Total Thyroidectomy in a 4 Year Old with Graves Disease and Uncontrolled Hyperthyroidism

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Intro:
Graves disease as a cause of thyrotoxicosis in children occurs at a rate of 1:10,000 (1). Less than 5% of children with Graves Disease present at 5 years or younger (1). The disease is caused by thyroid gland stimulation by thyroid stimulating antibodies resulting in hyperthyroidism. Patients present with excessive physical activity, tremor, tachycardia, flushing, palpitations, weight loss, accelerated linear growth, reduced bone mineralization, and poor school performance. Ophthalmopathy occurs in less than 50% of patients and is usually mild when present. Because of this, most pediatric patients will require definitive treatment with either radioactive iodine or surgical thyroidectomy. Surgical intervention is done with either subtotal thyroidectomy or total thyroidectomy. Almost all pediatric patients with a total thyroidectomy will have hypothyroidism as a complication and 10-15% of patients with subtotal thyroidectomy will have recurrence of hyperthyroidism. (1) Surgery is the treatment of choice for children less than 5 years old. (1)

Case:
A 4-year-old male with Grave’s disease and symptomatic hyperthyroidism presented for total thyroidectomy. Past medical history was significant for prematurity at 24 5/7 weeks, retinopathy of prematurity, prior grade 2 intraventricular hemorrhage, gastroesophageal reflux disease (GERD), bronchopulmonary dysplasia and reactive airway disease. Despite taking methimazole 12.5 mg QHS 6 months prior to the procedure, he remained clinically hyperthyroid with elevated thyroid levels (TSH <0.01, Free T4 3.1, T3 439). In the pre-anesthesia holding area, he was tachycardic with heart rate greater than 115 bpm and upon examination he had marked thyromegaly and exophthalmos. Prior to induction he was pre-medicated with oral versed (0.5 mg/kg). Induction of general anesthesia was achieved with 70% N2O/30% O2 followed by 8% sevoflurane. Two 22g IV catheters were placed in the left hand and boluses of propofol 100 mg and fentanyl 25 mg were given to facilitate tracheal intubation. A pediatric Glidescope was used to visualize the placement of a 5.0 mm NIM tube. Prior to placement of the NIM tube, the vocal cords and upper airway were directly anesthetized with 2 mL of 4% lidocaine. The patient was maintained with 1 MAC of sevoflurane and a dexmedetomidine infusion at 0.4 mcg/kg/hr. Pain control was achieved with intermittent boluses of fentanyl and IV acetaminophen. At the conclusion of the case, a deep extubation was performed. The patient recovered in the pediatric PACU where he remained sedated for several hours post-op. The patient was discharged home on post-operative day one with no complications.

Discussion:
Pediatric patients presenting for thyroidectomy are at risk for the precipitation of thyrotoxicosis or “thyroid storm” secondary to the physiologic stress response. As a preventative measure, patients are rendered euthyroid preoperatively with treatment of methimazole or propylthiouracil. Response is measured by normalization of T4 levels. A week before surgery, patients are treated with iodine drops to inhibit thyroid hormone production and causes the gland to become firm and less vascular. (1) Early recognition of thyroid storm is important, as mortality can be up to 20-30%. (2) Presenting symptoms can include hyperthermia to temperature greater than 40 degrees Celsius, tachycardia, arrhythmia and acidosis. It can look very similar to malignant hyperthermia but differences include less than expected acidosis, decreased creatinine kinase and lack of response to dantrolene. (2) Immediate treatment intra-operatively includes resuscitation, antagonizing peripheral adrenergic effects and intravenous beta-blockade such as propranolol to treat tachycardia as well as decrease the production and peripheral conversion of thyroid hormones. In preparation for the possibility of thyroid storm, boluses of esmolol and an esmolol drip was made available in the operating room and dexmedetomidine was used intraoperatively. Dexmedetomidine is a selective alpha2-adrenoceptor agonist with anesthetic and sedative properties thought to be due to activation of G-proteins by alpha2a-adrenoceptors in the brainstem resulting in inhibition of norepinephrine release. Major side effects include hypotension, bradycardia, hypertension and tachycardia. (3) The use of dexmedetomidine provided numerous benefits. It allowed for a smoother emergence and the prevention of coughing. In the absence of paralytic it helped provide a deep plane of anesthesia and kept the patient still throughout the case. Dexmedetomidine also reduces the heart rate which helped prevent tachycardia and prevented a stress response by the patient that could have potentially lead to thyroid storm.