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Case Report

Rapid control of thyrotoxicosis for urgent thyroidectomy

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ABSTRACT

Surgery for Grave's disease is one possible option for definitive treatment. Thyroidectomy is performed after achieving a euthyroid state. Pre-operative preparation of the hyperthyroid patient is essential to avoid peri- and post-operative serious complications due to thyrotoxicosis mainly thyrotoxic crisis and death. The mainstay of preparation is the administration of anti-thyroid drugs. Occasionally rapid pre-operative optimization is required for special reasons (intolerance or side effects to anti-thyroid drugs or an aggravated serious disease with thyrotoxicosis). Various regimens for this aim were suggested with protocols composing various combination iodine (as a Lugol's solution, or iopanoic acid, or ipodat sodium), steroids, usually dexamethasone and beta blockers, alternatively other options are plasmapheresis, lithium and exchange ionic resin. The restoration of hyperthyroid Grave's disease to euthyroidism in our patient was rapidly accomplished with iodine, dexamethasone and beta blockers (at seventh day without complications). We propose a possible treatment protocol for these cases.

Keywords: Grave's disease, Urgent thyroidectomy, Hyperthyroid

INTRODUCTION

The main goal of the treatment of thyrotoxicosis is to normalize thyroid hormone secretion and achieve remission. Three modalities of the definitive treatment of Grave's disease exist: anti-thyroid drugs (ATD), radio iodine ablation (RIA) and surgery. Surgery as a first choice is preferred in pregnant women, or in those with a severe eye disease or a large goiter.[1]

Other indications are thyroid malignancy, mechanical syndrome and contraindication for ATD and RIA. The risk of a perioperative thyroid storm is reduced by adequate pre-operative optimization with appropriate medical treatment.[2] Guidelines from the American thyroid association strongly recommend that patients should be rendered euthyroid prior to the procedure.[3]

CASE REPORT

A 29 years old women complains of increased sweating for the last two weeks, fatigue, tremulousness, a shortness of breath after minimal walking (about 50 meters) and weight loss of 2 kg per week. The clinical finding was a heart rate of 150 per minutes, with ECG sinus tachycardia, lightly wet and sweaty skin. She did not exhibit the clinical finding of goiter, orbitopathy or dermopathy consistent with Grave's disease.

Thyroid laboratory studies revealed thyrotoxicosis - thyroid stimulating hormone (TSH) 0.003 mU/l (reference range 0.350-4.940), free thyroxine (fT4) 34.7 pmol/l (reference range 9-19), free triiodothyronine 29 pmol/l (reference range 2.63-5.7)- causally Grave's disease with anti-TSH receptor antibodies 8.5 U/l (reference range <1.8). An ultrasound of the thyroid: right lobus 11.3 ml, left lobus 10.8 ml, parenchyma is light hypoechogenic, coarse structure with sparse areas to

maps, without nodules, lobes were ovoid, with thyroid inferno. She was commenced on thiamazole 60 mg daily, but one day after she was transferred back to our department from emergency with maculo-papulous itching exanthem on the neck, upper half of the body and upper extremities, tachypnoea 24 per minutes, with prolonged expirium with wheezing. The history was negative for using some new cosmetics, laundry detergent, some exotic meal or contact with animals. This allergic reaction was resolved after 80 mg of methyl prednisolone i.v. twice per day and levocetirizine 5mg p.o. We decided for an urgent thyroidectomy because there is a cross allergy to propylthiouracil and radioiodine was unreachable. Preoperative therapy was begun with dexamethasone 4 mg q8h, metoprolol zok 50 mg q12h, inorganic iodine 480 mg daily (as a Lugol solution). On the seventh day of this regimen, laboratory values were: TSH 0.003 U/ml, fT4 8.8 pmol/l, fT3 2.12 pmol/l (Table 1) and the thyroidectomy was performed. The microscopic finding was of a diffuse, partially-rested thyroid with focal activities, consistent with the clinical diagnosis. Post-operatively (on the 4th day) transient hypoparathyroidism appeared with parathormone (PTH 1-84) 0.59 pmol/l (reference range 1.58-6.03).

Table 1: Thyroid laboratory values.

Hormones	TSH	Free tyroxin	Free tri-iodothyronin
Before treatment	0, 003	34, 7	29
After treatment	0, 003	8, 8	2, 12
Reference range	0, 350-4, 940 mU/l	9-19 pmol/l	2, 63-5, 7 pmol/l

DISCUSSION

The indication for rapid optimization with the target thyroidectomy is poorly-controlled/non-response of disease to thionamides, intolerance or serious side effects to thionamides (allergy, agranulocytosis, hepatitis and vasculitis). Urgency is warranted when hyperthyroidism exacerbates heart failure or coronary artery disease and for patients with severe hyperthyroidism complicated by a thyroid storm.[4] Treatment is aimed at blocking the symptoms of hyperthyroidism and inhibiting the synthesis, release, and peripheral conversion of T4 to T3. This includes a combination of ATD, iodine preparation, beta blockers and corticoids in various proposed patterns.

ATD blocks the biosynthesis of thyroid hormones by inhibiting thyroid peroxidase. Beta adrenergic blockers block the excessive adrenergic activity of hyperthyroidism, also causing a modest reduction of serum T3 concentration, blocking T4 to T3 conversion. Inorganic iodine (a supersaturated solution of potassium iodine, or Lugol's solution) have principal use because they reduce the peripheral conversion of T4 to T3, inhibit thyroid peroxidase (TPO) and decrease the synthesis of thyroid hormones within 24-48 hours and release hormones from the gland.[5]

Iodine also decreases the rate of the blood flow, thyroid vascularity and intraoperative blood loss during the thyroidectomy mediated by an immunosuppressive effect on the vascular endothelial growth factor (VEGF) and the inflammatory mediator interleukine-16.[5,6] Radiographic contrast agents - sodium ipodate or iopanoic acid 1 g daily- dramatic decrease of thyroid hormones. Corticosteroids reduce the secretion of thyroid hormones by an inhibition the 5-deiodinase and also inhibit the peripheral conversion of T4 toT3.[7]

Other options are plasmapheresis, lithium and cholestyramine. Plasmapheresis is considered to be an alternative therapy for patients with thyrotoxicosis, especially when conventional treatment modalities are problematic. Plasmapheresis removes protein-bound hormones from circulation, and the dilution of thyroid hormones from the intracellular compartment decreases concentrations of free thyroid hormones.

Lithium is an effective alternative. The dose in case reports for preoperative preparation is 900-1200 mg daily with serum monitoring of levels for possible toxicities.

Cholestyramine, an ionic exchange resin, sequesters T4 in the intestine during enterohepatic circulation, preventing hormonal absorption and increasing faecal excretion. It is usually given in a dose of 4 grams 2 to 4 times a day. The addition of cholestyramine achieved normal thyroid hormone levels after one week of treatment.[6,8]

An effective protocol is the combination of 500mg of iopanoic acid twice a day, 1mg of dexamethasone twice a day, beta blockers and thionamides reduced thyroxine and tri-iodothyronine in the majority of patients (80%) to normal levels in 7 days.[9] Other regimens, including ATD in addition, were various combinations of steroids, iopanoic acid, sodium ipodat and beta blockers.[10,11]

CONCLUSION

Rapid pre-operative treatment is effective in special events before surgery for thyrotoxicosis. The aim to achieve normalization of the hormonal status before the operation is possible with 1-2 weeks of this therapy. We propose a possible treatment protocol for these cases.

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