Case Report

# THYROTOXIC PERIODIC PARALYSIS IN A COMPETITIVE BODYBUILDER WITH THYROTOXICOSIS FACTITIA

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## ABSTRACT

***Objective:*** We report a case of thyrotoxic periodic paralysis (TPP) in a bodybuilder who developed paralysis secondary to thyrotoxicosis factitia after taking a supplement containing thyroid hormone. Interestingly, the patient had no intrinsic thyroid disease. Prompt recognition of thyrotoxicosis is critical to avoid progression of paralysis and subsequent complications.

***Methods:*** We discuss a 27-year-old body builder who presented after a 3-day bodybuilding competition with sudden upper and lower extremity paralysis. He admitted to taking anabolic steroids, a supplement containing an unknown amount of thyroid hormone for 2 weeks, and furosemide 40 mg twice daily with near-complete fluid restriction for 3 days.

***Results:*** Laboratory results showed a thyroid-stimulating hormone (TSH) level of <0.010 μIU/mL (normal, 0.3 to 5.8 μIU/mL), normal total triiodothyronine level, elevated free thyroxine level of 3.6 ng/dL (normal, 0.8 to 1.9 ng/dL), and potassium level of 1.9 mEq/L (normal, 3.7 to 5.2 mEq/L). Thyroid peroxidase antibody, thyroid-stimulating immunoglobulin, and thyroglobulin antibody levels were normal. Thyroid uptake was 1% (normal, 8 to 25%) after administration of I-123 and thyroglobulin level was 9 ng/mL (normal, 1.4 to 29.2 ng/mL). The patient was treated with normal saline infusion, magnesium supplementation and a total of 230 mEq of potassium within 12 hours of hospitalization. Muscle weakness resolved within this time period and potassium level normalized. By the third day of hospitalization free thyroxine level also normalized and TSH improved to 0.1 mIU/L.

***Conclusion:*** TPP is a rare complication of thyrotoxicosis that should be considered in bodybuilders who are presenting with acute muscle weakness. **(AACE Clinical Case Rep. 2020;6:e252-e256)**

**Abbreviations:**
**$Na^+$-$K^+$ATPase** = sodium-potassium adenosine triphosphatase; **T3** = triiodothyronine; **T4** = thyroxine; **TF** = thyrotoxicosis factitia; **TPP** = thyrotoxic periodic paralysis

## INTRODUCTION

Thyrotoxic periodic paralysis (TPP) is an acute life-threatening complication of thyrotoxicosis characterized by transient muscle weakness or reversible paralysis of the extremities (1). Paralysis is an effect of hypokalemia induced by the intracellular shift of potassium, which is intensified by thyroid hormone (2). TPP may be caused by common causes of hyperthyroidism, including Graves disease and toxic multinodular goiter. A rare cause of TPP is thyrotoxicosis factitia (TF), or exogenous thyrotoxicosis from surreptitious use of thyroid hormone. TF has been described in patients, including athletes and bodybuilders, who are taking weight-reducing agents containing thyrox-

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ine (T4) or triiodothyronine (T3) for its proposed effect on metabolism and improved performance. While these patients often present only with symptoms of thyrotoxicosis, cases of TPP in patients with TF are less commonly reported, especially in competitive bodybuilders. Only 7 cases of TF induced TPP have been reported. To our knowledge, our case is the first reported case of TPP due to TF in a competitive bodybuilder without any underlying thyroid disease.

## CASE REPORT

A 27-year-old Hispanic man presented with sudden profound weakness and paralysis of the arms and legs 6 hours after a 3-day bodybuilding competition. He reported having palpitations and insomnia for 2 weeks prior to presentation. In preparation for the competition, he admitted to a 3-day history of near-complete fluid restriction and administration of furosemide 40 mg twice daily. He was also administering trenbolone 100 mg daily and unknown doses of testosterone enanthate, boldenone undecylenate, and stanozolol, all of which he reported purchasing at his gym. In addition to these anabolic steroids, he was taking a weight loss supplement containing an unknown amount of thyroid hormone, which was initiated 2 weeks prior to presentation and discontinued 3 days prior to presentation. The patient was seen in consultation by endocrinology. On examination, blood pressure was 193/78 mm Hg, pulse was 109 beats per minute, temperature was 97.9 ºF. He had lid lag, but otherwise thyroid and eye examinations were unrevealing. There was a fine tremor of the hands. Severely decreased strength (2/5) of the upper and lower extremities was evident on physical exam. Laboratory evaluation revealed a suppressed thyroid-stimulating hormone (TSH) level, normal total T3 level, elevated free T4 level, and very low potassium level (Table 1). Urine drug screen was negative. Electrocardiogram showed T wave inversions in the inferior leads and U waves (Fig. 1). Thyroid antibody levels were normal, including thyroid peroxidase antibody level, thyroid-stimulating immunoglobulin, and thyroglobulin antibody level (Table 1). Thyroid uptake was 1% (normal, 8 to 25%) after administration of I-123 and thyroglobulin level was 9 ng/mL (normal, 1.4 to 29.2 ng/mL). A diagnosis of TPP secondary to TF was made by the consulting endocrinologist based upon the clinical presentation, laboratory values, and very low uptake on I-123 thyroid scan.

He received intravenous saline, magnesium and phosphorus supplementation, and a total of 230 mEq of potassium within 12 hours of hospitalization. Muscle weakness resolved within this time period and potassium level normalized to 3.8 mEq/L. No further doses of potassium were given. By the third day of hospitalization, potassium level was 5.1 mEq/L, TSH improved, free T4 normalized and total T3 was slightly low (Table 1). The patient has been lost to follow-up; therefore, repeat thyroid tests after discharge are not available.

## DISCUSSION

Our case describes a young competitive bodybuilder with TPP after 2 weeks of consuming a weight loss supplement containing an unknown amount thyroid hormone. TPP was triggered by significant dehydration from fluid restriction and use of a potassium-excreting diuretic, as well as the hyperadrenergic state associated with thyrotoxicosis. He had no family history of thyroid disorders and antibodies did not indicate underlying autoimmune thyroid disease. A low thyroglobulin level and a very low uptake of 1% on radioiodine scan confirmed the presence of exogenous thyroid hormone. The patient did not have any family history to suggest familial hypokalemic periodic paralysis, which has an autosomal dominant inheritance. He had no other neurologic deficits that would suggest demyelinating or neuromuscular disorders, and he had no exposures or symptoms of infection to suggest acute paralysis from an infectious etiology.

It is unclear whether the supplement contained T4 or a combination of T4 and T3. An elevated free T4 level may indicate exogenous T4 intake; however, T3 ingestion may still be suspected. The half-life of T4 is 7 days, while the half-life of T3 is significantly shorter at 18 hours (3). Our patient stopped taking thyroid hormone 3 days prior to presentation which can normalize T3 levels without elimination of its thyrotoxic effects. Alternately, patients who ingest T4 may in turn have elevated T3 due to the peripheral conversion of T4 to T3 (4).

Patients with TPP usually present with sudden weakness of the extremities and commonly have preceding clinical features of hyperthyroidism (5). This is contrary to the more progressive myopathy that may be seen with other causes of paralysis, such as profound dehydration or electrolyte depletion. Paralysis in TPP is the consequence of severe hypokalemia, which is caused by a mechanism involving sodium-potassium adenosine triphosphatase ($Na^+$-$K^+$ ATPase) channels. In skeletal muscle, thyroid hormone upregulates the translocation of $Na^+$-$K^+$ ATPase channels and promotes insertion of the pump within the cell membrane. These channels are critical in maintaining cellular ion gradients by inducing an intracellular shift of potassium and extracellular shift of sodium (2). The influx of potassium causes a state of hypokalemia, which induces membrane hyperpolarization and excitability. While thyroid hormone levels do not correlate with the severity of paralysis, there is a relationship between the severity of paralysis and the degree of hypokalemia (6). β-adrenergic stimulation induced by hyperthyroidism also increases the sensitivity and number of β-receptors that activate the $Na^+$-$K^+$ ATPase channels, which further exacerbates hypokalemia and paralysis (7).

Treatment of TPP consists of potassium supplementation, though caution should be taken in order to avoid rebound hyperkalemia. Given that hypokalemia is due to an intracellular shift of potassium rather than decreased total potassium stores, studies have suggested replacement should not exceed 90 mEq over a 24-hour period to prevent overcorrection (5). It is important to note that patients on potassium-excreting diuretics, like our patient, may require higher doses of potassium supplementation. Normalization of potassium levels and restoration of a euthyroid state leads to a quick recovery of muscle strength. Withdrawal of exogenous thyroid hormone in cases of TF is critical. Patients who have TPP secondary to endogenous hyperthyroidism benefit from restoration of euthyroidism with

**Table 1**
**Laboratory Values on Presentation and After Treatment**

| | Day 1 | Day 3 | Reference range |
|---|---|---|---|
| Sodium, mEq/L | 143 | 139 | 135-145 |
| Potassium, mEq/L | 1.9 | 5.1 | 3.7-5.2 |
| Chloride, mEq/L | 105 | 100 | 96-106 |
| Bicarbonate, mEq/L | 32 | 26 | 23-30 |
| Blood urea nitrogen, mg/dL | 14 | 12 | 7-20 |
| Creatinine, mg/dL | 0.6 | 0.7 | 0.6-1.2 |
| Glucose, mg/dL | 128 | 256 | <125 |
| Phosphorus, mg/dL | 0.9 | 4.5 | 2.5-4.5 |
| Magnesium, mEq/L | 1.5 | 1.9 | 1.5-2.5 |
| TSH, μIU/mL | <0.010 | 0.100 | 0.3-5.8 |
| Free T4, ng/dL | 3.6 | 1.6 | 0.8-1.9 |
| Total T3, ng/mL | 0.9 | 0.5 | 0.8-1.5 |
| Thyroglobulin, ng/mL | 9 | | 1.4-29.2 |
| Throglobulin antibody, IU/mL | <1.0 | | 0-0.9 |
| Thyroid peroxidase antibody, IU/mL | 7.0 | | 0-34 |
| Thyroid-stimulating immunoglobulin | 36% | | <130% |

Abbreviations: T3 = triiodothyronine; T4 = thyroxine; TSH = thyroid-stimulating hormone.

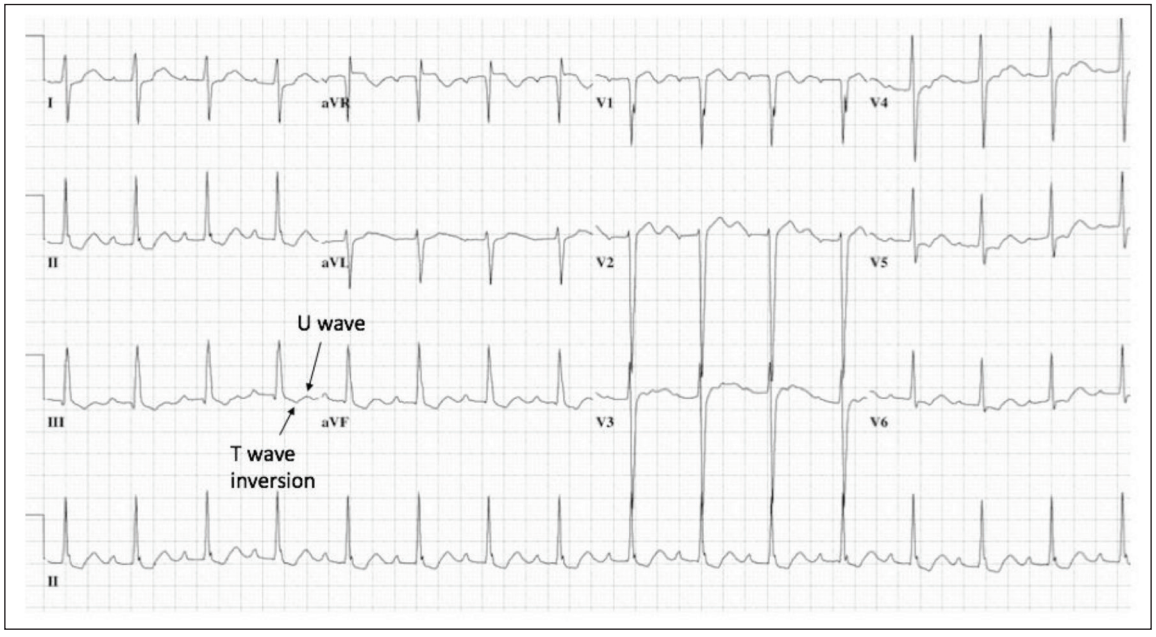


**Fig. 1.** Electrocardiogram of our patient showed T wave inversions and U waves consistent with hypokalemia.

oral antithyroid medications or definitive therapy with surgery or radioactive iodine. A nonspecific beta blocker, such as propranolol, blunts the hyperadrenergic activation of $Na^+$-$K^+$ ATPase channels, which in turn prevents further influx of potassium (8).

There have been 7 reported cases of TPP secondary to TF in the literature so far (Table 2). All 7 cases involved patients between 20 and 40 years old taking weight loss supplements containing thyroid hormone. The few that were named were "Killer Bee's Fat Burner" (4) and "Eltroxine," (9) both containing T4, and "Triax Metabolic Accelerator" (7) which contained tiratricol, a thyroid hormone analogue. The average potassium level on presentation was 1.9 mEq/L, although only 1 other case had changes consistent with hypokalemia on electrocardiogram. Of the 7 cases, 5 were males, despite hyperthyroidism being more common in the female population. This can be explained by the potential role of androgen on $Na^+$-$K^+$ ATPase activity (10), though this concept is still being studied. A genetic predisposition to TPP has also been described in the Hispanic and Asian populations. This is thought to be due to mutations in the genes encoding the inward rectifying potassium channel Kir2.6, a muscle specific channel responsive to thyroid hormone. Variation in this potassium channel modifies skeletal muscle membrane excitability, which has been associated with the development of TPP (11). Our patient was of Hispanic origin, and of the other 7 cases reported, 1 patient was from Latin America and 3 were from Asian descent.

Cohen-Lehman et al (7) discuss the only other case of TPP due to TF in a professional bodybuilder. This patient was taking "Triax Metabolic Accelerator," a weight loss supplement containing a thyroid hormone analogue, for 4 weeks. In contrast to our patient, the patient reported by Cohen-Lehman et al (7) was noted to have thyroid gland enlargement on exam and a family history of Hashimoto disease, indicating undiagnosed underlying autoimmune thyroid disease.

In the weeks leading up to a competition, bodybuilders often use dietary supplements and diuretics to potentiate weight loss and excrete excess fluid. Diuretics have been used for aesthetic purposes to better display muscle tone,

**Table 2**
**Reported Cases of Thyrotoxic Periodic Paralysis Secondary to Thyrotoxicosis Factitia**

| | Age (years) | Place of origin | Source of thyroid hormone | K+ (mEq/L) | TSH<br>Free T4<br>Total T3 |
|---|---|---|---|---|---|
| Akinyemi et al (14) | 35 | Russia | "Diet pills" from Russia | 2.6 | 0.013<br>1.06<br>2.49 |
| Akinyemi et al (14) | 27 | Burma | "Diet pills" from Thailand | 2.0 | 0.025<br>1.31<br>1.38 |
| Chen et al (9) | 36 | Taiwan | Thyroxine for weight loss - "Eltroxine" | 1.6 | 0.003<br>0.10<br>0.74 |
| Chou et al (15) | 23 | Taiwan | Liothyronine for weight loss | 1.6 | <0.005<br>1.5<br>0.76 |
| Panikkath and Nugent (3) | 24 | Latin America | Liothyronine for weight loss | 1.9 | 0.010<br>0.38<br>NA |
| Cohen-Lehman et al (7) | 23 | United States | Tiratricol containing supplement for body building - "Triax Metabolic Accelerator" | 2.6 | <0.010<br>0.10<br>0.32 |
| Cheema et al (4) | 33 | Brazil | T3 and T4 containing weight loss supplement - "Killer Bee's fat burner" | 1.6 | Low<br>0.98<br>NA |
| Current case | 27 | Hispanic | Weight loss supplement | 1.9 | <0.010<br>3.6<br>0.90 |

Abbreviations: K+ = potassium; NA = not applicable; T3 = triiodothyronine; T4 = thyroxine; TSH = thyroid-stimulating hormone. Reference ranges: K+ = 3.7 to 5.2 mEq/L; TSH = 0.3 to 5.8 μIU/mL; free T4 = 0.8 to 1.9 ng/dL; total T3 = 0.8 to 1.5 ng/mL.

muscle striation, and vasculature during performance (12). This often leads to severe electrolyte imbalances and dehydration. Many athletes also use nonaromatizing steroids in conjunction with thyroid hormone to stimulate metabolism. Thyroid hormone enables bodybuilders to consume a high-calorie diet prior to performance without compromising metabolism, and is cycled every few weeks to prevent long-term complications (13). It is important for physicians to consider the possibility of underlying thyrotoxicosis in athletes and bodybuilders presenting with acute muscle weakness.

## CONCLUSION

This report discusses a case of TPP in a bodybuilder caused by surreptitious use of thyroid hormone. We illustrate the risks of taking supplements containing thyroid hormone, and highlight the importance of recognizing the clinical presentation of TPP for early diagnosis and prevention of disease progression.

## DISCLOSURE

The authors have no multiplicity of interest to disclose.

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