospital, Dazhou Second People's Hospital, Dazhou, China.

Correspondence: Zhilong Liu. e-mail: liuzhilong@gris.org.cn

ORCID ID: 0000-0001-8196-2401

Submission completed: 13-03-2025 **1st Revision received:** 15-08-2025

Acceptance: 18-07-2026

Last Revision received: 17-07-2026

In addition to neuromuscular symptoms, hyperthyroidism exerts systemic effects, significantly impacting cardiovascular, metabolic, and skeletal systems.⁵ Chronic hyperthyroidism leads to increased bone turnover, characterised by enhanced osteoclastic activity and suppressed osteoblastic function, resulting in reduced bone mineral density (BMD) and heightened fracture risk.⁶ Although the association between hyperthyroidism and osteoporosis is well-documented, its specific implications in TPP cases remain underexplored. Given that young males, who are typically less prone to osteoporosis, constitute the majority of TPP patients, addressing this risk is particularly critical.⁷

Despite advancements in the understanding of TPP pathophysiology, management strategies often prioritise acute neuromuscular symptoms, overlooking secondary complications such as skeletal health. This case report highlights the importance of incorporating bone metabolism monitoring into TPP management, emphasising early intervention to mitigate long-term complications.

Case Report

A 20-year-old male presented to the emergency department of Dazhou Integrated Traditional Chinese Medicine & Western Medicine Hospital on July 14, 2024, with acute lower limb weakness, hand tremors, and excessive sweating. He reported similar intermittent episodes over the past six months, each resolving spontaneously. He had no history of hypertension, familial periodic paralysis, or medication use. Physical examination revealed mild thyroid enlargement, reduced lower limb muscle strength, and stable vital signs (blood pressure: 126/80 mmHg; heart rate: 99 beats per minute).

Laboratory findings showed significant hypokalaemia (serum potassium: 2.10 mmol/L), mild elevation of sodium (144.6 mmol/L) and chloride (108.3 mmol/L), and normal calcium, magnesium, and phosphate levels. Thyroid function tests revealed elevated free triiodothyronine (FT3: 41.30 pmol/L) (n= 12.60-21.00 pmol/L) and free thyroxine (FT4: >100 pmol/L) (n=12.60-21.00 pmol/L) levels, with suppressed thyroid-stimulating hormone (TSH: <0.005 uIU/mL) (n= 0.51-4.17 uIU/ml). Thyroid autoantibody tests demonstrated elevated thyroglobulin antibody (Anti-TG: 189 IU/mL) (n=<115.00 IU/mL), thyroid peroxidase

Table-1: Laboratory Test Parameters of the Patient's Serum.

Parameters	Value	Changes	Reference
K	2.1	↓↓	3.50-5.30 mmol/L
Na	144.6	↑	136.0-144.0 mmol/L
Cl	108.3	↑	96.0-108.0 mmol/L
Ca	2.34		2.10-2.70 mmol/L
Mg	0.72		0.62-1.30 mmol/L
P	1.46		0.80-1.60 mmol/L
FT3	41.30	↑	3.94-7.67 pmol/L
FT4	>100	↑↑	12.60-21.00 pmol/L
TSH	<0.005	↓↓	0.51-4.17 uIU/ml
Anti-TG	189.00	↑	<115.00 IU/mL
TPO-Ab	343.00	↑↑	<34.00 IU/mL
TRAb	29.80	↑↑	<1.75 IU/L
CK	662	↑↑	25-200 U/L
CK-MB	30	↑	7-24 U/L
cTnI	2.10		<34.1 ng/L
Myo	225.31	↑↑	0-110 ng/mL
N-MID	93.200	↑↑	6.000~24.660 ng/ml
PINP	793.000	↑↑	9.060~76.240 ng/ml
β-CTX	2.220	↑	0.043~0.783 ng/ml

K: Potassium; Na: Sodium; Cl: Chloride; Ca: Calcium; Mg: Magnesium; P: Phosphorus; FT3: Free Triiodothyronine; FT4: Free Thyroxine; TSH: Thyroid Stimulating Hormone; Anti-TG: Anti-Thyroglobulin Antibodies; TPO-Ab: Thyroid Peroxidase Antibodies; TRAb: Thyroid-stimulating hormone receptor antibody; CK: Creatine Kinase; CK-MB: Creatine Kinase-MB; cTnI: Cardiac Troponin I; Myo: Myoglobin; N-MID: N-terminal Mid-fragment of Osteocalcin; PINP: Procollagen Type I N-terminal Propeptide; β-CTX: Beta-CrossLaps. ↑ indicates a mild increase, ↑↑ indicates a significant increase, and ↓ is used similarly to indicate a decrease.

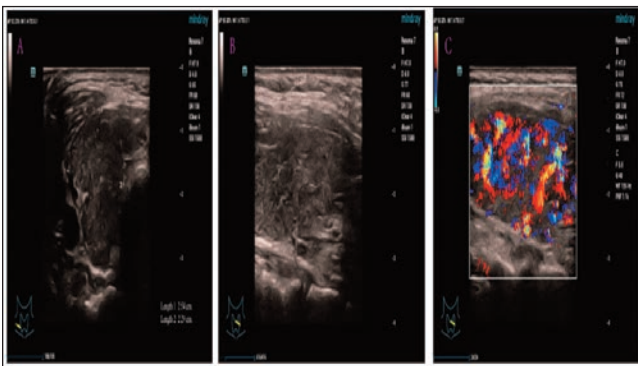


Figure-1: (A) The thyroid gland is enlarged, measuring 2.54 x 2.29 cm. (B) The thyroid parenchyma exhibits heterogeneous echogenicity. (C) The thyroid parenchyma shows increased blood flow.

Table-2: DXA Scan Results of the Patient.

	BMD (g/cm ²)	Z score
Lumbar spine L1	0.882	-2.18
Lumbar spine L2	0.870	-2.30
Lumbar spine L3	0.952	-1.48
Lumbar spine L4	0.931	-1.69
Left total hip	0.863	-1.37
Right total hip	0.859	-1.41

DXA scan: Dual-energy X-ray Absorptiometry; BMD: Bone Mineral Density. The Z-score reflects the patient's bone density in comparison to the same age, gender, and ethnic group. Normal bone density: Z-score close to 0; Low bone mass or osteoporosis: Z-score < -2.0.

antibody (Anti-TPO: 343 IU/mL) (n=<34.00 IU/mL) and Thyroid-stimulating hormone receptor antibody (TRAb: 29.80 IU/mL)(n=<1.75 IU/L), (Table 1).

Thyroid ultrasound revealed bilateral thyroid enlargement with heterogeneous echotexture and increased blood flow, consistent with Graves' disease (Figure 1). Cardiac biomarkers showed elevated creatine kinase (CK: 662 U/L) and mildly elevated creatine kinase-MB (CK-MB: 30 U/L) and myoglobin (Myo: 225.31 ng/mL), indicating skeletal muscle injury. Bone metabolism markers, including N-terminal midfragment of osteocalcin (N-MID: 93.20 ng/mL), procollagen type I N-terminal propeptide (PINP: 793.00 ng/mL), and beta-CrossLaps (β-CTX: 2.22 ng/mL), were significantly elevated, indicating increased bone turnover (Table 1). Dual-energy X-ray Absorptiometry (DXA) revealed reduced BMD with a Z-score of -2.18 at the lumbar spine L1, consistent with low bone mass (Table 2).

The patient was diagnosed with TPP complicated by decreased BMD. Treatment included intravenous potassium supplementation to correct hypokalaemia, methimazole (20 mg/day) to manage hyperthyroidism, and alendronate sodium (70 mg/week) to inhibit bone resorption. After one week of treatment, the patient's paralysis episodes resolved. At a three-month follow-up, his potassium levels, thyroid function, and BMD had significantly improved.

Discussion

This case illustrates the dual impact of hyperthyroidism on the neuromuscular and skeletal systems in TPP. The hallmark of TPP is hypokalaemia, which disrupts the electrical potential across muscle membranes, leading to paralysis. In this case, severe hypokalaemia (2.10 mmol/L) was the primary trigger for the patient's symptoms. Elevated CK levels confirmed skeletal muscle injury, consistent with acute TPP episodes.⁶

Hyperthyroidism, particularly in Graves' disease, exacerbates bone metabolism disruption through enhanced osteoclastic activity and suppressed osteoblastic activity.⁷ Elevated thyroid hormones accelerate bone turnover, as evidenced by increased levels of N-MID, PINP, and β-CTX in this patient. These findings align with previous studies linking hyperthyroidism to reduced BMD and increased fracture risk.^{8,9} Chronic hyperthyroidism also alters calcium and phosphate homeostasis, further contributing to skeletal fragility.¹⁰

Management of TPP requires a comprehensive approach. While potassium supplementation addresses acute hypokalaemia, antithyroid therapy is critical for preventing recurrent episodes. However, this case underscores the

importance of addressing secondary complications, particularly those related to skeletal health. Young males with TPP are at a paradoxical risk of developing low bone mass or osteoporosis due to prolonged untreated hyperthyroidism. Early initiation of bisphosphonate therapy, as demonstrated in this case with alendronate sodium, can significantly reduce bone resorption and improve BMD.⁶

The findings from this case support the integration of skeletal health assessments into routine TPP management. This includes periodic DXA scans, monitoring of bone turnover markers, and consideration of adjunctive therapies, such as calcium and vitamin D supplementation, particularly for patients with prolonged hyperthyroidism.⁸ Addressing bone health in TPP not only prevents long-term complications but also improves overall patient outcomes by providing a more holistic treatment approach.

Conclusion

This case highlights the importance of comprehensive management in TPP, including correction of hypokalaemia, control of hyperthyroidism, and monitoring of bone health. Early intervention in bone metabolism can prevent long-term complications, underscoring the need for an integrated treatment approach in TPP.

Patient Consent: Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

Disclaimer: None.

Conflict of interest: None.

Funding disclosure: None.

References

1. Fralick M, Sarma S. Thyrotoxic Periodic Paralysis. *N Engl J Med* 2021;384:e71. doi: 10.1056/NEJMcm2030770.
2. Wiersinga WM, Poppe KG, Effraïmidis G. Hyperthyroidism: aetiology, pathogenesis, diagnosis, management, complications, and prognosis. *Lancet Diabetes Endocrinol* 2023;11:282-98. doi: 10.1016/S2213-8587(23)00005-0.
3. Holm-Yildiz S, Witting N, Dahlqvist J, de Stricker Borch J, Solheim T, Fornander F, et al. Permanent muscle weakness in hypokalemic periodic paralysis. *Neurology* 2020;95:e342-52. doi: 10.1212/WNL.0000000000009828.
4. Shahrizaila N, Lehmann HC, Kuwabara S. Guillain-Barré syndrome. *Lancet* 2021;397:1214-28. doi: 10.1016/S0140-6736(21)00517-1.
5. Deshmukh H, Papageorgiou M, Aye M, England J, Abdalla M, Sathyapalan T. Hyperthyroidism and bone mineral density: Dissecting the causal association with Mendelian randomization analysis. *Clin Endocrinol (Oxf)* 2021;94:119-27. doi: 10.1111/cen.14330.
6. Smith TJ, Hegedüs L. Graves' Disease. *N Engl J Med* 2016;375:1552-65. doi: 10.1056/NEJMra1510030.
7. Lin SH, Huang CL. Mechanism of thyrotoxic periodic paralysis. *J Am Soc Nephrol* 2012;23:985-8. doi: 10.1681/ASN.2012010046.
8. Diedrich DA, Wedel DJ. Thyrotoxic periodic paralysis and anesthesia report of a case and literature review. *J Clin Anesth* 2006;18:286-92. doi: 10.1016/j.jclinane.2005.08.016.
9. Madurga A, Ceacero-Marín D, González-Peiro C. Thyrotoxic periodic paralysis: a case report and review of relevant pathophysiology. *Emergencias* 2024;36:230-1. doi: 10.55633/s3me/025.2024.
10. Seshadri P, Frank KD, Iqbal N. Thyrotoxic hypokalemic periodic paralysis in a Native American patient: case report and literature review. *Endocr Pract* 2002;8:362-3.

Author Contribution:

LW: Data analysis, interpretation and writing.

DL: Design, coordination and data analysis.

DW: Performed thyroid ultrasound to assess thyroid morphology and blood flow.

PL: Conducted DXA scans to evaluate bone mineral density.

YL: Laboratory data collection and analysis.

ZL: Comprehensive analysis of clinical data, writing and revision.