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Anaesthesia management for thyroidectomy in a non-euthyroid patient following cardiac failure

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Abstract

A 24-year-old male thyrotoxic student of a tertiary institution had thyroidectomy in the presence of a persistently elevated thyroxine (T_4) and tri-iodothyronine (T_3) levels. He was initially managed for hypertension at a private hospital before he was referred to our hospital for expert management. He had symptoms of thyrotoxicosis three years before presentation at our hospital. Physical examination revealed bilateral anterior neck masses and a diagnosis of toxic goitre was made. The serum T_3 and T_4 were elevated. Chest X-Ray, electrocardiogram and echocardiography showed abnormal findings. He was commenced on antithyroid drugs. Surgery was postponed several times due to persistently elevated thyroid hormones. However, he developed cardiac failure after six months on medical treatment and was treated with digoxin and frusemide. He had thyroidectomy under general anesthesia after his cardiovascular status was optimized in order to prevent further deterioration of his cardiac function. The anesthetic management is presented and discussed.

Key words: Anaesthetic management, cardiac complications, thyroid storm, toxic goitre

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Introduction

Thyroid diseases and thyrotoxicosis are common in many African countries.^[1,2] The classical features of thyrotoxicosis are hyperactivity, weight loss, and tremor. Of importance to the anesthetist are the cardiovascular effects of hyperthyroidism including atrial fibrillation, congestive cardiac failure, and ischemic heart disease.^[3] Treatment of thyrotoxicosis include medical therapy, radioactive iodine and surgery.

While subtotal thyroidectomy is now less commonly performed for toxic goitres in developed countries,^[4] it remains the mainstay of management in developing countries.^[5,6] In an attempt to prevent a thyroid storm, patients are usually rendered euthyroid before surgery by using anti-thyroid drugs.

A series of successful thyroidectomies in non-euthyroid patients in this environment has been reported.^[5] The necessity for surgery was attributed to the fact that

antithyroid drugs were beyond the reach of some of these patients because of the cost. Apart from the cost, failure of antithyroid therapy in our environment can result from non-compliance on medication by patients due to long duration of therapy or the drugs could have lost their potencies due to logistic reasons. In these circumstances, the cardiovascular effects of hyperthyroidism get worse. In order to ameliorate these effects, there may be a need to operate on a non-euthyroid patient as long as precautionary measures are taken to prevent a 'thyroid crisis'

Case Report

A 24-year-old male university student presented with a three-year history of excessive sweating, undue heat intolerance, weight loss and palpitations, and, a month history of easy fatigability. He did not notice that he had an associated anterior neck swelling. He was managed for

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hypertension in a private hospital but was referred because of poor response.

Physical examination revealed a young man weighing 70 kg with mild bilateral proptosis, lid lag, and lid retraction. There were fine tremors of the outstretched hands and the palms were warm. He had bilateral anterior neck masses, multinodular, and non-tender; the right mass measuring 8 cm×6 cm while the left measured 5 cm×5 cm. The trachea was deviated to the right. The pulse rate was 96 bpm, regular rhythm and the apex beat was displaced to the 6th left intercostal space lateral to the mid-clavicular line. The heart sounds were normal. A diagnosis of toxic multinodular goitre was made.

Investigations revealed a hematocrit of 36%, white cell count $4.6 \times 10^9/l$ (normal differential count), platelet count $129 \times 10^9/l$. Serum electrolytes and urea- Na^+ 135 mmol/l, K^+ 3.9 mmol/l, urea 7.7 mmol/l, creatinine 8.3 μ mol/l, Ca^{2+} 2.30 mmol/l, phosphate 1.3 mmol/l. Urinalysis was normal.

Electrocardiogram showed sinus tachycardia, bilateral atrial enlargement, and left ventricular hypertrophy while echocardiography showed dilated left atrium and ventricle with normal left ventricular systolic and diastolic function. There was moderate regurgitation across the mitral and tricuspid valves. Chest X-ray showed soft tissue swelling in the anterior neck region with tracheal deviation to the right. The lung fields were clear. There was cardiomegaly with left ventricular preponderance.

The patient was commenced on anti-thyroid therapy with carbimazole 15 mg, 8 hourly, propranolol 40 mg b.d., and diazepam 5 mg b.d. on an outpatient basis. Surgery was postponed several times because of persistently elevated T3 and T4 [Table 1]. He developed congestive cardiac failure six months after commencing anti-thyroid medication and was treated with oral digoxin 0.125 mg daily and frusemide 40 mg. Antithyroid therapy was later reviewed to include Lugol's iodine 5 drops t.i.d. and oral dexamethasone 4 mg t.i.d., while carbimazole was increased to 20 mg t.i.d. In order to prevent further deterioration of his cardiac function, he had subtotal thyroidectomy under general anesthesia nine months after he presented although the serum T_3 and T_4

were still elevated [Table 1]. Lugol's iodine was discontinued 48 hrs before surgery. The sleeping pulse rate ranged between 67-82 bpm three weeks before surgery.

Anesthetic management

The patient was calm during the pre-anesthetic visit. The pulse was 80 bpm, while the blood pressure was 130/80 mmHg. The hematocrit was 42%. He was classified as American Society of Anesthesiologists' (ASA) physical status 2 with a Mallampati airway grade 2. Two units of whole blood were cross-matched for surgery. He was premedicated with 10 mg oral diazepam on the morning of surgery while he also had his antithyroid drugs.

In the operating room, a multi-parameter patient monitor, "Nellcor-Puritan-Bennette NPB 4000" was used to monitor the patient and the baseline parameters were: Blood pressure 126/78 mmHg, heart rate 84beats/min, arterial oxygen saturation (SpO_2) 98-100% (room air), temperature 36.8°C. The ECG was continuously monitored intraoperatively. An intravenous access was secured with an 18 g cannula. Intravenous lidocaine, dopamine, and a defibrillator were available for emergency use.

During pre-oxygenation, he had 3 μ g/kg of fentanyl followed by 1.5 mg/kg of lidocaine iv. Anesthesia was induced with 6 mg/kg of sodium thiopental and tracheal intubation was facilitated with 1.5 mg/kg suxamethonium iv. The lungs were ventilated with 50% nitrous oxide in oxygen using a Bain breathing system with a total fresh gas flow of 6l/min. Anesthesia was supplemented with isoflurane 1%.

Neuromuscular paralysis was initially achieved with atracurium 0.5 mg/kg followed by supplemental doses of 0.1 mg/kg increments up to a total dose of 80 mg. Further analgesia was continued with fentanyl given in 50 μ g increments up to a total dose of 375 μ g.

At surgery, a highly vascularised and grossly enlarged thyroid gland was found. Subtotal thyroidectomy was done. The gland weighed 225 g.

Intraoperatively, the monitored parameters were all within normal range. Intra-operative blood loss was 550 ml and fluid therapy consisted of 2 l of normal saline within surgery and anesthesia time of 3 h 10 min and 3 h 35 min, respectively. No intraoperative adverse event was recorded.

At the end of surgery, nitrous oxide was discontinued and the patient had 0.5% isoflurane in oxygen for the purpose of post-extubation laryngoscopy. Residual neuromuscular paralysis was antagonized with a combination of atropine 1.2 mg and neostigmine 2.5 mg iv. With adequate spontaneous tidal exchange, i.v. lidocaine 1.5 mg/kg was administered 2 min before tracheal extubation. A post extubation laryngoscopy revealed normal vocal cord movements.

Table 1: Serial thyroid function tests

	T3 (Normal 0.8–2.1 ng/ ml)	T4 (Normal 5.7–11.0 ng/ ml)	TSH (Normal 0.4–6.0 μIU/l)
1 st test	9.8	29.0	0.10
2 nd test	11.8	27.0	0.20
3 rd test	6.2	22.0	0.25
4 th test	5.4	16.0	0.30
5 th test	5.8	12.3	0.30

ng/ml - Nanogram per ml. μ IU/ml - Micro international units per ml;
 T_3 - Tri-iodothyronine, T_4 - Thyroxine, TSH - Thyroid stimulating Hormone

Postoperatively, oxygen was administered via nasal prongs at a flow rate of 2l/min in the recovery room. He was transferred to the ward fully conscious with stable vital signs. Post operative analgesia was ensured with intramuscular pethidine 100 mg 6 hourly for 48 hours followed by oral paracetamol 1000mg t.i.d. for three days. The patient was discharged home on the 10th postoperative day.

Discussion

There are three distinct treatments for hyperthyroidism; antithyroid medications, thyroidectomy, and radioiodine (¹³¹I).^[7] Medical treatment consists of antithyroid drugs (propylthiouracil, methimazole, or carbimazole), potassium or sodium iodide. β -adrenergic antagonists (propranolol) are often used to counteract the exaggerated sympathetic effects of hyperthyroidism. Radioiodine destroys thyroid cell function and is not readily available in our environment.

The reported patient's symptoms grew worse and elevated T3 and T4 levels persisted [Table 1] despite six months of medical treatment and he subsequently developed congestive cardiac failure. This could have been due to late presentation as diagnosis of hyperthyroidism was made only after three years of onset of the symptoms. This period was long enough to result in cardiac manifestations of hyperthyroidism. Secondly, the patient might not have been compliant on medical treatment due to the high cost of the drugs and long duration of treatment. Thirdly, due to logistic reasons, e.g., storage conditions, potencies of the drugs might not be guaranteed.

The patient was placed on steroid when thyroid hormone levels remained persistently high. Steroids cause a prompt reduction in the serum level of thyroid hormones, particularly T₃.^[8]

However, there was clearly a failure of medical treatment in the control of thyrotoxicosis in this patient. Therefore, subtotal thyroidectomy in the presence of persistently elevated thyroid hormone levels became inevitable in this patient in order to prevent further deterioration of his cardiac function. Some surgeons promote thyroidectomy as the treatment of choice for Grave's disease (the commonest cause of hyperthyroidism).^[9] However, very few patients with uncomplicated Grave's disease are treated by surgery in the United States and Europe.^[10] Other indications for surgery include a co-existing nonfunctioning nodule or a nodule suggestive of cancer on fine needle aspiration, large toxic multinodular goitres and solitary toxic adenomas. Thyroidectomy still remains the main option of therapy in our environment after the patient has been rendered euthyroid with antithyroid drugs.

Fentanyl and lidocaine were administered before induction

of anesthesia to blunt the cardiovascular responses, which occur during laryngoscopy and intubation. These responses, which were not desirable in this patient with thyrotoxic heart disease, may even increase blood pressure and heart rate; cardiac arrhythmias may also occur. These cardiovascular changes can be attenuated by intravenous administration of lidocaine (1.5 mg/kg) 1–2 min, remifentanyl (1.0 μ g/kg) 1 min, alfentanil (10–20 μ g/kg) 2–3 min or fentanyl (0.5–1.0 μ g/kg) 4–5 min before laryngoscopy.^[11] Thiopental, used for induction of anesthesia in this patient, is an agent of choice because it possesses some antithyroid activity at high doses.^[4] Indirect-acting adrenergic agonists that stimulate the sympathetic nervous system such as ketamine and pancuronium were avoided in the anesthetic management of this patient. Atracurium that was used for neuromuscular paralysis in this patient is cardiostable. Isoflurane, used for maintenance of anesthesia causes less myocardial depression when compared with enflurane or halothane and arrhythmias are less common than with other agents.^[12]

The most serious threat to hyperthyroid patients in the postoperative period is thyroid storm, which is characterized by hyperpyrexia, tachycardia, hypotension, and altered consciousness (agitation, delirium, coma). In the intraoperative period, it may mimic malignant hyperthermia. Treatment includes hydration and cooling, an esmolol infusion or intravenous propranolol (0.5 mg increments until the heart rate is less than 100 beats/min), propylthiouracil (250–500 mg every 6 h orally or by nasogastric tube) followed by sodium iodide (1 g intravenously over 12 h) and correction of any precipitating cause. Cortisol (100–200 mg every 8 h) is recommended to prevent complications from co-existing adrenal gland suppression. This patient had an uneventful perioperative management.

Conclusion

Thyroidectomy is traditionally performed in the hyperthyroid patient who has been rendered euthyroid with antithyroid drugs. Long standing hyperthyroidism may be complicated by cardiac disease and congestive heart failure. Failure of medical treatment may be an indication for surgery in order to prevent further deterioration of cardiac function. With precautionary measures put in place, it is possible to perform thyroidectomy in patients that can not be rendered euthyroid as was done in this case and in one centre in our environment.

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