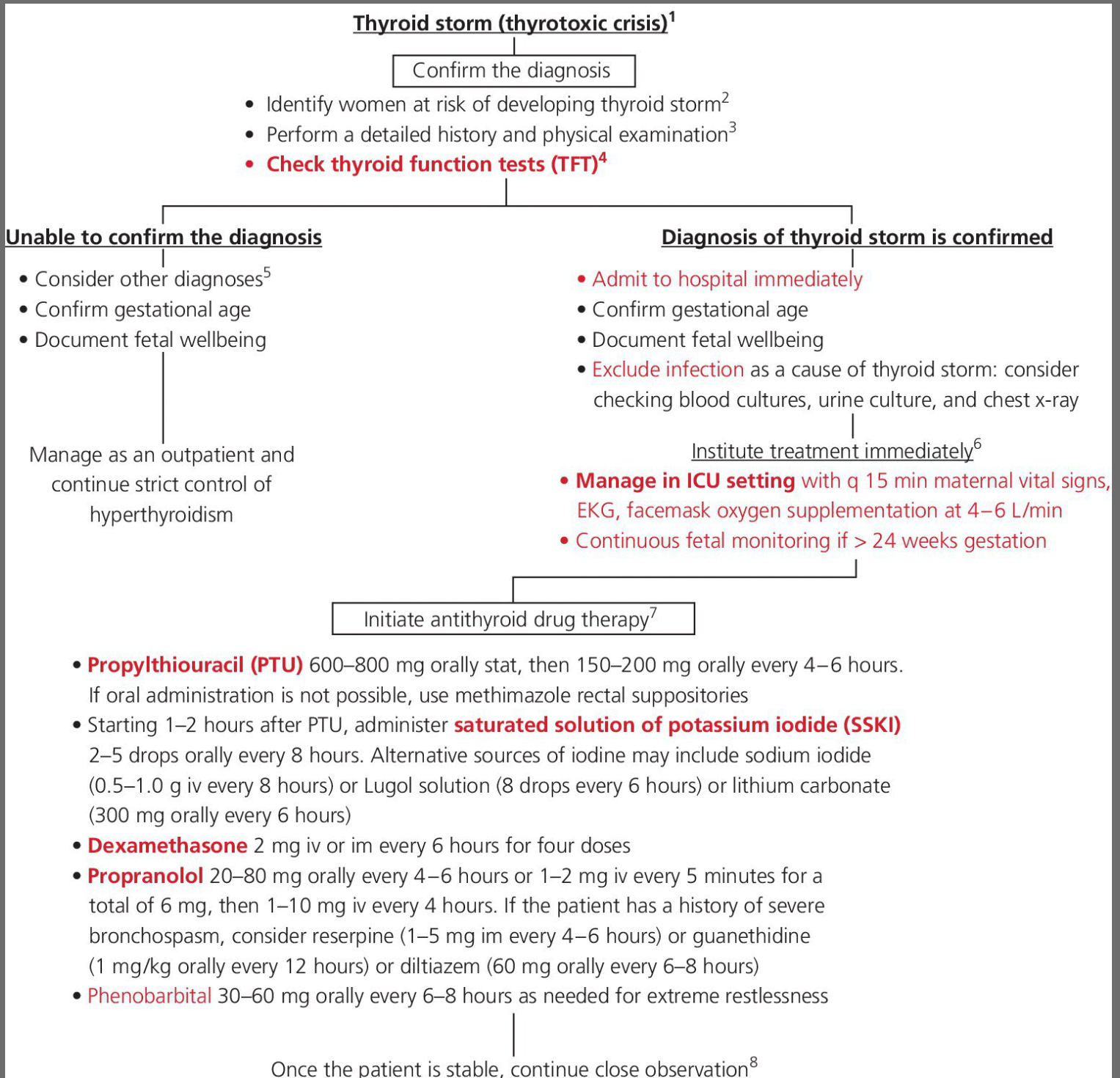




Learn simply

Thyroid Storm



1. Thyroid storm (thyrotoxic crisis) is a medical emergency characterized by a severe acute exacerbation of the signs and symptoms of hyperthyroidism. It is a rare complication, occurring in approximately 1% of pregnant patients with hyperthyroidism, but is associated with significant maternal and perinatal mortality and morbidity.
2. The vast majority of women presenting with thyroid storm have a history of hyperthyroidism. Hyperthyroidism refers to the clinical state resulting from an excess production of and exposure to thyroid hormone. The most common cause is Graves' disease, which accounts for 95% of all cases of hyperthyroidism in pregnancy and is caused by circulating thyroid-stimulating autoantibodies. Ophthalmopathy (exophthalmos, lid lag, lid retraction) and dermopathy (pretibial edema) are clinical signs that are specific to Graves' disease.
3. Other causes of hyperthyroidism in pregnancy include inflammation (thyroiditis), toxic multinodular goiter, solitary toxic thyroid nodule, hyperemesis gravidarum/gestational trophoblastic neoplasia, ingestion of exogenous thyroid hormone, and a TSH-secreting pituitary adenoma. In order to minimize complications (including thyroid storm), hyperthyroidism is best diagnosed and treated prior to pregnancy. An inciting event (such as infection, hypoglycemia, diabetic ketoacidosis, venous thromboembolism, surgery, and/or labor and delivery) can be identified in many instances of thyroid storm.



1. Thyroid storm is diagnosed by a combination of symptoms and signs in patients with thyrotoxicosis, including fever, tachycardia out of proportion to the fever >140–160 bpm, altered mental status (such as restlessness, nervousness, confusion or seizures), diarrhea, vomiting, and cardiac arrhythmia. However, the diagnosis can be difficult to make clinically.
2. If thyroid storm is suspected, serum thyroid function tests (TFTs) should be sent immediately.
3. Biochemical findings supportive of the diagnosis include suppressed levels of thyroid-binding globulin (<0.05 mU/mL) and increased free levothyroxine (T4) and L-triiodothyronine (T3) in the maternal circulation.
4. Although most women with Graves' disease will have circulating anti-TSH receptor, antimicrosomal, and/or antithyroid peroxidase autoantibodies, measurement of such antibodies is neither required nor recommended to establish the diagnosis. Moreover, antibody levels do not correlate well with either maternal or perinatal outcome.



1. The differential diagnosis of thyroid storm includes anxiety disorders, drug intoxication and/or withdrawal, and pheochromocytoma.
2. Thyroid storm is associated with significant maternal and perinatal mortality and morbidity, including shock, stupor, coma, and death. If the clinical index of suspicion for thyroid storm is high, treatment should be initiated immediately and should not be withheld pending the results of the biochemical tests.
3. The goals of treatment of thyroid storm are:
 4. (i) to reduce the synthesis and release of hormone from the thyroid gland using thioamides (such as propylthiouracil (PTU) or methimazole), supplemental iodide, and/or glucocorticoids;
 5. (ii) to block the peripheral actions of thyroid hormones using glucocorticoids, PTU, and/or β -blockers;
 6. (iii) to treat complications and support physiologic functions (manage in an ICU setting, acetaminophen, cooling blankets, supplemental oxygen, fluid and caloric replacement); and
 7. (iv) to identify and treat precipitating events (such as hypoglycemia, thromboembolic events, and diabetic ketoacidosis).
8. As with other acute maternal illnesses, fetal wellbeing should be appropriately evaluated and consideration given to delivery, if appropriate. Fetal tachycardia (>160 bpm) is a sensitive index of fetal hyperthyroidism. Only 1–5% of neonates born to women with poorly controlled thyrotoxicosis will develop transient hyperthyroidism or neonatal Graves' disease caused by the transplacental passage of maternal antithyroid antibodies.



1. Once stable, it is important:
2. (i) to follow serum electrolytes (especially potassium) and arterial blood gas q 2–4 hourly, as indicated;
3. (ii) to catheterize the patient if unconscious or not passing urine;
4. (iii) to decompress the stomach if unconscious; and
5. (iv) to continue fetal surveillance.
6. For women who fail to respond to initial medical therapy, options are limited. Radioactive iodine (^{131}I) administration to ablate the thyroid gland is absolutely contraindicated in pregnancy, because it will permanently damage the fetal thyroid. Surgery is best avoided, but may be required.



Kawita Bapat

