Acute Thyrotoxicosis and Thyroid Storm Following Thyroidectomy for Nontoxic Multinodular Goiter

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Abstract

Background: Thyroid storm is a sequelae of surgery in patients with unrecognized or poorly controlled thyrotoxicosis.

Methods: We report a case of thyroid storm following total thyroidectomy for a multinodular goiter in a patient with normal thyroid function preoperatively.

Results: Thyroid storm, manifested by nausea, vomiting, abdominal pain and atrial fibrillation with a rapid ventricular response, was precipitated by a surge of thyroid hormone release during manipulation of a large retrosternal goiter, as evidenced by a massive increase in serum thyroglobulin.

Conclusion: Thyroid storm can occur from manipulation and retraction of the thyroid gland in a patient with normal thyroid function as a result of traumatic thyroiditis and release of thyroid hormone.

Introduction

Acute thyrotoxicosis and thyroid storm are well recognized sequelae of thyroidectomy in patients with Graves’ disease whose FT4 and FT3 levels remain elevated at the time of surgery and in patients who undergo nonthyroid surgery with unrecognized thyrotoxicosis. When untreated, mortality from thyroid storm has been reported in 8-25% of patients [1-3]. However, thyroid storm has largely disappeared following thyroid surgery because of the routine preparation of thyrotoxic patients with thioamides, iodine, beta receptor blocking agents, or some combination thereof. Thyroid storm may also occur in patients undergoing thyroidectomy for toxic multinodular goiter or a toxic adenoma, but is less likely. Acute symptomatic thyrotoxicosis with or without thyroid storm following thyroidectomy for nontoxic multinodular goiter is a rare phenomenon.

Case presentation

A 54 year old, 98 kg African American woman was referred to our institution for management of a large, symptomatic goiter that was first diagnosed on a routine physical exam 5 years prior to presentation. She complained of increasing size of the goiter, dysphagia, dyspnea, cough, choking and hoarseness. She had undergone fine needle aspiration biopsies of multiple thyroid nodules, all of which were benign. The remainder of her medical history was notable only for hypertension and hyperlipidemia. On examination, she had a body mass index of 37.55 kg/m², a blood pressure of 134/66 mmHg, a heart rate of 88 beats/min, a temperature of 97.60°F and marked enlargement of both lobes her thyroid gland with a nodule palpable in the right lobe, that was extending retrosternally. An ultrasound exam revealed marked bilateral thyroid enlargement with a 4.3 cm solid hypoechoic nodule in the right lobe, a 2.6 cm solid-cystic, isoechoic nodule in the isthmus and a 4.2 cm solid hypoechoic nodule in the left lobe of the thyroid gland. A CT scan of the neck and chest with intravenous contrast revealed significant enlargement of the thyroid gland with mass effect on the trachea and retrosternal extension (Figure). The patient was euthyroid with a serum TSH level of 0.576 uIU/ml (0.450-5.330 uIU/ml).

The patient opted to proceed with total thyroidectomy, which was completed with removal of the retrosternal goiter through a cervical approach. At operation, a 166 gram thyroid gland (a normal thyroid gland weighs 10-20 grams) and an incidental 340 mg right inferior parathyroid adenoma were removed. Extensive traction and manipulation were required to deliver the goiter from beneath sternum. Her blood pressure and heart rate intraoperatively and postoperatively increased to a maxi-
Citation: Christopher R McHenry. Acute Thyrotoxicosis and Thyroid Storm Following Thyroidectomy for Nontoxic Multinodular Goiter. J Clin Med Img Case Rep. 2024; 4(1): 1611.

Figure 1: Computed tomographic images demonstrating a large retrosternal goiter with tracheal displacement and compression in axial (A), coronal (B) and sagittal (C) views. (SG=substernal goiter and T=trachea).

Table 1: Serial laboratory results.

<table>
<thead>
<tr>
<th>Component</th>
<th>Pre-op</th>
<th>POD4</th>
<th>POD6</th>
<th>POD9</th>
<th>POD14</th>
<th>POD21</th>
<th>POD28</th>
<th>POD32</th>
<th>POD46</th>
<th>POD53</th>
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</thead>
<tbody>
<tr>
<td>TSH (0.450 - 5.330 uIU/mL)</td>
<td>0.5676</td>
<td>0.033</td>
<td>0.016</td>
<td>0.477</td>
<td>7.251</td>
<td>36.883</td>
<td>35.670</td>
<td>15.94</td>
<td>7.785</td>
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</tr>
<tr>
<td>Free T3 (2.3 - 4.2 pg/mL)</td>
<td>3.5</td>
<td>3.4</td>
<td></td>
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<td></td>
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<tr>
<td>Total T3 (87.0 - 179.0 ng/dL)</td>
<td>47.6</td>
<td>66.8</td>
<td>84.2</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Free T4 (0.45 - 1.80 ng/dL)</td>
<td>1.50</td>
<td>0.90</td>
<td>0.40</td>
<td>0.70</td>
<td>0.60</td>
<td>0.70</td>
<td>0.90</td>
<td></td>
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<tr>
<td>Thyroglobulin (0.8 - 48.0 ng/mL)</td>
<td>&gt;2250</td>
<td>380.1</td>
<td>112.8</td>
<td>23.9</td>
<td>4.4</td>
<td>1.80</td>
<td></td>
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</table>

The patient presented to the emergency department on post-operative day 4 with nausea and vomiting, abdominal pain, increased anxiousness, chills, and tachycardia. The patient reported that her nausea was persistent since surgery. Her blood pressure was 190/86 mmHg, heart rate 126 beats/min and temperature was 99°F. Her serum TSH was 0.020 uIU/mL. She was admitted to the hospital and subsequently diagnosed with atrial fibrillation with a ventricular rate of 156 beats/min. An echocardiogram was normal. A repeat serum TSH level was 0.016 uIU/mL with a FT4 level of 1.5 (0.45-1.8 ng/dl) and a FT3 level of 3.4 pg/ml (2.3-4.2 pg/ml) (Table 1). Her hepatic function panel was normal. She was diagnosed with postoperative thyrotoxicosis and thyroid storm based on a Burch-Wartofsky score of 55 (Table 2). She was admitted to the intensive care unit and her levothyroxine was stopped; her heart rate was controlled with metoprolol and she was started on Xarelto.

In order to determine the cause of the thyrotoxicosis, a serum thyroglobulin level was measured and it was greater than 2,250 ng/ml (0.8-48.0 ng/ml) with an antithyroglobulin antibody titer that was less than 1 (<15 IU/ml). Over the course of her hospitalization, her serum thyroglobulin level decreased (Table 1); her serum TSH progressively normalized (Table 1) and the atrial fibrillation spontaneously resolved. Her symptoms resolved and she was discharged home after a 7 day hospital stay.

The patient’s thyroid function tests and serum thyroglobulin continued to be monitored as an outpatient. Her serum TSH level normalized approximately 2 weeks after operation. The serum thyroglobulin progressively decreased to 1.80 ng/ml. The levothyroxine was resumed on postoperative day 16, but at a lower dose of 0.1 mg. She developed increasing fatigue and low energy level and her levothyroxine was increased to 0.137 mg on postoperative day 21.

Discussion

Burch and Wartofsky developed a point system for diagnosis of thyroid storm using an assessment of dysfunction in various organ systems (Table 2) [2,3]. A score of 45 or greater is highly suggestive of thyroid storm [2]. Based on our patient’s increased anxiety, nausea, vomiting and abdominal pain, a heart rate of 156, atrial fibrillation and a precipitating event (surgical resection of a large retrosternal goiter), she was di-
our patient's thyrotoxicosis was likely secondary to a surge in release of pre-formed thyroid hormone as a result of traumatic injury to the thyroid follicles during manipulation at surgery in combination with initiation of thyroid hormone replacement therapy postoperatively. This is supported by the massive increase in serum thyroglobulin, a protein only expressed in the thyroid follicles, and the rapid resolution of symptoms that occurred concomitantly with the depletion of thyroid hormone and normalization of her serum TSH level. The patient’s FT4 and FT3 levels on postoperative day 6 presumably reflect a decline in peak levels, which likely occurred at surgery. Iodine-induced thyrotoxicosis was also considered as a potential etiology since the patient had a CT scan of her neck and chest with intravenous contrast 2 weeks prior to surgery, but the timing of onset of her symptoms which correlated with the massive surge in thyroglobulin does not support this.

The treatment of thyrotoxicosis secondary to the release of the thyroid hormone is principally beta-blocker therapy. Beta blockade is necessary to inhibit the peripheral actions of thyroid hormone. Esmolol can be administered intravenously or propranolol, atenolol or metoprolol can be administered orally. Glucocorticoids reduce the conversion of T4 to T3 and as a result may also be useful for treatment of thyroid storm related to release of thyroid hormone. Plasmapharesis and plasma exchange have been used for rapid reduction of thyroid hormone levels in patients who fail to improve using conventional therapy. Supportive care with intravenous fluids and electrolyte replacement is important to replace fluid and electrolyte losses related to vomiting and diarrhea. Thioamide drugs, which inhibit thyroid hormone synthesis, are not effective for treatment of thyrotoxicosis that occurs as a result of release of thyroid hormones stored in the gland.

**Conclusion**

Our case report is important because it illustrates that extensive manipulation and traction that is necessary to deliver a nontoxic multinodular goiter from a retrosternal location can result in traumatic thyroiditis with follicular cell damage and release of thyroid hormone. This is a rare phenomenon and a cause for thyroid storm, which is a life threatening condition.

**References**