Clinical Review and Update on the Management of Thyroid Storm

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Thyroid storm is a severe manifestation of thyrotoxicosis and can lead to multi organ failure with a high mortality rate.

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Abstract

Thyroid storm is a severe manifestation of thyrotoxicosis. Thyroid storm is diagnosed as a combination of thyroid function studies showing low to undetectable thyroid stimulating hormone (TSH) (<0.01mU/L) with elevated free thyroxine (T4) and/or triiodothyronine (T3), positive thyroid receptor antibody (TRab) (if Graves' disease is the underlying etiology), and with clinical signs and symptoms of end organ damage. Treatment involves bridging to a euthyroid state prior to total thyroidectomy or radioactive iodine ablation to limit surgical complications such as excessive bleeding from highly vascular hyperthyroid tissue or exacerbation of thyrotoxicosis. The purpose of this article is a clinical review of the various treatments and methodologies to achieve a euthyroid state in patients with thyroid storm prior to definitive therapy.

Introduction

Thyroid storm is a severe manifestation of thyrotoxicosis. In the United States, up to one in six hospitalizations for thyrotoxicosis were related to thyroid storm, with a 12-fold higher mortality.[1] It is most commonly seen in Graves' disease but can also occur in toxic multinodular goiter and with solitary toxic adenomas, and can be precipitated by thyroid or non-thyroid surgery, trauma, infection, acute iodine load, parturition, amiodarone use, and non-compliance to anti-thyroid medications.

Thyroid storm is diagnosed as a combination of thyroid function studies showing low to undetectable thyroid stimulating hormone (TSH) (<0.01mU/L) with elevated free thyroxine (fT4) and/or free triiodothyronine (fT3), positive thyroid receptor antibody (TRab) (if the underlying etiology is Graves' disease), and with clinical signs and symptoms of end organ damage. The fT3 may be normal in severely ill patients due to reduced conversion of peripheral fT4 to fT3.[2] Symptoms that highly correlate with thyroid storm include fever ≥ 38°C, tachycardia ≥130 beats per minute, central nervous system manifestations, congestive heart failure, and gastrointestinal/hepatic manifestations.[2,3,4] Two validated severity rating scales for thyrotoxicosis are the Burch–Wartofsky Point Scale (BWPS) and the Japanese Thyroid Association (JTA) scale. A BWPS of ≥45 or JTA categories of thyroid storm 1 (TS1) or thyroid storm 2 (TS2) with evidence of systemic decompensation require aggressive therapy.[2,3,4] Once diagnosed, all patients with thyroid storm

should be admitted to the ICU for aggressive medical management. Management then includes both addressing end organ damage and attaining a euthyroid state.

Definitive treatment involves either total thyroidectomy or radioactive iodine ablation of the thyroid gland. Initial treatment involves bridging to a euthyroid state prior to definite treatment. In the case of considering total thyroidectomy, the goal is to limit surgical complications such as excessive bleeding from highly vascular hyperthyroid tissue or exacerbation of thyrotoxicosis.[5] Both the American Thyroid Association (ATA) and the JTA do not specify recommended definitive treatment, likely due to the clinical variability of severity and response to initial medical treatment.[2,3] In general, those with more severe thyroid storm may benefit from total thyroidectomy, whereas those with milder thyroid storm who respond well to initial treatment may benefit from radioactive iodine ablation for definitive treatment if no other contraindications are present. There are several safe and effective pharmacological options available to achieve a euthyroid state, including a combination of modalities that can be used. The purpose of this article is a clinical review of the various treatments and methodologies to achieve a euthyroid state in patients with thyroid storm prior to definitive therapy.

Treatment Options

Antithyroid Drugs

First-line treatments include propylthiouracil (PTU), carbimazole, and methimazole (MMI), which are antithyroid medications that inhibit thyroid hormone synthesis by acting on thyroid peroxidase.[6] Carbimazole is not widely used in the United States. PTU inhibits the peripheral conversion of T4 to T3 by inhibiting type-1 deiodinase.[6] PTU is preferred over MMI due to its effects on peripheral conversion and has been shown to have a more significant effect in lowering serum T4 and T3 within 24 hours compared to MMI.[3,7] The JTA prefers MMI over PTU due to findings that MMI corrected thyroid hormone levels more rapidly than PTU, and a more favorable safety profile of MMI.[2,8] Ultimately, choosing between PTU and MMI depends on patient characteristics and tolerability. In the first trimester of pregnancy, PTU is preferred over methimazole due to increased risk of birth defects.[6]

Dosing of PTU is a 500–1000 mg load, then 250 mg every four hours, which can be given orally or intravenously.[3] The recommended maximum dose of PTU is 1600 mg per day.[2] Dosing for MMI is 60-80 mg/day, with maximal dose of 100 mg/day.[2,3] MMI is not available as intravenous dosing in the United States. However it should be noted that there have been cases of both MMI and PTU being given per rectum.

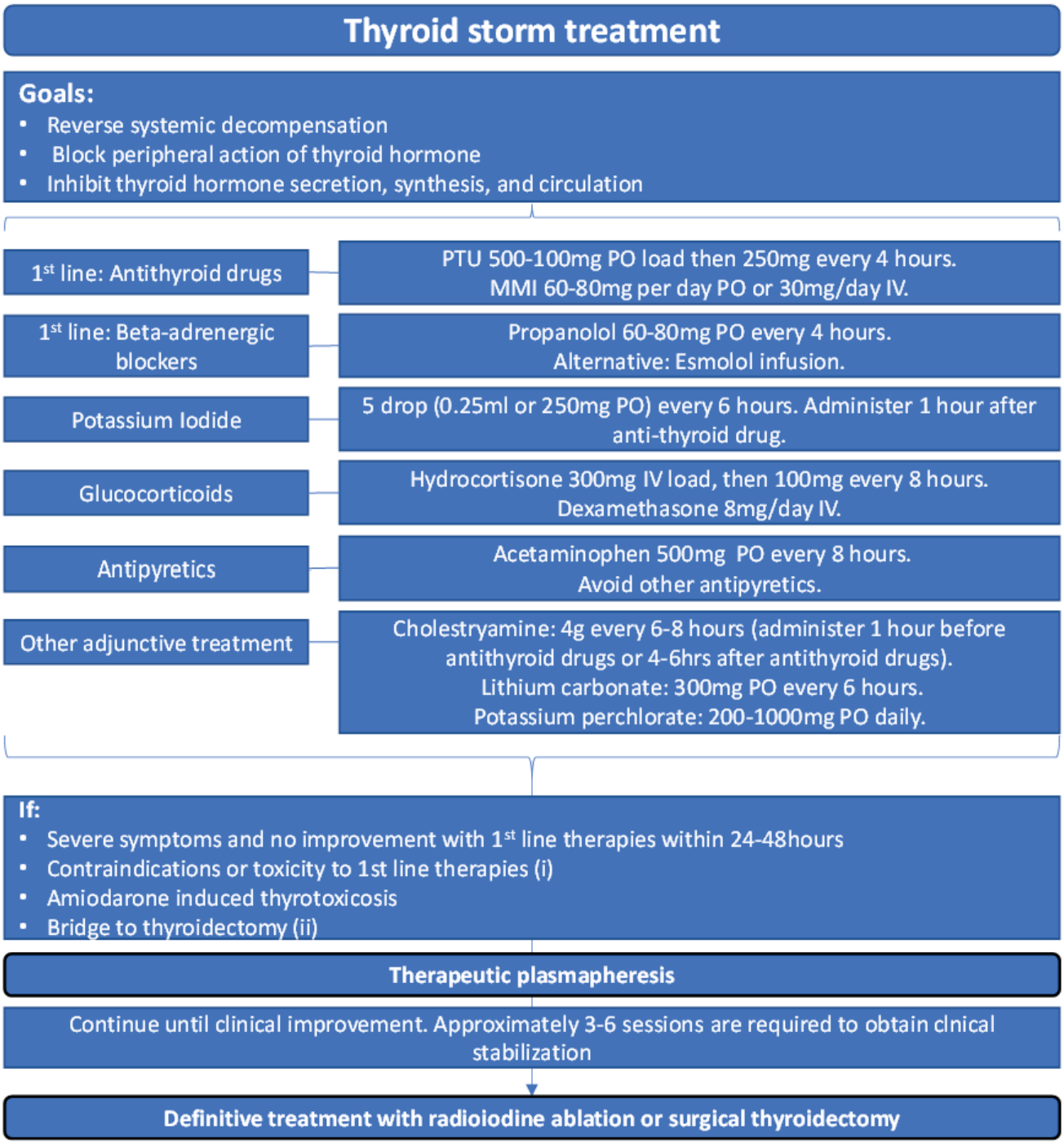
The major adverse effects of PTU and MMI are agranulocytosis, hepatotoxicity, and ANCA positive vasculitis.[6] In men of Asian descent, MMI is associated with hypoglycemia secondary to autoimmune insulin syndrome.[6] Minor adverse reactions include cutaneous reactions, arthralgias, gastrointestinal upset, abnormal taste sensation, sialadenitis, and lymphadenopathy.[6]

Iodine

Iodine acts to inhibit thyroid hormone via the "Wolff-Chaikoff effect" that reduces hormone biosynthesis by inhibition of iodine organification.[9] In preparation for total thyroidectomy, short-term iodine solutions are given as adjunctive therapy with either anti-thyroid thionamides or other non-thionamide medications. When administered preoperatively, iodine helps to decrease gland vascularity and blood loss.[10] Another advantage of iodine pretreatment is potentially lower doses of thionamides required to achieve the euthyroid state, reducing overall drug toxicity. Takata et al. (2010) and Sato et al. (2015) demonstrated that 15 mg/day of MMI combined with potassium iodide was significantly more effective at achieving a euthyroid state and had fewer adverse effects than 30 mg/day of MMI alone.[11,12] Note that in patients with iodine deficiency, toxic adenoma or toxic nodular goiter, treatment with iodine may actually increase thyroid hormone production due to increased availability of substrate. The ATA recommends iodine should either be avoided or only given an hour after thyroid synthesis inhibition with thionamide administration. However, the JTA recommends iodide supplementation at the same time as the thionamide due to no findings of increased thyrotoxicosis adverse effects.[2,12] In patients intolerant to thionamides, iodine is given as pretreatment pre-operatively.

Dosing of potassium iodide is either 5–7 drops (0.25–0.35 mL) of Lugol's solution (8 mg iodide/drop) or 1–2 drops (0.05–0.1 mL) of saturated solution potassium iodide (SSKI) (50 mg iodide/drop) three

Figure 1.Algorithm for the management of complicated thyrotoxicosis or thyroid storm. (i) Examples include acute liver failure and agranulocytosis associated with antithyroid drugs. (ii) Therapeutic plasma exchange allows a thyrotoxic patient to reach a euthyroid state prior to thyroidectomy or radioactive iodine ablation. IV, intravenous; MMI, methimazole; PO, oral; PTU, propylthiouracil. Figure adapted from Vinan-Vega et al., 2020.[31]



times daily mixed in water or juice for 10 days before surgery.[3] Up to 200mg/day of total iodine can be administered, with higher doses given to patients with suspected decreased gastrointestinal absorption.[2]

Adverse effects may include acne, loss of appetite, or upset stomach, fever, weakness, unusual tiredness, swelling of the neck or throat, mouth sores, skin rash, nausea, vomiting, stomach pains, irregular heartbeat, numbness or tingling of the hands or feet, a metallic taste in the mouth, anaphylaxis, sialadenitis, and esophageal ulcers.[13]

Lithium

Lithium has been shown to inhibit TSH stimulated thyroxine release via inhibition of the TSH-mediated cyclic adenosine monophosphate (cAMP) secondary signaling.[14] Due to its narrow therapeutic range and side effect profile, lithium is reserved for patients with contraindications to thionamides. In patients with thyrotoxicosis, lithium at therapeutic doses was shown to decrease serum thyroxine levels by ~25% within 4-10 days.[15,16] In amiodarone-induced thyrotoxicosis, lithium in combination with PTU was shown to achieve a euthyroid state within ~4 weeks, compared to ~11 weeks with PTU alone.[15] The dose of lithium is 300–450mg orally every 8hrs.[17] Ng et al. (2006) used a median dose of 750mg/day, with a range of 500-1500mg/day.[16] Adverse effects include tremor, nausea, vomiting, obtundation, cerebellar signs.[16]

Potassium Perchlorate

Perchlorate acts on the sodium iodide symporter and competes for iodine, and in addition, it discharges stored intrathyroidal iodide.[18] Amiodarone-induced thyrotoxicosis is partly mediated by high iodine load from the amiodarone molecule. Perchlorate administration helps to decrease the iodine burden.[19,20] It is often used in conjunction with thionamides, depending on patient tolerance.[20] The dose ranges from 200mg to 1000mg daily.[19] Common adverse effects include irritation, rashes, drug fever, lymphadenopathy, nephrotic syndrome, and agranulocytosis. Rarely, it can lead to aplastic anemia.[17]

Glucocorticoids

Glucocorticoids reduce the peripheral conversion of T4 to T3 and reduce thyroxine production in patients with Graves's disease.[17] Thyrotoxicosis is associated with adrenal insufficiency states [21,22] and steroid co-administration helps reduce the risk of exacerbation of adrenal insufficiency. The dose of hydrocortisone is a 300 mg intravenous load, followed by 100 mg every eight hours, or dexamethasone 8 mg/day.[3] Adverse effects are related to the short course of steroids, and include high blood pressure, hyperglycemia, and immune suppression.

Cholestyramine

T4 and T3 are concentrated in the liver and secreted in bile as either free form or conjugated to glucuronides or sulfates.[23] Cholestyramine is a bile resin, primarily used as a treatment for hypercholesterolemia. It has been shown to help reduce serum T4 and T3 in patients with hyperthyroidism refractory to thionamides by interfering with the enterohepatic circulation mediated reabsorption of thyroid hormone.[24,25,26] In a randomized control study, Tsai et al. (2005) demonstrated that in patients with Graves' hyperthyroidism, treatment with a combination of PTU, propranolol, and cholestyramine showed a significant decrease in serum T3/T4 at weeks two and four, compared to patients treated with PTU and propranolol alone.[25] Mercado et al. (1996) showed similar findings in a randomized study using methimazole and propranolol instead of PTU and propranolol in combination with cholestyramine.[24]

Dosing is 1-4 grams orally one to four times per day. It is administered one hour before other medications since it may reduce their absorption. The above noted studies indicated cholestyramine given for short four-week durations is well tolerated. However, known adverse effects are bloating, constipation, and flatulence.

Beta Adrenergic Blockers

Beta-adrenergic blockade (BB) is recommended in all patients with thyrotoxicosis for symptomatic control. The mechanism of action of BB is still unknown as they have no discernible effect on the thyroid gland itself, and studies have found conflicting effects on the reduction of peripheral T4 and T3.[27] Propranolol, at high doses of 120-160mg per day, is the only BB agent shown

to decrease peripheral T3, an effect not mediated by beta-adrenergic receptor blockade.[28] The main effects are related to controlling symptoms related to the sympathomimetic state of hyperthyroidism such as tachycardia, palpitations, tremor, and nervousness. In a randomized control study, Tagami et al. (2012) found that in patients with mild Graves' thyrotoxicosis, the combination of MMI and propranolol or atenolol significantly improved heart rate and subjective symptoms and quality of life compared to those treated with MMI alone after four weeks.[29] However, there was no significant effect on fT4 or fT3 between the groups.

Propranolol, the preferred agent, is administered at 10-40 mg three to four times per day. Atenolol dosing is 25-100 mg once to twice daily and is avoided in pregnancy. Metoprolol is dosed at 25-50 mg two to three times per day. Nadolol is dosed at 40-160 mg once daily. For severe thyroid storm requiring ICU management, esmolol at 50-100 ug/kg/minute can be administered.[3] Adverse effects include exacerbation of bronchospasm in patients with obstructive airway disease, and Raynaud's phenomenon.

Therapeutic Plasmapheresis

In cases of refractory severe thyrotoxicosis unresponsive to standard therapy, plasmapheresis is a useful modality for inducing a euthyroid state.[30,31] Plasmapheresis works through extracorporeal separation and removal of plasma from the body using centrifugation, including removal of plasma proteins with bound T3/T4 such as thyroid binding globulin and albumin, auto-antibodies, cytokines, and catecholamines.[30,31] Replacement plasma, albumin and crystalloid are returned to the circulation. This results in a rapid reduction of thyroid hormones that were bound to plasma proteins and anti-thyroid antibodies. In a retrospective study examining 46 patients who required plasmapheresis for severe thyroid storm, Simsir et al. (2018) found that after a median and interquartile range (IQR) of four sessions of plasmapheresis, there was a significant 45% reduction in fT4 and a 60% reduction in fT3, with no significant difference in number of sessions required between Graves' and non-Graves'-related thyrotoxicosis.[32] These findings were similar to other studies.[33,34,35,36,37] Plasmapheresis was also shown to be a safe option in pregnancy.[32,36]

The specific indications for plasmapheresis are: [30,31]

- Poor response to initial medical management with BB and PTU/MMI (agranulocytosis, leukopenia, hepatitis);
- Patients with severe symptoms and rapid deterioration;
- Contraindications to medical therapy - Liver disease or leukopenia precluding use of PTU/methimazole, hyperthyroidism caused by molar pregnancy, Graves' disease-related ophthalmopathy;
- Amiodarone induced thyrotoxicosis due to the long half-life of amiodarone; and
- Bridge to thyroidectomy or radioactive iodine ablation.

On review of prior case studies, it takes on average four to six rounds of plasmapheresis to achieve a euthyroid state, and should be continued until clinical improvement.[33,34,35,36,37] Serum free T3/T4 should be monitored daily. Once a euthyroid state is achieved, patients can either undergo radioactive iodine ablation or surgical thyroidectomy.

Plasmapheresis is generally well tolerated. Reported severe adverse effects of plasmapheresis include anaphylaxis and other allergic reactions, coagulopathy, hypocalcemia, hypotension, plasma-exchange related transfusion reactions, vascular injury, volume-electrolyte shifts, tetany and seizure, citrate-related nausea and vomiting, respiratory distress, and catheter related reactions.[2,34]

Conclusion

Thyroid storm is a severe manifestation of thyrotoxicosis and can lead to multi organ failure with a high mortality rate. There are various treatments and methodologies to achieve a euthyroid state in patients with thyroid storm prior to definitive therapy. Management and choice of therapy is based on clinical judgement and the patient's tolerance for the various treatment modalities. See Figure 1 for an algorithm for the management of thyroid storm.

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Disclosure

None reported.

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