Thyrotoxicosis Occurring in Secondary Hyperparathyroidism Patients Undergoing Dialysis after Total Parathyroidectomy with Autotransplantation

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Secondary hyperparathyroidism (SHPT) is a common complication in chronic kidney disease (CKD) that is characterized by excessive synthesis of parathyroid hormone (PTH) and parathyroid hyperplasia.[1] The prevalence of CKD is estimated to be 5–10%, and the burden of CKD-associated diseases is alarmingly high.[2,3] Despite advances in medical therapy for SHPT, surgical parathyroidectomy remains the definitive therapy for refractory SHPT, which drastically decreases PTH levels and ameliorates symptoms related to severe SHPT.[4] Total parathyroidectomy, subtotal parathyroidectomy, or total parathyroidectomy with autotransplantation represents the current surgical options in the treatment of SHPT.[5] Total parathyroidectomy with autotransplantation may be preferable in patients who will require long-term hemodialysis after surgery, and total parathyroidectomy without autotransplantation should be prohibited in patients who have a better chance of receiving kidney transplantation because of the risks of iatrogenic hypoparathyroidism and hypocalcemia after kidney transplantation.[4]

It is reported that postoperative complications of parathyroidectomy for SHPT include hyperkalemia, hypocalcemia, local bleeding, and general complications of acute myocardial infarction and stroke.[5] However, there have been few reports on thyrotoxicosis as a postoperative complication of parathyroidectomy in SHPT patients with normal thyroid undergoing dialysis.

We give an uncommon report on a case of thyrotoxicosis occurred in an SHPT patient with normal thyroid undergoing dialysis after total parathyroidectomy with autotransplantation.

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A 35-year-old man was admitted to our department because of bone pain for 1 year. Seven years previously, he was diagnosed with CKD and began to undergone regular dialysis because of anuria and high level of serum creatinine. One year previously, he was diagnosed with SHPT after developing bone pain with high level of serum PTH and calcium. Medications were ineffective in reducing PTH level and controlling his bone pain.

On physical examination, the patient had a blood pressure of 152/100 mmHg, temperature of 36.6°C, respiration rate of 15/min, and pulse rate of 85/min. The thyroid was not abnormal. Accessory examination showed serum calcium of 2.85 mmol/L, serum phosphate of 1.03 mmol/L, and serum PTH of 3684 pg/ml. The results of thyroid function and antibodies tests were not abnormal [Supplementary Table 1]. Ultrasonography and ^{99m}Tc methoxyisobutylisonitrile single-photon emission computed tomography of the parathyroid showed bilateral enlargement of parathyroid. After active preoperative preparation, the patient underwent total parathyroidectomy with autotransplantation successfully. Postoperative pathology showed bilateral superior and inferior parathyroid hyperplasia. Postoperative serum PTH was 10.1 pg/ml, calcium 1.52 mmol/L, and

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phosphate 0.52 mmol/L. Hypocalcemia was effectively controlled with alfacalcidol and calcium agents. Regular dialysis three times a week was still applied for the patient.

From the 1st postoperative day, the patient showed fear of heat and sweating and used to wear thin clothes and disliked sleeping covered with quilt, and thyroid function test showed obviously raised thyroid hormones and thyroid globulin; however, thyroid stimulating hormone (TSH) and TSH receptor antibodies (TRAbs) were normal [Supplementary Table 1].

On the 3rd day, thyroid function test showed further raise in thyroid hormone and decreased TSH [Supplementary Table 1]. Then, thyrotoxicosis was diagnosed, and the dialysis was strengthened to eliminate the excessive thyroid hormones, and dexamethasone and β-adrenergic antagonist propranolol were administered to inhibit the thyroid function and control beta-adrenergic activity. The symptoms of thyrotoxicosis were effectively controlled. Thyroid function test on the 11th postoperative day improved significantly [Supplementary Table 1], and the patient was discharged successfully.

This is a case report about as SHPT patient with normal thyroid undergoing dialysis, who suffered from thyrotoxicosis after total parathyroidectomy with autotransplantation. It has rarely been reported that thyrotoxicosis could be one of the complications of total parathyroidectomy with autotransplantation for SHPT patients with normal thyroid undergoing dialysis. Usually, thyrotoxicosis can be caused by the following conditions:[6] (1) the thyroid is excessively stimulated by trophic factors; (2) constitutive activation of thyroid hormone synthesis and secretion occurs, leading to autonomous release of excess thyroid hormones; (3) thyroid stores of preformed hormone are passively released in excessive amounts due to autoimmune, infectious, chemical, or mechanical insult; or (4) there is exposure to extrathyroidal sources of thyroid hormone.

Before operation, the patient's thyroid was normal without abnormal findings of thyroid ultrasonography, thyroid function tests (triiodothyronine, thyroxine, free triiodothyronine, free thyroxine, and hypersensitive thyrotropin), thyroid globulin, and relative thyroid antibodies (thyroglobulin antibody, thyroperoxidase antibody, TRAb). The reason of thyrotoxicosis for this SHPT patient after total parathyroidectomy may be that thyroid hormones stored in the thyroid were excessively released into the blood circulation due to mechanical irritation during operation. In this patient, all enlarged parathyroids were adherent to the thyroid tissues; therefore, during isolation of parathyroid, part of the thyroid tissues were mechanically injured and excessive amounts of the stored thyroid hormones were released into the blood. While excessive amounts of thyroid hormones were not adequately eliminated by dialysis, it could result in thyrotoxicosis. Thus, from the 1st postoperative day, the patient began to manifest with symptoms of thyrotoxicosis, such as being afraid of hot and sweating, and tachycardia. He began to dress thin and dislike covering the quilt to sleep. Appropriate treatment of thyrotoxicosis requires an accurate diagnosis.[6] When the patient was confirmed with the diagnosis of thyrotoxicosis by laboratory tests, the dialysis was strengthened to eliminate the excessive thyroid hormones, and dexamethasone and the β-adrenergic antagonist propranolol were given to inhibit the thyroid function and control beta-adrenergic activity. Then, the symptoms of thyrotoxicosis were effectively controlled.

It is suggested that thyrotoxicosis may occur in SHPT patients undergoing dialysis after total parathyroidectomy with autotransplantation. As we all know, the lethal complications of total parathyroidectomy for SHPT patients undergoing dialysis are hyperkalemia, acute myocardial infarction and stroke. While uncontrolled thyrotoxicosis is related to neuropsychiatric symptoms, weight loss, muscle weakness, tremor, atrial fibrillation, embolic events, and even cardiovascular collapse and death.[6] Hence, much more attention should be paid to thyrotoxicosis as one of the complications after the total parathyroidectomy with autotransplantation for SHPT patients undergoing dialysis.

Supplementary information is linked to the online version of the paper on the Chinese Medical Journal website.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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Supplementary Table 1: Change of thyroid function after parathyroidectomy with autotransplantation

Determination time	T3 (ng/dl) 0.66–1.61	T4 (μg/dl) 5.44–11.85	FT3 (pg/ml) 2.14–4.21	FT4 (ng/dl) 0.59–1.25	uTSH (mIU/L) 0.49–4.91	TGAb (IU/ml) 0.00–4.00	TPOAb (IU/ml) 0.00–9.00	Tg (pg/ml) 0.00–50.3	TRAb (IU/L) 0.3–1.8
Before operation	1.14	5.71	3.51	0.64	4.66	<0.1	1.3	16.07	<0.3
First postoperative day	1.94↑	13.77↑	17.02↑	2.34↑	0.63	<0.1	0.60	>480.0	<0.3
Third postoperative day	2.02↑	19.1↑	11.57↑	3.56↑	0.12↓	<0.1	0.50	>480.0	–
Fifth postoperative day	1.53	15.33↑	7.51↑	2.78↑	0.05↓	<0.1	0.70	>480.0	–
Seventh postoperative day	1.95↑	13.86↑	6.77↑	2.11↑	0.03↓	<0.1	1.40	155.34	–
Eleventh postoperative day	1.68↑	11.11	5.96↑	1.57↑	0.02↓	<0.1	2.50	69.10↑	–

FT3: Free triiodothyronine; FT4: Free thyroxine; uTSH: Ultrasensitive thyroid stimulating hormone; TGAb: Thyroglobulin antibody; TPOAb: Thyroperoxidase antibody; Tg: Thyroglobulin; TRAb: Thyrotropin receptor antibody; ↑: Increase; ↓: Decrease; -: No relevant data.