Intraoperative thyroid storm: A case report

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An 18-year-old woman being treated for Graves disease underwent elective thyroidectomy. Tachycardia was noted before surgery. The patient's heart rate and temperature started to rise 30 minutes into surgery. Malignant hyperthermia was excluded on clinical grounds, and treatment with beta blockers was started. The patient's conditions stabilized, and surgery was completed. A review of the patient's laboratory test results revealed a high free thyroxine level before surgery. Diagnosis and management of thyroid storm are discussed.

Key words: Intraoperative thyroid storm.

Thyroid storm, or thyrotoxic crisis, is an acute exacerbation of the hyperthyroid state brought on by the sudden release of the thyroid hormones thyroxine (T_4), triiodothyronine (T_3), or both into the circulation. This results in an exaggerated thyrotoxic manifestation characterized by tachycardia, fever, agitation, dehydration, shock, congestive heart failure, and death. It most frequently is seen in patients with thyrotoxicosis secondary to Graves disease who are undergoing treatment or in whom disease recently developed and is untreated.[1] Many medical and surgical conditions can precipitate the crisis.[1] Mortality is said to range from 20% to 30% despite early recognition and treatment.[2] With universal treatment of the thyrotoxic state to euthyroid status, thyroid storm occurring during surgery is rare. More often, it occurs in a nonsurgical setting. We report the case of a thyroid storm occurring in an 18-year-old woman undergoing thyroidectomy for treatment of hyperthyroidism.

Case summary

An 18-year-old woman had been given a diagnosis of Graves disease 1 year before surgery. Her symptoms included palpitations, tremors of the hands, and heat intolerance. She also had noticed hair loss. She had a history of asthma, which necessitated the occasional use of inhaled albuterol. The diagnosis of thyrotoxicosis was made on clinical grounds and confirmed by a free T_4 level of 7.01 ng/dL (reference range, 0.8-1.89 ng/dL) (Table). Treatment was begun with propylthiouracil, 250 mg, twice daily and propranolol, 40 mg, twice daily. A year after institution of therapy, the patient opted for surgery. Laboratory test results during the year of treatment are detailed in the Table.

The patient was admitted the morning of surgery. She weighed 78 kg and had a pulse rate of 113 beats per minute, a blood pressure reading of 134/71 mm Hg, and an oral temperature of 36.8°C (98.2°F). The lungs were clear on auscultation. The hemoglobin concentration was 11.4 g/dL, and the hematocrit value was 35.7. An electrocardiogram done 3 months before surgery showed sinus tachycardia. The thyroid was slightly enlarged. The patient had not undergone previous surgery or anesthesia. The patient withheld all medications the morning of surgery on advice of the surgeon. The resting tachycardia was ascribed to anxiety, and it was decided to proceed with the surgery.

Premedication included 2 mg of midazolam, 0.2 mg of glycopyrrolate, and 2 puffs of albuterol. General endotracheal anesthesia was induced with 130 mg of propofol, 150 µg of fentanyl, and 50 mg of rocuronium. A single dose of esmolol, 20 mg, was given just before intubation. Anesthesia was maintained with isoflurane, 1.2% to 2.5%; nitrous oxide, 50%; and oxygen. Thirty minutes after skin incision, when the surgeon began dissecting around the thyroid gland, an increase of the heart rate from 110 to 114 to 120 beats per minute was noted. Additional boluses of fentanyl, 50 µg, and esmolol, 20 mg, were given. Her temperature (measured by esophageal probe) began rising between 0.3°F and 0.4°F every 15 minutes. The end-tidal carbon dioxide level remained around the low 30s mm Hg, and no mottling of the facial skin was seen. No undue warmth of the carbon dioxide absorber was noted.

A diagnosis of thyroid storm was made. Esmolol was given on 20-mg aliquots. The history of asthma precluded the use of propranolol (although it had been used preoperatively), and 1-mg boluses of metoprolol were given. Cooling measures were instituted.

Table. Thyroid function tests*

	Free T_4 (0.8-1.89 ng/dL)	Thyrotropin (0.4-5 µIU/mL)	Total triiodothyronine (78-176 ng/dL)
September 25, 2001	7.01	< 0.1	—
October 26, 2001	7.47	< 0.1	—
December 8, 2001	7.83	< 0.1	—
January 2, 2002	2.36	< 0.1	279
February 5, 2002 (day of surgery)	—	—	—
February 13, 2002	1.41	—	—

* Reference ranges are given in parentheses.

The warming blanket beneath the patient, which had not been turned on, was switched to a cooling mode at 32.0°C (89.6°F), and ice packs were placed around the head and neck and groin. The room temperature was then 21.1°C (70°F). A rapid infusion of 500 mL of cool lactated Ringer's was given.

Pulse oximetry readings stayed at 97% to 100%, and the blood pressure was stable at 105-120/40-50 mm Hg. The patient's body temperature stabilized at 38.6°C (101.4°F) and remained at this level for the last 2 hours of surgery. The subtotal thyroidectomy was completed in 6 hours with minimal blood loss. A total of 10 mg of metoprolol was given, and 8 mg of morphine was titrated in toward the end of surgery. Muscle paralysis was reversed with neostigmine, 3 mg, and glycopyrrolate, 0.2 mg. The patient was extubated and transferred to the intensive care unit. Urine (an indwelling urinary catheter was introduced during the case) remained a clear, pale yellow.

In the intensive care unit, she was responsive to verbal commands, her heart rate was 108 beats per minute, blood pressure was 146/65 mm Hg, respiratory rate was 23 breaths per minute, and tympanic temperature was 38.4°C (101.1°F). Arterial blood gas analysis showed a pH of 7.28, P_{CO_2} of 48 mm Hg, P_{O_2} of 209 mm Hg, and H_{CO_3} of 22, with a base deficit of –5 mm Hg with 40% oxygen by face mask.

Six hours after the end of surgery, oral propylthiouracil was resumed, and 12 hours after surgery, the patient's heart rate came down to 100 beats per minute. She was discharged the following day.

Discussion

The pathophysiology of thyroid storm is well known.[2] Thyroid storm in patients with hyperthyroidism is brought on by illness, usually an infection, or by surgery. Thyrotoxic crisis associated with surgery can manifest intraoperatively but more likely occurs 6 to 18 hours postoperatively.[3] Previous studies document thyroid storm occurring in 10% to 32% of unprepared or inadequately prepared hyperthyroid surgical patients and associated with a high mortality rate.

The diagnosis of thyroid storm is made on clinical features. Diagnostic criteria on a point scale; clinical features grouped by history and systems; and thermoregulatory, central nervous system, gastrointestinal, and cardiovascular symptoms have been established.[2] While such diagnostic points may be helpful to the clinician, intraoperatively hyperpyrexia and tachycardia are the main clues for the anesthesia provider. Few laboratory tests are of help during the crisis.

A rising temperature and tachycardia in a surgical patient would suggest malignant hyperthermia. Distinction can be difficult and has to be made on clinical grounds.[4] A low or normal end-tidal carbon dioxide level would draw attention away from malignant hyperthermia.

This patient exhibited the features of thyroid storm, mainly a rising heart rate and a rising temperature. Malignant hyperthermia was thought unlikely because of the low end-tidal carbon dioxide level and absence of other features (eg, cardiac arrhythmias and skin flushing). An arterial blood gas sample would have confirmed the low or normal carbon dioxide level and the absence of arterial hypoxemia. Early intervention with beta-adrenergic blockers and supportive measures prevented what could have been a disaster. The youth of the patient probably was a factor in the good outcome.

The management of thyroid storm is well known.[5] There are 4 objectives in its management: treating any underlying illness, reducing the secretion and production of thyroid hormones, general supportive measures, and diminishing the metabolic effects of thyroid hormones. General supportive measures such as replacement of fluids and reducing the temperature by using ice packs or hypothermic blankets were used in this case. Beta-adrenergic blockers reduced the metabolic effects. Propranolol remains the drug of choice, controlling cardiac symptomatology when given at a

rate of 1 mg per minute for a total of 2 to 10 mg; each dose lasts for 3 to 4 hours.[5] Alternatively, a continuous infusion of esmolol, 100 to 300 µg/kg per minute could have been titrated in to maintain the heart rate at less than 100 beats per minute.[3]

Thyroid storm, although uncommon, still can occur. This case serves to remind practitioners that thyroid storm can occur, especially in an inadequately treated patient, as in the case described herein. In these days of outpatient surgery, the practitioner needs to stay alert when seeing for the first time a patient with hyperthyroidism admitted for surgery, whether the surgery involves the thyroid or not. The brief period the anesthesia provider is given to assess the patient and formulate an anesthesia plan does not serve the patient well. The anesthesia provider needs to keep a high degree of skepticism about euthyroid status to ensure that patients will not be subjected to the storm that can kill.

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