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Case Report

Thyrotoxic periodic paralysis complicated by life-threatening acute hypercapnic respiratory failure in a Chinese male with painless thyroiditis

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ABSTRACT

Context: Thyrotoxic periodic paralysis (TPP) is a relatively common complication seen in Asian hyperthyroid patients. However, it is a rare occurrence to find a TPP case comprised of acute hypercapnic respiratory failure in patients with painless thyroiditis.

Patient: A 29-year-old Chinese man presented with flaccid paralysis of all four limbs and he was brought to emergency room. Severe hypokalemia was found on admission. Although treatment had been initiated with potassium chloride supplementation, he went on to develop acute hypercapnic respiratory failure likely due to muscle fatigue. The patient was intubated for mechanical ventilatory support. Once his serum potassium levels were normalized, he was able to be weaned off ventilator support. Thyroid function tests showed elevated free thyroxine concentration and low thyroid-stimulating hormone concentration. He underwent a thyroid uptake scan with ^{131}I which revealed decreased uptake rate of thyroid area. Based on the patient's clinical presentation and associated findings, we diagnosed him with TPP due to painless thyroiditis. We have reviewed TPP cases caused by painless thyroiditis and TPP cases associated with acute hypercapnic respiratory failure.

Conclusion: It is important to note that potentially fatal complications such as acute hypercapnic respiratory failure might occur in acute attacks of TPP even in cases of TPP due to painless thyroiditis.

1. Introduction

Thyrotoxic periodic paralysis (TPP) is characterized by excessive thyroid hormone and profound hypokalemia accompanied by transient weakness of muscles, predominantly in the lower extremities. Some of the TPP attacks could be potentially fatal when respiratory muscles are involved [1-3]. In the present study, we report a rare case of TPP manifesting as acute hypercapnic respiratory failure due to painless thyroiditis.

2. Case report

A 29-year-old Chinese man presented with flaccid paralysis of all limbs and he was brought to emergency room in January 2015. He had no prior similar episodes. His past medical history was unremarkable, and he denied taking any medications. He denied heat intolerance, weight loss, and palpitations.

On arrival, his respiratory rate was 20 breaths/min and oxygen saturation was 99% on room air. Blood pressure was 137/77 mm Hg, heart rate was 96 beats/min, temperature was 36.5 °C, and the patient was alert. On examination, no goiter was observed and the lungs were clear on auscultation. Neurological findings showed cranial nerve and sensory examination were intact, but all four limbs were flaccid and motor strength of each was 1 out of 5. Deep tendon reflexes were absent, and the remainder of systematic examinations were unremarkable.

Laboratory findings showed profound hypokalemia (1.50 mmol/L) and hypophosphatemia (0.58 mmol/L). Intravenous potassium chloride was started at a rate of 20 mmol/h immediately. Ninety minutes later, he complained of chest tightness. Two hours and 50 min after arrival, he was short of breath and was observed to be drowsy, but still conscious. And his oxygen saturation had dropped down to 85–88% on room air. Electrocardiogram indicated sinus tachycardia (heart rate:110 beats/min), prolonged QTc and ST depression. At that time, laboratory findings showed a serum potassium level of 2.10 mmol/L. The arterial blood gas analysis findings were as follows: pH 7.12; pCO_2, 66.5 mm Hg; pO_2, 61.1 mm Hg; and HCO_3^-, 20.4 mmol/L. He was intubated and mechanical ventilation was initiated. His muscle strength progressively improved and was eventually restored. After eight and a half hours of mechanical ventilation, he was able to be weaned off artificial ventilatory support. His serum potassium level had risen to 3.8 mmol/L at that time. He was

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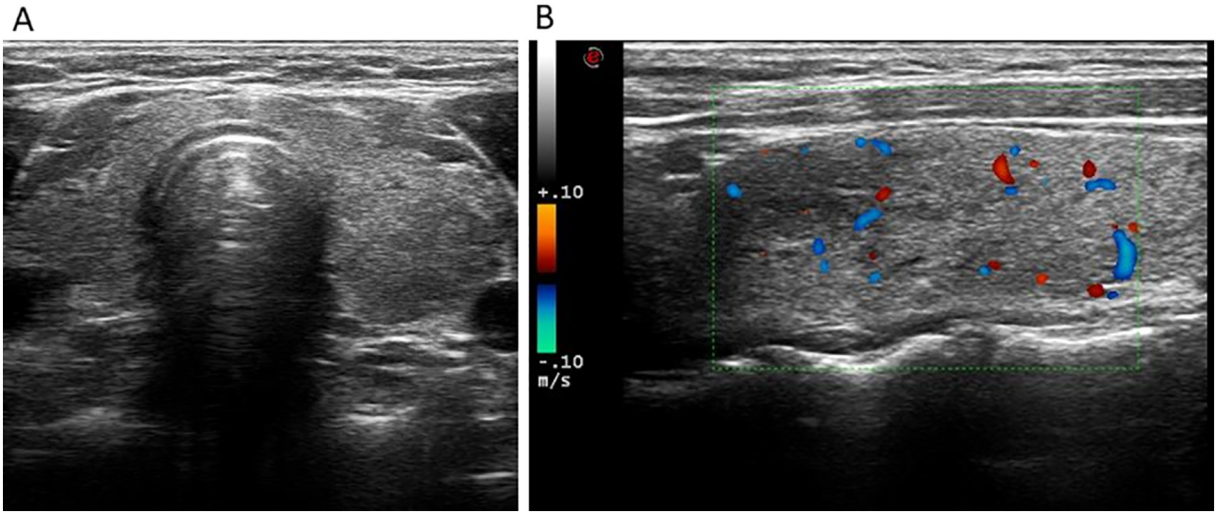

Fig. 1. Ultrasound findings of the thyroid. (A) A gray scale showed a normal sized thyroid gland, and no nodules were detected. (B) Power doppler image revealed decreased vascularity.

then admitted to endocrinology department. His thyroid function tests were as follows: free-thyroxine (FT4) concentration was 15.95 ng/dL (normal range 0.89–1.76 ng/dL), triiodothyronine concentration was 42.1 ng/dL (6.0–18.1 ng/dL), thyroid-stimulating hormone was 0.03 μIU/mL (0.55–4.78 μIU/mL). Anti-thyroglobulin antibody was 11 IU/mL (0–115 IU/mL), and thyroid anti-peroxidase antibody was 15 IU/mL (0–34 IU/mL), antibody of TSH receptor was <0.30 IU/L (0–1.75 IU/L). Three days after the admission, thyroid ultrasound study indicated a normal sized thyroid gland and doppler image revealed decreased vascularity as shown in Fig. 1. And a thyroid uptake scan with 2 μCi of ^{131}I was performed on him as he lacked the typical thyrotoxicosis related symptoms and his TSH receptor antibody was negative. It revealed an uptake rate of 3% after 4 h, and 3% after 24 h over the thyroid bed, thereby confirming the diagnosis of painless thyroiditis.

He was started on oral propranolol 10 mg q6h. At 41 days follow-up, thyroid function tests were within normal limits.

3. Discussion

The incidence of thyrotoxic periodic paralysis (TPP) is relatively high in young Asian men who have hyperthyroidism [4,5]. Graves' disease accounts for the main cause in TPP patients [6].

In this case, based on the patient's clinical presentation and associated findings, a diagnosis of TPP due to painless thyroiditis was made. Painless thyroiditis presenting with TPP has been rarely reported [7-11]. A literature review revealed only 8 cases of TPP due to painless thyroiditis including our case (Table 1). All the patients were of male gender of Asian ethnicity, namely Korean (2/8), Indian (4/8) and Chinese (2/8). The age of the majority of TPP patients due to painless thyroiditis varied from 21 to 38 years with a median of 29 years with one exception of a patient being 61 years old. The major symptom noted was flaccid paralysis especially in the lower extremities. Among the 8 cases reported, 7 patients had no clinical symptoms of thyrotoxicosis. Six patients had no goiter or enlargement of the thyroid and in 2 patients there was no more than degree 1. The increase of FT4 concentration was no >3-fold of the upper limit of normal range among the 8 reported cases. The patients were scanned with Tc-99m thyroid scan or thyroid ^{131}I uptake scan and the results indicated that the uptake of thyroid area was low. The serum potassium levels in the 8 cases ranged from 1.50 to 2.50 mmol/L. It is important to note that the associated finding of acute hypercapnic respiratory failure was only reported in our case.

There are multiple known triggers for TPP patients. Other than hyperthyroidism, racial and genetic predisposition, hyperadrenergic state, exaggerated insulin response and other mechanism could also

Table 1
Clinical and laboratory findings of TPP patients due to painless thyroiditis.

Case	Gender	Age at onset (years)	Ethnicity	Clinical feature of thyrotoxicosis	Goiter	[K^+] (mmol/L)	FT4 (ng/dL)	T3 (ng/dL)
Lee et al.	M	38	Korean	No	Grade 1	2.50	2.40 (0.90–1.80)	179 (78–200)
Oh et al.	M	25	Korean	No	No	2.42	2.38 (0.75–2.00)	205 (80–170)
Sanyal et al.	M	23	Indian	No	No	1.80	2.80 (0.60–2.00)	200 (70–190)
Sanyal et al.	M	21	Indian	No	No	2.20	2.40 (0.70–2.00)	210 (70–190)
Sanyal et al.	M	35	Indian	No	Grade 1	1.50	2.50 (0.70–2.00)	NA
Sanyal et al.	M	32	Indian	Yes	No	1.80	3.20 (0.70–2.00)	NA
Chang et al.	M	61	Korean	No	No	1.90	1.70 (0.70–1.48)	204 (58–159)
Qian et al.	M	29	Chinese	No	No	1.50	4.20 (0.89–1.76)	42 (6–18)

TSH (μIU/mL)	Anti TG Ab (IU/mL)	Anti TPO Ab (IU/mL)	TSH receptor Ab (IU/L)	Tc-99m thyroid scan	Thyroid ^{131}I uptake scan	Reference no.
0.02	NA	NA	NA	Little or no	NA	9
0.00 (0.3–5.0)	93 (<60)	643 (<60)	0.37 (<1.5)	Little or no	NA	11
0.03 (0.5–4.7)	NA	74 (<35)	0.5 (<1)	0.10% (0.4–1%)	NA	7
0.06 (0.5–5.0)	NA	54 (<35)	NA	0.15% (0.4–1%)	NA	8
0.005 (0.5–5.0)	NA	68 (<35)	NA	0.10% (0.4–1%)	NA	8
0.03 (0.5–5.0)	NA	117 (<35)	NA	0.21% (0.4–1%)	NA	8
0.02 (0.35–4.94)	20 (<40)	NA	1.08 (<1.75)	Nonhomogeneous uptake	NA	10
0.03 (0.17–4.65)	11 (<115)	15 (<34)	<0.30 (<1.75)	NA	3% after 4 h 3% after 24 h	Current case

TPP, thyrotoxic periodic paralysis; K^+, potassium; P, Phosphorus; TSH, thyroid-stimulating hormone; T3, triiodothyronine; FT4, free-thyroxine; Anti TG Ab, anti-thyroglobulin antibody; Anti TPO Ab, thyroid anti-peroxidase antibody; Ab, antibody; M, male; NA, not available.

Table 2
Clinical and laboratory findings of TPP patients comprised with acute hypercapnic respiratory failure.

Case	Gender	Age of onset (years)	Ethnicity	$[K^+]$ (mmol/L)	Diagnosis	Mechanical ventilation	Duration of mechanical ventilation	Reference no.
Liu et al.	M	29	Chinese	1.30	Hyperthyroidism	Yes	14 h	2
Sthaneshwar et al.	M	36	Thai	1.80	Hyperthyroidism	NA	NA	14
Wu et al.	M	29	Chinese	1.40	Graves' disease	Yes	2 days	3
Abbasi et al.	M	27	Korean	2.20	Graves' disease	Yes	NA	1
Qian et al.	M	29	Chinese	1.50	Painless thyroiditis	Yes	8.5 h	Current case

TPP, thyrotoxic periodic paralysis; K^+, potassium; M, male; NA, not available.

lead to the occurrence of TPP [12,13]. Mechanisms of TPP due to painless thyroiditis are not fully understood because of the rare occurrence of these cases. The majority of the reported TPP cases due to painless thyroiditis were seen in young patients between 20 and 40 years old of Asian origin. They usually present with subtle thyrotoxicosis symptoms and unpalpable thyroid associated with mild elevated FT4 levels. According to Chang et al., most TPP (96.3% of which were Graves' disease) patients who develop acute TPP attacks, present with a mild clinical manifestation of thyrotoxicosis [4,6]. Given the similarities between the characteristics of TPP due to painless thyroiditis and TPP caused by hyperthyroidism, we argue that the mechanism that causes TPP with painless thyroiditis and TPP due to hyperthyroidism may share something in common.

A few cases have reported TPP patients associated with acute paralysis of the respiratory muscles (Table 2) [1-3,14]. All of the patients were of male Asian origin and their ages varied from 27 to 36 years. Their serum potassium levels varied from 1.30 mmol/L to 2.20 mmol/L. The majority of TPP patients reported presenting with acute hypercapnic respiratory failure were diagnosed with hyperthyroidism. The cause of TPP accompanied by acute respiratory failure was not confined to hyperthyroidism as seen in the current case. Among the 5 cases, four patients were intubated for a duration of no >2 days in 3 patients. All four patients were successfully weaned off the artificial ventilation support.

For patients presenting with TPP, during an acute attack, the emergency management goals are to terminate severe hypokalemia and prevent the associated fatal complications such as respiratory arrest or cardiac arrhythmias [5]. Both immediate intravenous or oral potassium chloride administration and nonselective β-blocker especially high-dose oral propranolol is feasible. As far as severe complications such as life-threatening respiratory failure is concerned, artificial mechanical respiratory support should be considered in those patients.

4. Conclusion

It should be kept in mind that potentially fatal complications such as acute hypercapnic respiratory failure might occur in acute attacks of TPP even in cases of painless thyroiditis.

Conflict of interest

There was no conflict of interest to be reported.

Acknowledgments

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