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Approach to the Patient



Approach to the patient with thyroid storm

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01

Recognize the syndrome before organ failure progresses

02

Apply BWPS and JTA/JES criteria without delaying treatment

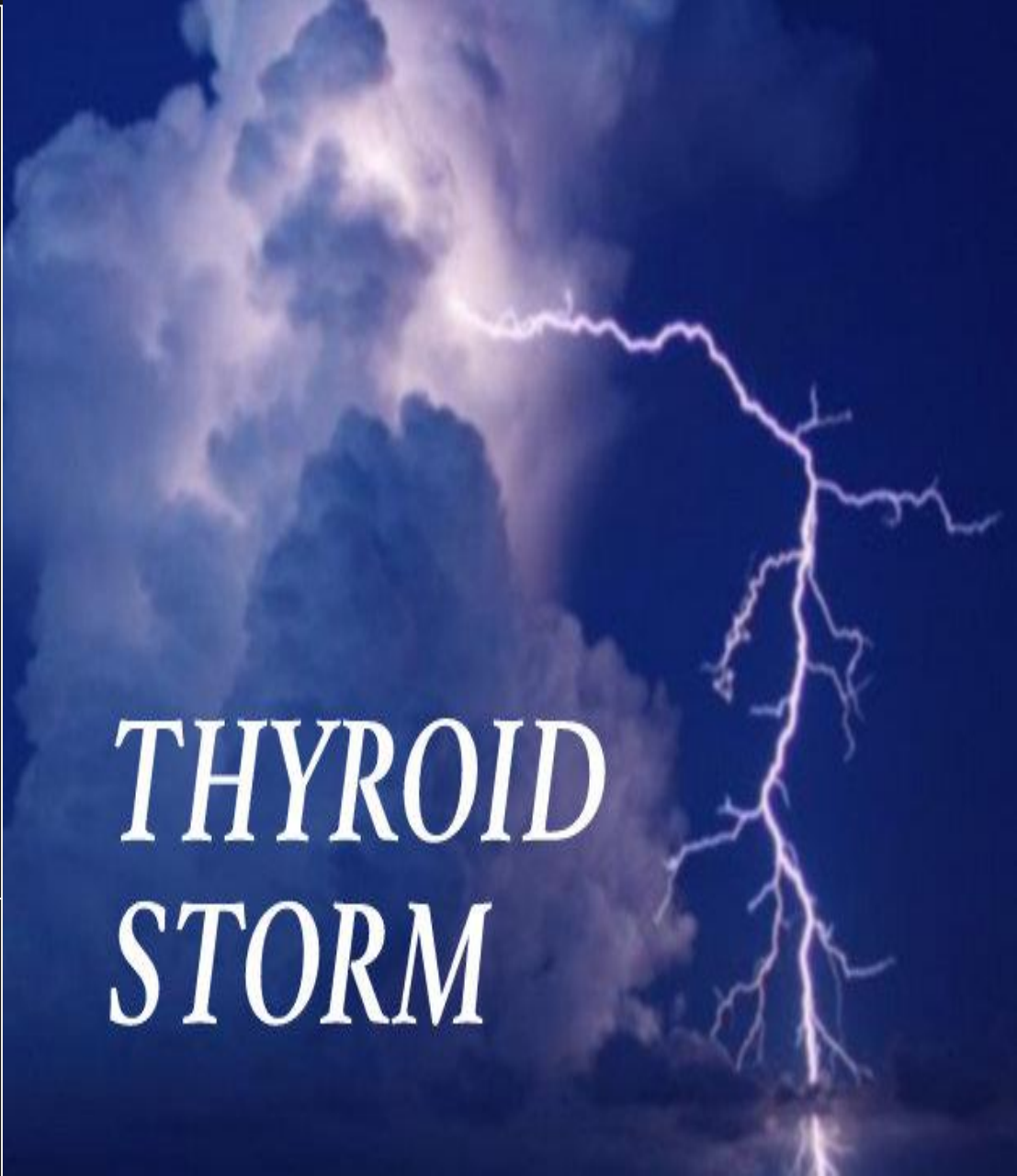
03

Sequence ICU support, beta-blockade, thionamide, iodine, and steroids correctly

04

Use rescue therapy and definitive treatment selectively

DEFINITION, ETIOLOGY & EPIDEMIOLOGY



*THYROID
STORM*

THYROID STORM: DEFINITION AND DIAGNOSTIC PRINCIPLE

- Thyroid storm is a critical, potentially life-threatening hypermetabolic state caused by severe manifestations of thyrotoxicosis.
- It requires rapid recognition and emergency treatment because delayed management can lead to fatal multiorgan decompensation.
- The diagnosis is clinical and depends on the constellation and severity of systemic manifestations.
- Functional decompensation of one or more organ systems is a **central feature** of thyroid storm.
- Two validated clinical scoring systems can support diagnostic assessment in patients with suspected thyroid storm.(Fig1,2)
- Standard thyroid function tests usually cannot distinguish thyroid storm from uncomplicated thyrotoxicosis.
- Therefore, the severity of systemic illness rather than the absolute thyroid hormone concentration determines the clinical diagnosis.

Underlying disease and precipitating factors

Precipitating event

Infection or sepsis
Withdrawal of antithyroid medication
Surgery or trauma
Acute cardiovascular illness
Iodine exposure
Severe physical or emotional stress

Underlying thyrotoxicosis

Graves disease
Toxic multinodular goiter
Toxic adenoma
Amiodarone-induced thyrotoxicosis
Rare: thyroiditis, hormone ingestion, trophoblastic disease, tyrosine kinase inhibitors or immune checkpoint inhibitors

No precipitant is identified in a minority of patients.

Incidence and Demographic Pattern

Table 1 Reported range estimates for incidence, sex ratio, and mortality

Incidence (per 100 000 persons/year)	• 0.2 to 1.1 (general population) • 4.8 to 6.3 (hospitalized patients)
Male–female ratio	• Approximately 1:2 to 1:3
Mortality	• 5% to 12% generally, • Up to 30% in older adults and patients with comorbidities

Estimates based on studies published between 2016 and 2025 (9, 13-20).

Among hospitalized patients, reported incidence was higher, ranging from approximately 4.6 to 6.3 cases per 100,000 hospitalized patients per year

MORTALITY AND MAJOR CAUSES OF DEATH

- The major causes of death include multiorgan failure, congestive heart failure, tachyarrhythmias, respiratory failure, Hypoxic brain injury, disseminated intravascular coagulation, and concomitant sepsis.
- In a Japanese national inpatient study of 1324 patients, in-hospital mortality was 10.1%.
- A multicenter French ICU study reported an ICU mortality of 17% and a six-month mortality of 22%.
- Across studies, advanced age(>60 years), neurological impairment, cardiovascular complications, cardiogenic shock occurring within 48 hours post-ICU admission, lack of antithyroid and beta-blocker therapy, and need for mechanical ventilation and plasma exchange along with hemodialysis consistently identify patients at higher risk of death.

HISTORICAL PERSPECTIVE



HISTORICAL EVOLUTION OF THYROID STORM

- In 1819, Jean-François Coindet introduced potassium iodide for goiter treatment in iodine-deficient regions of Switzerland.
- By 1821, severe tachycardia and other manifestations of thyrotoxicosis were recognized as adverse effects of iodine therapy.
- Fatal thyrotoxic events were subsequently reported, providing some of the earliest descriptions of thyroid crisis.
- The development of thyrotoxicosis after iodine exposure in autonomous multinodular goiters was later termed Jod-Basedow phenomenon by Theodor Kocher.
- In the 1920s, Frank Howard Lahey emphasized the high mortality associated with thyroid crisis, particularly after thyroid surgery.
- At that time, thyroidectomy was an important precipitating factor for thyroid storm in patients with uncontrolled hyperthyroidism.
- These early observations established the relationship between uncontrolled thyrotoxicosis, acute stressors, and life-threatening systemic decompensation.

FROM IODINE TO MODERN PHARMACOLOGIC THERAPY

- Henry Plummer introduced Lugol's solution before thyroidectomy in patients with Graves' disease and observed a rapid improvement in hyperthyroid manifestations.
- Preoperative iodine therapy was associated with a marked reduction in surgical mortality at the Mayo Clinic, from approximately 4–5% to less than 1%.
- High intrathyroidal iodide concentrations inhibit thyroid hormone release, an effect now known as the **Plummer effect**.
- This mechanism remains clinically relevant because iodide can rapidly suppress thyroid hormone release during thyroid storm.
- The introduction of thiourea and thiouracil compounds by Ted Astwood in the early 1940s fundamentally changed the pharmacologic treatment of hyperthyroidism.
- The subsequent development of beta blockers, particularly propranolol, in the 1960s provided an additional means of controlling the cardiovascular manifestations of thyrotoxicosis.
- Modern thyroid storm therapy therefore reflects more than a century of observations leading to the combined use of antithyroid drugs, iodide, and beta blockade.

THYROID STORM — RECOGNITION & DIAGNOSIS

Clinical emergency: severe thyrotoxicosis + organ decompensation

Think thyroid storm when:



Fever, often $>38.5^{\circ}\text{C}$



Marked tachycardia or atrial fibrillation



Agitation, delirium, psychosis, somnolence or coma



Heart failure, pulmonary edema or shock



Nausea, vomiting, diarrhea or jaundice



Common triggers



Infection or sepsis • Stopping antithyroid medication • Surgery or trauma • Iodine contrast • Acute illness

Diagnosis is clinical



FT4 and FT3 do not distinguish storm from uncomplicated thyrotoxicosis



BWPS: <25 unlikely | $25-44$ impending | ≥ 45 highly suggestive



JTA/JES: thyrotoxicosis plus CNS, fever, tachycardia, heart failure or GI/hepatic features



DO NOT WAIT FOR LAB RESULTS WHEN CLINICAL SUSPICION IS HIGH

Clinical pattern of thyroid storm

Central nervous system

Agitation, delirium, psychosis, somnolence, seizure, or coma

Cardiovascular and respiratory

atrial fibrillation, heart failure, pulmonary edema, shock, or cardiac arrest, occasionally reversible dilated or Takotsubo cardiomyopathy
Marked tachycardia, particularly when disproportionate to the degree of fever, is an important clinical warning sign.

Thermoregulatory and gastrointestinal

Fever, diaphoresis, nausea, vomiting, diarrhea, abdominal pain, jaundice, or liver failure

Older adults may present with apathetic hyperthyroidism and fewer classic adrenergic signs.

07 Diagnosis remains clinical

**CNS or organ
decompensation**

**High fever and
marked tachycardia**

**Goiter or known
hyperthyroidism**

**Start treatment when the clinical pattern is convincing.
Laboratory confirmation must not delay care.**

**Scoring systems support clinical judgment and help
estimate severity, but neither score replaces bedside
assessment.**

Burch–Wartofsky Point Scale

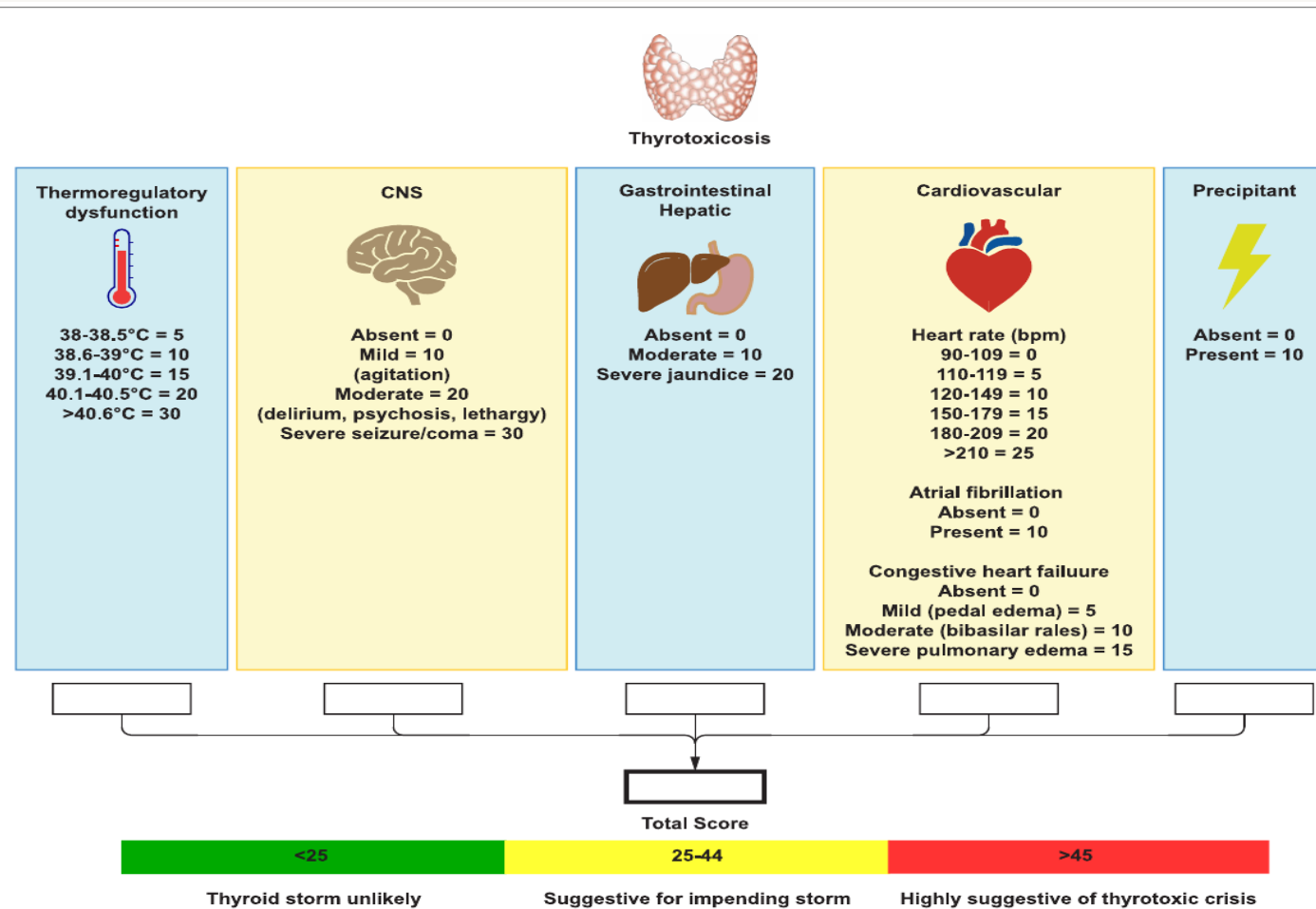


Figure 2 Graphical illustration of the Burch–Wartofsky scoring system for the diagnosis of thyroid storm. The Burch–Wartofsky Point Scale was first published in 1993 (1). CNS, central nervous system.

<25

Storm unlikely

25–44

Impending storm

≥45

Highly suggestive

Temperature, CNS, GI/hepatic, cardiovascular, and precipitant domains

Higher scores are associated with increased mortality and longer hospital and intensive care unit stays

09 JTA/JES diagnostic criteria

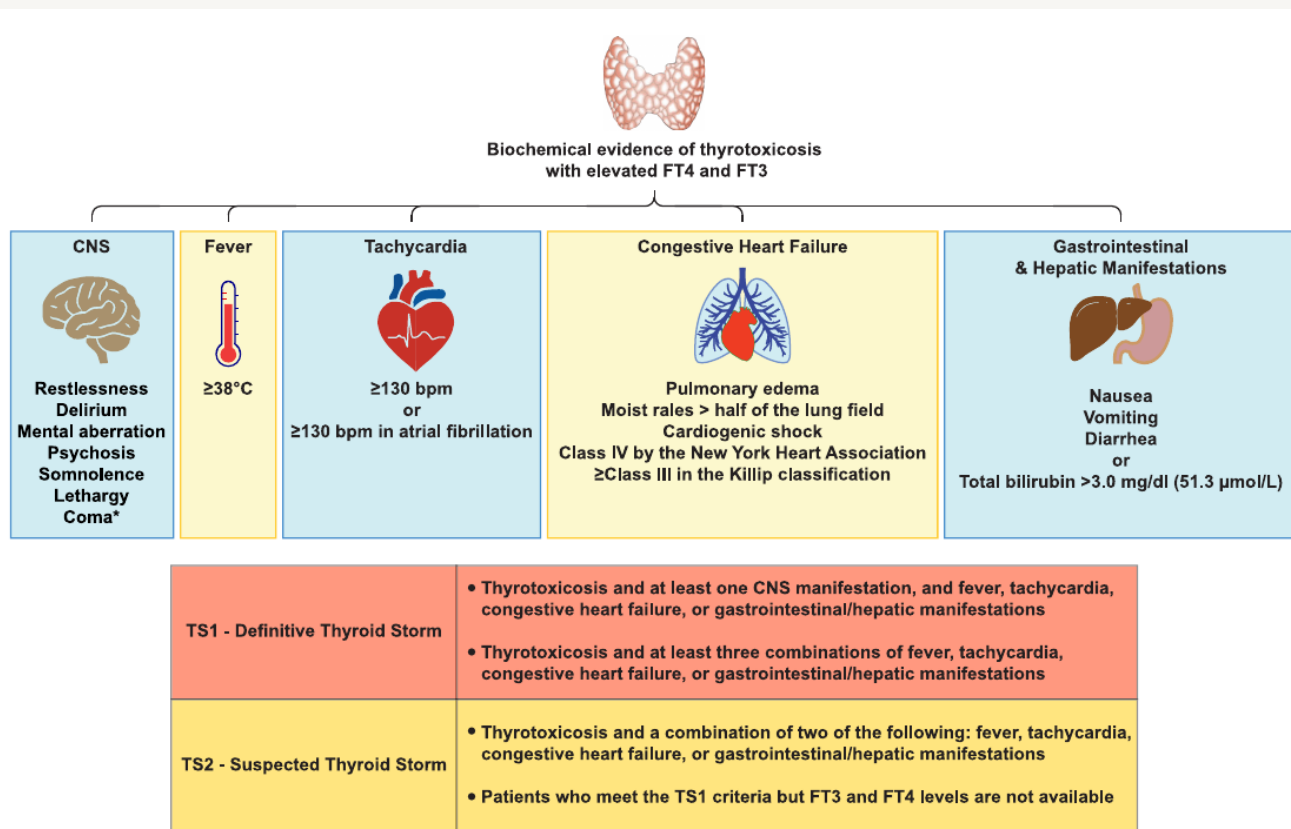


Figure 3 Graphical illustration of the Japan thyroid association/Japan endocrine society scoring system for the diagnosis of thyroid storm. The diagnostic criteria by the JAS/JES for thyroid storm were developed based on national surveys published in 2012 (7), and summarized in formal guidelines in 2016 (5). CNS, central nervous system; *: ≥ 1 on the Japan Coma Scale or ≤ 14 on the Glasgow Coma Scale; bpm, beats per minute; FT3, free triiodothyronine; FT4, free thyroxine).

TS1: definite

Thyrotoxicosis plus a CNS manifestation and at least one additional feature, or at least three non-CNS features

TS2: suspected

Two clinical features, or a TS1 pattern before FT4 and FT3 results become available

Laboratory findings and differential diagnosis

Important mimics

Sepsis
Meningitis or encephalitis
Heatstroke
Cocaine or amphetamine
intoxication(sympathomimetic agents)
Neuroleptic malignant syndrome
Adrenal crisis or diabetic ketoacidosis

Laboratory clues

Suppressed TSH

Elevated FT4 and FT3

FT3 may be normal during critical illness

Leukocytosis, hyperglycemia, lactate elevation

Electrolyte and acid-base disorders

Hepatic injury or hyperbilirubinemia,

Serum cortisol should be high during acute illness; low levels are suggestive of adrenal insufficiency

Lower FT3 concentrations and lower FT3/FT4 ratios may indicate more severe illness ,
Therefore, a relatively modest free T3 concentration does not exclude thyroid storm in a critically ill patient

Sepsis may mimic thyroid storm and may also trigger it.

WHY DOES THE STORM OCCUR?

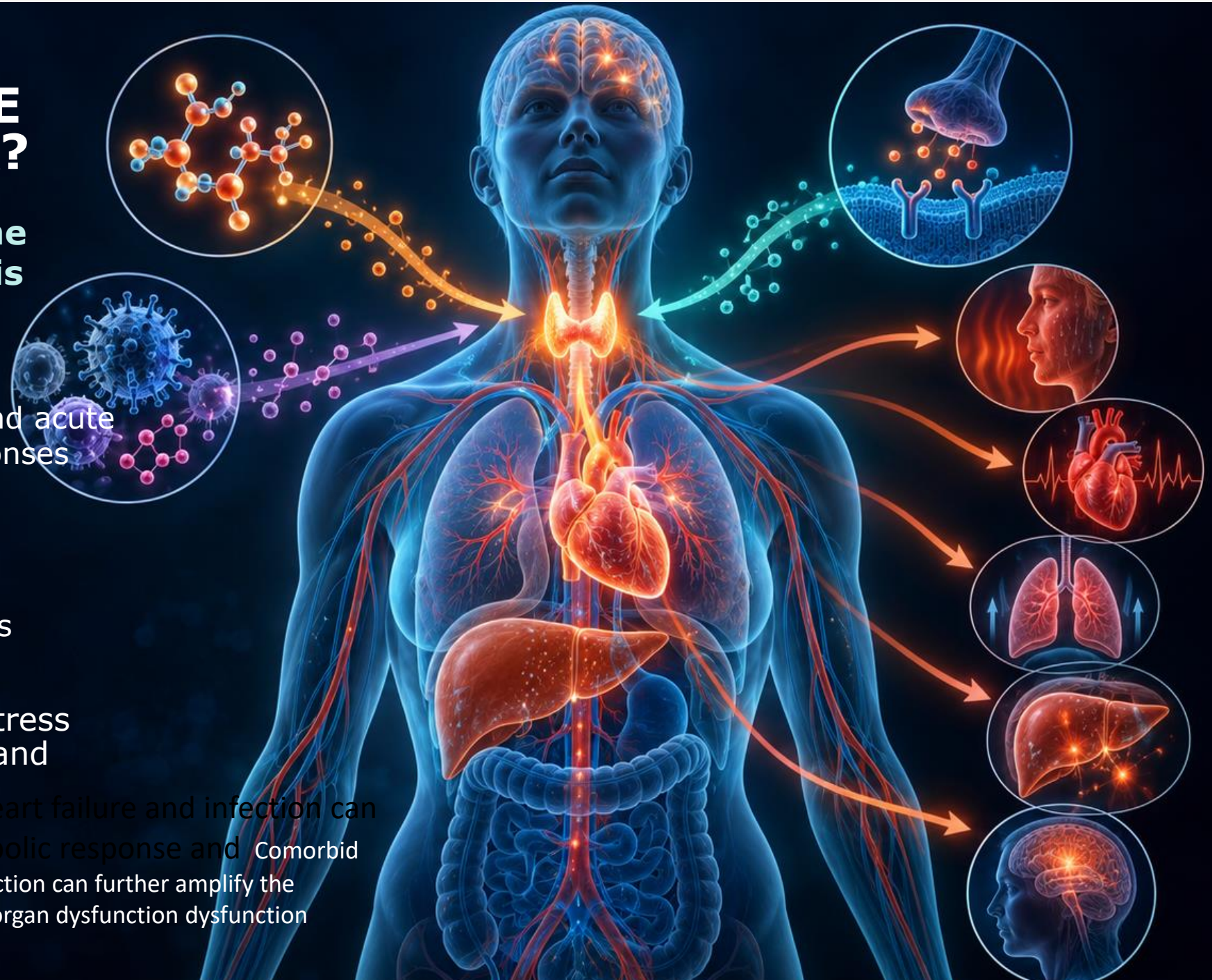
Hormone excess alone does not explain crisis severity

Inflammatory cytokines and acute stress amplify tissue responses

Enhanced beta-adrenergic responsiveness drives cardiovascular toxicity, neurological manifestations

Hypoxia and metabolic stress increase thermogenesis and organ injury

Comorbid conditions such as heart failure and infection can further amplify the hypermetabolic response and Comorbid conditions such as heart failure and infection can further amplify the hypermetabolic response and promote organ dysfunction



MANAGEMENT



IMMEDIATE MANAGEMENT: MULTIMODAL EMERGENCY THERAPY

- Suspected thyroid storm should be treated immediately when characteristic clinical features and a precipitating illness are present.
 - Treatment should not be delayed while waiting for thyroid hormone results because biochemical severity does not reliably reflect clinical severity.
 - Patients require intensive care with continuous cardiovascular, respiratory, neurological, and metabolic monitoring.
 - Initial management must address hyperthermia, tachycardia, heart failure, respiratory failure, and other manifestations **of organ dysfunction**.
 - Antithyroid therapy, iodide, corticosteroids, and appropriate beta blockade should be combined according to the clinical context.
- Simultaneous **identification and treatment of the precipitating condition** are essential for successful control of the crisis.

ICU support and precipitating illness

Cardiorespiratory support

Continuous ECG and hemodynamic monitoring, oxygen or ventilation, cautious fluids, and treatment of shock or heart failure

Temperature control External cooling and acetaminophen. Avoid aspirin because salicylates increase free thyroid hormone.
Chlorpromazine may be used if needed to reduce agitation and fever

Metabolic care

Correct sodium, potassium, magnesium, glucose, and acid–base abnormalities. Provide thiamine when indicated.

Obtain cultures and start appropriate antimicrobial therapy promptly when infection is suspected.

Beta-blockade requires hemodynamic judgment

Heart failure or instability

Use lower initial doses with close monitoring (cardiovascular collapse)

Esmolol allows rapid withdrawal if output falls

Load 500 mcg/kg IV, then 50–100 mcg/kg/min

Diltiazem may be considered when beta-blockers are contraindicated (asthma or severe cardiac decompensation)

Hemodynamically stable

Propranolol 60–80 mg orally every 4 hours

IV propranolol 1–3 mg slowly when oral treatment is impossible

Additional effect: reduced T4-to-T3 conversion

Beta-blockade can precipitate cardiovascular collapse in severe decompensated heart failure.

they reduce excessive adrenergic effects and control tachycardia, tremor, and anxiety.

Thionamides (inhibit thyroid hormone synthesis)

Clinical interpretation

Recent comparative data show no clear mortality advantage for PTU over methimazole.

Thionamides have limited value in purely destructive thyroiditis.

Methimazole

60–80 mg/day in divided doses
Carbimazole 80–100 mg/day
Preferred when significant hepatic dysfunction is present(>3 ULN)

PTU

500–1000 mg loading dose
Then 250 mg every 4 hours
Oral, nasogastric, or rectal route
Also reduces T4-to-T3 conversion

Taper doses according to peripheral hormone concentrations after stabilization.

Iodine

rapidly inhibits thyroid hormone release through the Plummer effect and suppresses hormone synthesis through the Wolff–Chaikoff effect

IODINE

2

**WAIT AT LEAST 1
HOUR**

THIONAMIDE

1

Lugol solution: 8 drops every 6 hours
SSKI: 5 drops every 6 hours
Potassium iodide Tablet: 65 mg three times daily

Giving iodine first may worsen hormone synthesis in autonomous nodular thyroid tissue.

They should be discontinued once the patient stabilizes. To minimize local irritation, iodine solutions should be diluted in fluids such as milk or juice before oral or nasogastric administration

Glucocorticoids and adjunctive clearance

Lithium

Inhibits hormone release
Reserve for specialist use
Requires toxicity monitoring

Cholestyramine

4 g three times daily
Interrupts enterohepatic recycling
Useful in severe or refractory disease

Hydrocortisone

300 mg IV loading dose
Then 100 mg every 8 hours
Reduces T4-to-T3 conversion
Provides adrenal coverage

Taper glucocorticoids after clinical improvement.

Rescue therapy and thyroidectomy

Thyroidectomy

Medical stabilization remains preferred.

Urgent surgery may be considered when aggressive treatment fails within 12–24 hours or medication cannot be used.

Management requires an experienced multidisciplinary center.

Plasma exchange

Consider when standard therapy fails, thionamides are contraindicated, severe liver failure is present, or rapid preoperative control is required.

The effect is rapid but temporary.

Surgery during uncontrolled thyrotoxicosis remains a rare and controversial rescue strategy.

THYROID STORM – PITFALLS, RESCUE & FOLLOW-UP

DANGEROUS PITFALLS



Aspirin for fever



Iodine before
a thionamide



High-dose beta-blocker
in decompensated
heart failure



Delaying ICU transfer



Waiting for FT4 or FT3
before treatment

WHEN STANDARD THERAPY FAILS

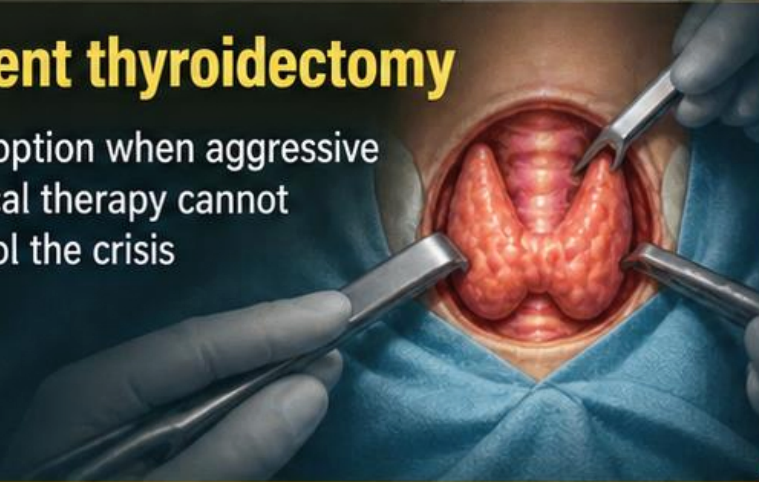
Plasma exchange

Consider when conventional therapy fails, thionamides are contraindicated, severe liver failure is present, or rapid preoperative control is required



Urgent thyroidectomy

Rare option when aggressive medical therapy cannot control the crisis



AFTER STABILIZATION



Taper therapy according
to FT4 and FT3



Treat the precipitating factor



Assess anticoagulation for
persistent atrial fibrillation



Plan definitive therapy:
radioactive iodine or thyroidectomy



Address nonadherence and
barriers to care



EARLY RECOGNITION + MULTIMODAL ICU CARE SAVES LIVES



Mortality: 5–12% overall;
up to 30% in older adults
and patients with comorbidities

After stabilization

Hormone-directed therapy

Taper thionamides according to FT4 and FT3. Stop short-course iodine after control.

Cardiovascular follow-up

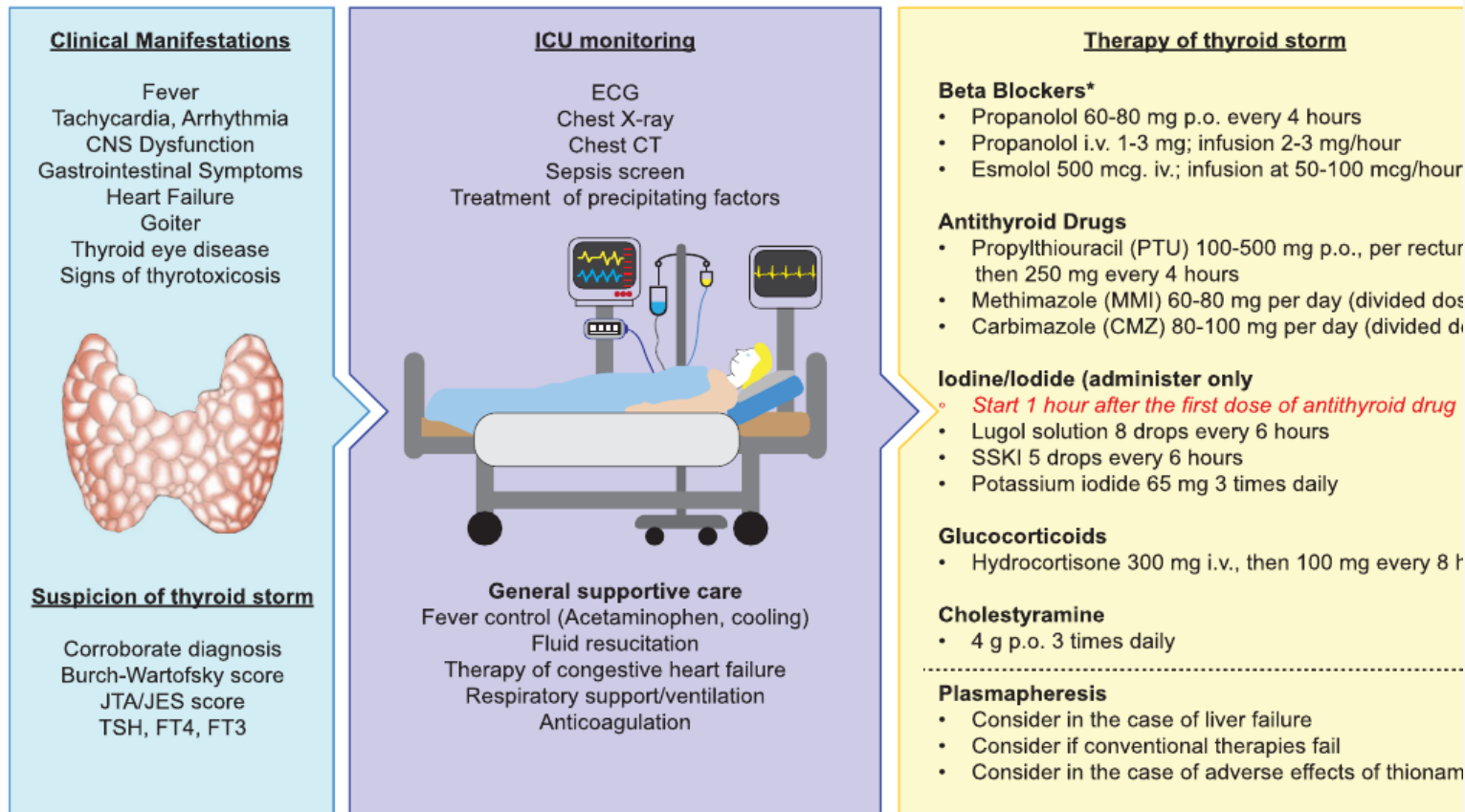
Assess anticoagulation for persistent atrial fibrillation(. (based on their CHADS2 score) and reassess ventricular function after euthyroidism.

Recurrence prevention

Identify the precipitant, address adherence and access barriers, and arrange early endocrine follow-up.

Definitive therapy usually involves radioactive iodine or thyroidectomy, selected according to etiology and comorbidity.(to prevent recurrent thyroid storm)

Treatment targets



Five parallel targets

- 1-Stabilize failing organs
- 2- Control adrenergic effects
- 3- Block hormone synthesis
- 4- Block hormone release
- 5- Treat the precipitating factor

Sequence matters, especially thionamide before iodine.

Figure 1 Diagnosis and management of thyroid storm. For beta-blockers, thyroid medications, and iodine, one of the listed options is in addition to these commonly used beta-blockers, alternative regimens with atenolol, metoprolol, and nadolol are also available, as well as landiolol or

THREE CASES, THREE MANAGEMENT LESSONS



Case 1 AIT + multiorgan failure



Case 2 Fatal treatment errors



Case 3 Guideline-directed rescue

CASE 1: THYROID STORM WITH ACUTE DECOMPENSATED HFrEF

- A 76-year-old man with a two-year history of prior amiodarone exposure presented with a fever of 39.2 °C, tachycardia of 160 (bpm), delirium, profuse diarrhea, jaundice, and bilateral basilar crackles suggestive of heart failure and pneumonia.
- Urgent transthoracic echocardiography (TTE) confirmed acute *Heart Failure with Reduced Ejection Fraction (HFrEF)* decompensation with a left ventricular ejection fraction (LVEF) of 20%, global hypokinesis, left ventricular dilation
- Laboratory findings revealed thyrotoxicosis with a TSH <0.005 mUI/L (0.4-4 mU/L), free T4 > 100 pmol/L (12-21), T3 24 pmol/L. Anti-TSH receptor antibodies were negative.
- A thyroid ultrasound showed an enlarged gland of about 32 mL with heterogeneous hypoechoic areas but no nodules or hypervascularity
- Liver function tests revealed hepatic dysfunction with AST 250 U/L (8-33), ALT of 180 U/L (7-45), and bilirubin 3.2 mg/dL (0.1-1.2).
- Chest X-ray showed bilateral infiltrates consistent with acute pulmonary edema overlaid with patchy consolidations suggestive of multifocal pneumonia.
- Comorbidities included longstanding HFrEF (mixed etiology: hypertensive, ischemic, alcoholic, with paroxysmal atrial fibrillation), chronic kidney disease, a 50-pack-year smoking history, risky alcohol use, and prostate cancer status post-radiotherapy

Case 1: amiodarone and multiorgan failure

BWPS 65

76-year-old man

Definite thyroid storm with cardiac, CNS, hepatic, and pulmonary involvement (multiorgan dysfunction)

Fever 39.2°C, heart rate 160, delirium, diarrhea, jaundice, pneumonia, and LVEF 20%

Teaching lesson: pneumonia triggered crisis in a patient with amiodarone-induced thyrotoxicosis. Mixed type 1 and type 2 mechanisms may coexist, and (Initial ICU management, Thyroid-directed therapy), biochemical recovery may take months.

CASE 2: RAPIDLY PROGRESSIVE MULTI ORGAN FAILURE

- A 72-year-old man presented with acute confusion, palpitations, high-grade fever, dyspnea, and diarrhea after a recent hospitalization for diverticulitis. He was hemodynamically unstable, with atrial fibrillation at 160 bpm, blood pressure of 95/55 mmHg, tachypnea, and oxygen saturation of 90%. Examination revealed a large multinodular goiter, warm moist skin, peripheral edema, and atrial fibrillation with rapid ventricular response.

Initial laboratory testing showed suppressed TSH, leukocytosis, elevated troponin I and mild transaminitis, Peripheral hormones (free T4 and free T3) were ordered.

- Therapy was initiated with intravenous propranolol and furosemide. Aspirin was prescribed as antipyretic therapy, but no other cooling measures were undertaken.
- Oral methimazole (MMI) was not administered due to difficulty swallowing, but Lugol solution at a dose of 10 drops was started. Transfer to a tertiary referral center was planned for the next morning. Within 12 hours, the patient developed progressive hypotension, acidosis, and multiorgan dysfunction. He sustained cardiac arrest and could not be resuscitated.
- Peripheral hormones, received on the following day, showed a free T4 of 61.2 pmol/L (10-22), and a free T3 of 9.1 pmol/L (3.5-6.5), confirming a thyrotoxic profile with a markedly elevated free T4 and a less strikingly increased free T3, possibly in the context of critical illness.

Case 2: treatment errors with a fatal outcome

72-year-old man with toxic multinodular goiter, recent iodinated contrast (triggering factor), rapid AF, hypoxia, and hypotension

No glucocorticoid

Lugol before thionamide

Aspirin for fever

Delayed tertiary transfer and no alternative route for antithyroid therapy

Progressive shock and multiorgan failure developed within 12 hours.

This case emphasizes that thyroid storm requires immediate protocol-based treatment rather than waiting for complete biochemical confirmation or clinical deterioration

CASE 3: THYROID STORM PRESENTING AS CARDIOPULMONARY ARREST

- A 30-year-old female migrant suffered a cardiopulmonary arrest on the street. CPR was initiated within a minute by a first responder. On arrival of the ambulance, she was in ventricular fibrillation, and she was defibrillated.
- Heart rate was normal after 5 minutes of resuscitation. Given persistent Glasgow Coma Scale score 3/15, she was intubated before transfer to a tertiary center. In the emergency room, she was hemodynamically stable, but she had atrial fibrillation with a tachycardic ventricular response.
- The ECG found no evidence of STEMI. A CT of the brain ruled out intracranial bleeding. Thoracic CT showed no pulmonary embolism but revealed right ventricular enlargement, an enlarged pulmonary trunk, and ground-glass opacities suggestive of pulmonary edema.
- TTE showed mild right ventricular dilation and possible pulmonary hypertension. Troponine T was 11 ng/L (<14), creatinine kinase 86 U/L (25-140), and CK MB was 25 U/L (>6% of CK)

CASE 3: THYROID STORM PRESENTING AS CARDIOPULMONARY ARREST

- On admission, TSH was <0.005 mU/L (0.27-4.2), free T4 was 89.1 pmol/L (11.2-24.1), and free T3 was 32.6 pmol/L (3.1-6.8).
- Therapy was initiated immediately with PTU 200 mg every 6 hours, (nasogastric tube.) About 1 hour later, she received a first dose of 65 mg of potassium iodide; it was then administered every 8 hours
- Hydrocortisone 100 mg was given i.v. every 8 hours. Beta blockade was commenced with labetalol 5 mg i.v. and then at a rate of 0.5 mg/min. It was switched to propranolol 60 mg every 6 hours after day 1.
- After extubation, cholestyramine 4 g was given 3 times daily. Free T3 was in the reference range after 3 days (6.8 pmol/L), and both peripheral hormones were within the reference range after 10 days, with a free T4 of 24 pmol/L and a free T3 of 3.5 pmol/L.
- Anticoagulation was started with heparin i.v. and later changed to oral apixaban.
- A non-urgent coronary angiography was negative for coronary artery disease and cardiac MRI did not find alterations suggestive of cardiomyopathy.
- The personal medical history revealed that she had been diagnosed with Graves' disease at age 23 and treated with carbimazole (CMZ), but the patient had stopped the medication about 4 months earlier.
- Follow-up TTE was performed one month later and found no evidence of pulmonary hypertension. Outpatient cardioversion for rhythm control was successfully performed 2 months after admission.

Case 3: successful treatment after cardiac arrest

30-year-old woman with Graves disease, carbimazole withdrawal (precipitating factor), ventricular fibrillation, and cardiac arrest

Immediate rescue

Bystander CPR, rapid defibrillation, intubation, and transfer to a tertiary center enabled continuous ICU monitoring.

Alternative causes of cardiac arrest, including STEMI, pulmonary embolism, and intracranial bleeding, were systematically investigated and excluded

Thyroid storm therapy

PTU by nasogastric tube, potassium iodide one hour later, hydrocortisone, then labetalol followed by propranolol

Response

Cholestyramine was added. FT3 normalized in 3 days and both peripheral hormones normalized in 10 days.

Coronary angiography was deferred until stabilization, illustrating the importance of balancing diagnostic procedures against potential iodine-related risks during uncontrolled thyrotoxicosis

The case also demonstrates that severe thyroid storm may occur despite young age and without longstanding cardiovascular disease.

The patient's migrant status, limited resources, socioeconomic difficulties, and linguistic barriers may have complicated access to continuous treatment

These circumstances highlight the importance of identifying barriers to medication adherence and continuity of care after initial stabilization

Practical bedside algorithm

1**ICU monitoring and organ support****2****Treat precipitant and control temperature****3****Hemodynamically appropriate beta-blockade****4****PTU or methimazole****5****Iodine at least one hour later****6****Hydrocortisone and cholestyramine**

Refractory crisis or drug contraindication: plasma exchange, then selected surgery

Take-home points

Treat on clinical suspicion. Do not wait for FT4 or FT3.

1

Organ decompensation, not the hormone concentration, defines the crisis.

2

Thionamide precedes iodine. Avoid aspirin. Use beta-blockers cautiously in heart failure.

3

Early recognition and multimodal ICU care change survival.



THANK YOU FOR LISTENING