

## Learn simply

Thyroid Storm

## Thyroid storm (thyrotoxic crisis)<sup>1</sup> Confirm the diagnosis • Identify women at risk of developing thyroid storm<sup>2</sup> Perform a detailed history and physical examination<sup>3</sup> Check thyroid function tests (TFT)<sup>4</sup> **Diagnosis of thyroid storm is confirmed** Unable to confirm the diagnosis • Consider other diagnoses<sup>5</sup> Admit to hospital immediately • Confirm gestational age • Confirm gestational age Document fetal wellbeing Document fetal wellbeing • Exclude infection as a cause of thyroid storm: consider checking blood cultures, urine culture, and chest x-ray Institute treatment immediately<sup>6</sup> Manage as an outpatient and • Manage in ICU setting with q 15 min maternal vital signs, continue strict control of EKG, facemask oxygen supplementation at 4–6 L/min hyperthyroidism • Continuous fetal monitoring if > 24 weeks gestation Initiate antithyroid drug therapy<sup>7</sup> • Propylthiouracil (PTU) 600–800 mg orally stat, then 150–200 mg orally every 4–6 hours. If oral administration is not possible, use methimazole rectal suppositories Starting 1–2 hours after PTU, administer saturated solution of potassium iodide (SSKI)

- 2–5 drops orally every 8 hours. Alternative sources of iodine may include sodium iodide (0.5–1.0 g iv every 8 hours) or Lugol solution (8 drops every 6 hours) or lithium carbonate (300 mg orally every 6 hours)
- Dexamethasone 2 mg iv or im every 6 hours for four doses
- Propranolol 20–80 mg orally every 4–6 hours or 1–2 mg iv every 5 minutes for a total of 6 mg, then 1–10 mg iv every 4 hours. If the patient has a history of severe bronchospasm, consider reserpine (1–5 mg im every 4–6 hours) or guanethidine (1 mg/kg orally every 12 hours) or diltiazem (60 mg orally every 6–8 hours)
- Phenobarbital 30–60 mg orally every 6–8 hours as needed for extreme restlessness

Once the patient is stable, continue close observation<sup>8</sup>

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 thyroidstimulating 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-triiodithyronine (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. he 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  $\beta$ -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 (131I) 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.



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