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THYROID STORM IN PRIMARY CARE AND EMERGENCY DEPARTMENTS: A SYSTEMATIC REVIEW OF EARLY WARNING SIGNS AND STABILIZATION PROTOCOLS

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ABSTRACT

Background: Thyroid storm is a rare but catastrophic endocrine emergency arising from decompensated, severe thyrotoxicosis. It triggers rapid multi-organ failure and carries a high mortality rate, particularly among critically ill patients and geriatric populations with significant comorbidities. Because its early constitutional signs are easily misattributed, frontline clinical vigilance is crucial for timely intervention.
Aim: This review synthesizes early warning signs, precipitating stressors, diagnostic frameworks, and acute stabilization protocols to guide primary care and emergency clinicians.
Materials and Methods: A systematic review of contemporary literature, clinical registries, and international guidelines published between 2016 and 2026 was conducted to evaluate clinical phenotypes, triggering factors, diagnostic criteria, and acute multi-targeted pharmacological strategies.
Results: A thyroid storm is almost universally precipitated by acute secondary stressors, such as medication non-compliance, infections, surgery, or trauma. While younger populations typically present with a hyperkinetic phenotype (hyperthermia, severe tachycardia, and central nervous system agitation), elderly patients frequently display an atypical "apathetic" phenotype. This apathetic presentation lacks dramatic hypermetabolic features but carries a high risk of covert, life-threatening cardiac complications. From a diagnostic standpoint, the Japan Thyroid Association (JTA) guidelines mandate objective biochemical confirmation using a combinatory algorithm, whereas the Burch-Wartofsky Point Scale (BWPS) relies on a cumulative, symptom-weighted score. Emergency management requires a strict, multi-targeted pharmacological regimen: beta-blockers (propranolol or esmolol) for hyperadrenergic cardiotoxicity, thionamides to halt hormone synthesis, timed inorganic iodine to block hormone release, and glucocorticoids to counter relative adrenal insufficiency. Refractory circulatory collapse requires advanced extracorporeal rescue systems, including therapeutic plasma exchange (TPE) and Extracorporeal Membrane Oxygenation (ECMO).
Conclusion: Combating the high mortality of a thyroid storm depends on a seamless, interprofessional continuum of care. Primary care physicians form the first line of defense through outpatient risk mitigation and medication optimization. When a crisis occurs, emergency clinicians must prioritize clinical acumen over rigid scoring algorithms, especially in apathetic presentations, to immediately initiate aggressive stabilization and secure prompt intensive care unit (ICU) containment.

KEYWORDS

Thyroid Storm, Thyroid Crisis, Decompensated Thyrotoxicosis, Emergency Stabilization, Burch-Wartofsky Point Scale, Japan Thyroid Association Criteria

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1. Introduction

Thyroid storm, otherwise known as thyroid crisis, is an acute, life-threatening condition that develops from severe thyrotoxicosis, frequently presenting as the initial manifestation of an undiagnosed hyperthyroid state. This medical emergency involves multi-organ dysfunction driven by excessive thyroid hormone (T3 and T4) production, regardless of the underlying source (Ross et al., 2016). Epidemiologically, it has a low general prevalence of 0.2–1.4 per 100,000 persons per year (Akamizu, 2018; Thiyagarajan et al., 2022), rising to 4.8–5.6 per 100,000 among hospitalized patients (Galindo et al., 2019). While a recent US study reported a baseline mortality rate of 5.3% (Seo et al., 2024), this figure can sharply escalate in elderly populations with significant comorbidities, and may exceed 10% to 30% among critically ill patients demonstrating acute organ failure (Bourcier et al., 2020; Ono et al., 2016; Satoh et al., 2016; Thiyagarajan et al., 2022). The condition predominantly affects middle-aged women, with a documented female-to-male ratio of 3:1 (Kornelius et al., 2021; Lath et al., 2024; Mallavarapu et al., 2025; Thiyagarajan et al., 2022). Because its early clinical signs are easily dismissed or misattributed, it is crucial for primary care and emergency physicians to maintain a high level of clinical vigilance, acting as the frontline of an interprofessional team dedicated to early recognition and aggressive intervention.

2. Etiology of Thyroid Storm

Hyperthyroidism precipitating a thyroid storm is most frequently caused by Graves' disease, accounting for the vast majority of clinical cases. Remarkably, a thyroid crisis can occasionally serve as the initial presentation of unrecognized or untreated Graves' hyperthyroidism, catching clinicians off guard (Kopp et al., 2026).

Less common yet significant etiologies include autonomous thyroid nodules, such as toxic multinodular goiter (TMNG) and toxic adenoma (Ross et al., 2016; Satoh et al., 2016). Additionally, drug-induced thyrotoxicosis represents a growing clinical challenge. Most notably, the prolonged or chronic use of amiodarone can trigger either destructive thyroiditis (Type 2) or iodine-induced hyperthyroidism (Type 1), both of which can culminate in a crisis (Malipeddi et al., 2025; Takemoto & Takada, 2020; Theodoraki & Vanderpump, 2016).

Among the rarest underlying causes documented in the literature are acute or subacute thyroiditis (Newman & Walthall, 2022), hydatidiform moles (where high levels of human chorionic gonadotropin cross-react with TSH receptors) (Jiménez-Labaig et al., 2022), and functional, metastatic hormone-secreting thyroid carcinomas (Kwon et al., 2023). Modern oncological strategies have also introduced new risks, with therapies involving tyrosine kinase inhibitors (TKIs) or checkpoint inhibitors (immunotherapy) increasingly implicated in severe immune-mediated thyroid dysfunction (Basolo et al., 2022; McMillen et al., 2016). Lastly, factitious thyrotoxicosis resulting from the inadvertent or intentional overdose of exogenous thyroid hormone tablets remains a rare but critical etiology (Du et al., 2022).

3. Precipitating Factors

A thyroid storm rarely occurs in isolation. Rather, it is almost universally precipitated by an acute underlying medical stressor or iatrogenic event superimposed on poorly controlled hyperthyroidism. Identifying these triggering factors is crucial for both diagnosis and immediate therapeutic intervention. Clinically, these underlying inciting factors can be grouped into several distinct patterns of physiological and iatrogenic stress.

Prominent among these is medication and treatment disruption, where the abrupt discontinuation of or non-adherence to antithyroid drug therapy stands out as one of the most common catalysts (Kopp et al., 2026). Additionally, interventions that directly manipulate thyroid tissue or introduce high iodine loads can paradoxically trigger a crisis. Notable examples include radioactive iodine therapy and a recent administration of iodinated contrast media (Basida et al., 2021; Vennard & Gilbert, 2018). Physical stress in the form of surgical and physical trauma represents another classic category of precipitants. Both thyroidectomy and major non-thyroidal surgeries can induce a crisis due to immense systemic stress or the potential intraoperative manipulation of the gland (de Mul et al., 2021; Shao et al., 2024), while severe physical trauma, extensive thermal burns, and traumatic brain injury (TBI) can similarly disrupt metabolic homeostasis acutely to precipitate a storm (Abdelmageed et al., 2026; Jinno et al., 2024; Sofianos et al., 2017).

Furthermore, acute cardiovascular and neurological events frequently serve as the physiological driving force behind this condition, as these critical systemic illnesses induce a profound hypermetabolic response. Specifically, acute myocardial infarction, heart failure exacerbations, and cerebrovascular accidents (CVA)

are well-documented stressors capable of unmasking an underlying thyroid crisis (Snyder & Joseph, 2020; Zayour et al., 2023). In a similar manner, profound metabolic and infectious stressors are heavily implicated. Severe systemic infections, including respiratory tract infections and COVID-19, often act as the tipping point, while acute metabolic derangements, most notably diabetic ketoacidosis, present a high-risk scenario due to the compounding effects of severe physiological stress and altered thyroid hormone binding (Bellamkonda et al., 2023; Keyal et al., 2020; Rangoonwala et al., 2024). Finally, obstetric conditions introduce unique physiological demands during pregnancy that carry inherent risks. Parturition itself functions as a massive physical trigger, while severe hyperemesis gravidarum can severely aggravate thyrotoxicosis, driven by high human chorionic gonadotropin (hCG) levels cross-reacting with TSH receptors (Ma et al., 2018; Saroyo et al., 2021; Zimmerman et al., 2022).

4. Clinical Presentation

Patients in a thyroid storm classically present with an extreme amplification of the signs and symptoms typical of thyrotoxicosis. On initial physical examination, clinicians frequently note a diffuse or nodular goiter alongside characteristic features of thyroid eye disease, such as exophthalmos or ophthalmopathy (Wiersinga et al., 2023). Beyond these primary thyroid signs, the systemic manifestations of a thyroid crisis vary in severity and typically present in a complex, multi-organ constellation.

Thermoregulatory dysfunction serves as a hallmark feature of this state, manifesting as a high fever with temperatures frequently exceeding 38.5°C (101.3°F), which is often accompanied by profuse diaphoresis (Farooqi et al., 2023; Kopp et al., 2026). Concurrently, profound cardiovascular stress is a prominent clinical finding, characteristically involving a severe tachycardia that is disproportionate to the severity of the fever. As the crisis progresses, patients may develop various tachyarrhythmias, most notably atrial fibrillation, as well as congestive heart failure, pulmonary edema, reversible dilated cardiomyopathy, or Takotsubo cardiomyopathy (H. Ali et al., 2021a; Burakiewicz et al., 2019; Marino et al., 2021; Muhammad Priangga Akbar & Arie Zainul Fatoni, 2025; Sourial et al., 2019; Taylor et al., 2019). This hypermetabolic strain extends heavily into the central nervous system, where neurological involvement spans a wide spectrum. Patients may experience initial marked agitation, anxiety, and restlessness, which can rapidly deteriorate into severe confusion, delirium, psychosis, seizures, and ultimately, coma (Asif et al., 2022; Kara et al., 2017; Sharp et al., 2016). Furthermore, gastrointestinal distress is a common and distressing component of the clinical picture, typically presenting as nausea, vomiting, abdominal pain, and significant diarrhea (Kopp et al., 2026; Muneem et al., 2021; Ritter & Wolfe, 2020). Notably, hepatic involvement can also occur and may manifest objectively as jaundice, a finding that typically signals a much poorer clinical prognosis (Vidanapathirana & Wijayaratne, 2024).

Although rare, a thyroid crisis can manifest atypically as "apathetic" or "masked" hyperthyroidism. A presentation observed predominantly in elderly populations. This phenotype lacks the classic hyperkinetic features of thyrotoxicosis. Instead, it is characterized by profound depression, apathy, severe weight loss, dry skin, muscle atrophy, chronic fatigue, and blepharoptosis. Despite the absence of dramatic hypermetabolic symptoms, cardiac complications, such as mild tachycardia, congestive heart failure, and atrial fibrillation accompanied by dyspnea, remain highly prevalent and require vigilant monitoring (A. Ali et al., 2020; H. Ali et al., 2021b; Bhandari et al., 2026; Farooqi et al., 2023).

Table 1. Classic vs Apathetic Thyroid Storm presentation.

Dysfunction	Classic presentation	Apathetic presentation
Primary Thyroid and Ocular	Diffuse or nodular goiter Exophthalmos Ophthalmopathy	Blepharoptosis
Thermoregulation and Skin	High fever Profuse diaphoresis	Dry skin
Cardiovascular System	Severe tachycardia Atrial fibrillation Congestive heart failure Pulmonary edema Dilated cardiomyopathy Takotsubo cardiomyopathy	Mild tachycardia, Congestive heart failure Atrial fibrillation Dyspnea
Central Nervous System	Agitation Anxiety Restlessness Severe confusion Delirium Psychosis Seizures Coma	Profound depression Apathy
Gastrointestinal and Hepatic	Nausea Vomiting Abdominal pain Significant diarrhea Jaundice	—
Systemic / Metabolic	—	Muscle atrophy Chronic fatigue Severe weight loss

5. Differential Diagnosis

The differential diagnosis of a thyroid storm is exceptionally complex, demanding extensive clinical scrutiny due to its profound phenotypic overlap with a myriad of acute illnesses that can simultaneously coexist with, or masquerade as, the crisis itself (Khurana et al., 2023). Clinicians must maintain a high level of vigilance and recognize that the clinical presentation represents a distinct diagnostic illusion. In essence, every individual, constitutional symptom manifesting during a thyroid crisis may not be a direct consequence of thyrotoxic collapse. Instead, each symptom can paradoxically stem from an entirely independent underlying pathology or represent a completely separate, co-occurring disease entity, severely complicating timely diagnostic closure. To ensure precise clinical differentiation, the following primary etiologies must be systematically evaluated and ruled out: sepsis and septic shock, acute pulmonary edema, aortic dissection, sympathomimetic toxicity, anticholinergic toxicity, neuroleptic malignant syndrome, serotonin syndrome, alcohol withdrawal, malignant hyperthermia, pheochromocytoma crisis, heat stroke, and also primary psychiatric emergencies like acute manic disorder, sever panic disorder and schizophrenic psychosis (Kopp et al., 2026).

6. Diagnostic Evaluation and Criteria

Diagnosing a thyroid storm remains a distinct challenge, as a predominantly clinical diagnosis, it lacks definitive, objective laboratory thresholds or biomarkers for confirmation. To mitigate diagnostic ambiguity, validated clinical scoring systems that systematically evaluate and weight thyrotoxic symptoms have become indispensable tools. Globally, two principal frameworks guide clinicians through the diagnostic process: the Burch-Wartofsky Point Scale and the Akamizu (Japan Thyroid Association) criteria.

The Burch-Wartofsky Point Scale (BWPS) is a standardized scoring system used to objectify the diagnosis of a thyroid storm. The system assigns weighted scores based on the severity of specific clinical manifestations, including thermoregulatory dysfunction, central nervous system effects, cardiovascular stress, and gastrointestinal-hepatic symptoms. Additionally, the presence of an identifiable precipitating factor is factored into the cumulative score. Once the points are aggregated, a specific diagnostic threshold dictates clinical management: a score below 25 points makes a thyroid storm unlikely, a score between 25 and 44 implies an impending crisis, and a cumulative score of 45 or greater is highly suggestive of an active thyroid storm, warranting immediate, aggressive intervention. Additionally, the Adapted Burch-Wartofsky Criteria represent a modified version of the original framework, specifically tailored to pediatric populations to account for their age-specific physiological parameters. (Elendu et al., 2024; Kopp et al., 2026; Nagamine et al., 2025)

Table 2. Burch-Wartofsky Point Scale

Criteria	Points	Criteria	Points
Thermoregulatory dysfunction – Body temperature		*Gastrointestinal and hepatic dysfunction*	
37.2–37.7°C (99.0–99.9°F)	5	Absent	0
37.8–38.3°C (100.0–100.9°F)	10	Moderate – diarrhea, abdominal pain, nausea, vomiting	10
38.4–38.8°C (101.0–101.9°F)	15	Severe – jaundice	20
38.4–38.8°C (102.0–102.9°F)	20	*Central nervous system disturbance*	
39.4–39.9°C (103.0–103.9°F)	25	Absent	0
≥ 40°C (≥ 140°F)	30	Mild – agitation	10
Cardiovascular dysfunction		Moderate – delirium, psychosis, extreme lethargy	20
Tachycardia – HR (beats per minute)		Severe – seizure coma	30
90–109	5	*Precipitant history*	
110–119	10	Absent	0
120–129	15	Present	10
130–139	20		
≥ 140	25		
Congestive heart failure			
Absent	0		
Mild – pedal edema	5		
Moderate – bibasilar rales	10		
Severe – pulmonary edema	15	**Score totaled**	
Atrial fibrillation		Thyroid storm	≥45
Absent	0	Impending Storm	25 – 44
Present	10	Storm unlikely	<25

In contrast to the purely symptom-driven Burch-Wartofsky Point Scale, the Japan Thyroid Association (JTA) diagnostic criteria require objective biochemical confirmation of thyrotoxicosis (elevated free T3 or free T4) as a fundamental baseline. Beyond this laboratory requirement, the framework evaluates five core clinical domains: fever, tachycardia/atrial fibrillation, congestive heart failure, gastrointestinal/hepatic dysfunction, and central nervous system (CNS) manifestations. Rather than using a cumulative point system, the JTA model utilizes a combinatory algorithm to classify cases into two diagnostic categories: definite thyroid storm and suspected thyroid storm. Definite Thyroid Storm (TS1) is diagnosed when biochemical thyrotoxicosis is accompanied by either a severe CNS manifestation plus one other systemic symptom, or any three non-CNS systemic symptoms are combined. Suspected Thyroid Storm (TS2) is designated when combinations of non-

CNS manifestations are present, or when a patient meets all clinical criteria, but lacks a confirmed precipitating cause or baseline hormone level availability. (Elendu et al., 2024; Furukawa et al., 2025; Satoh et al., 2016)

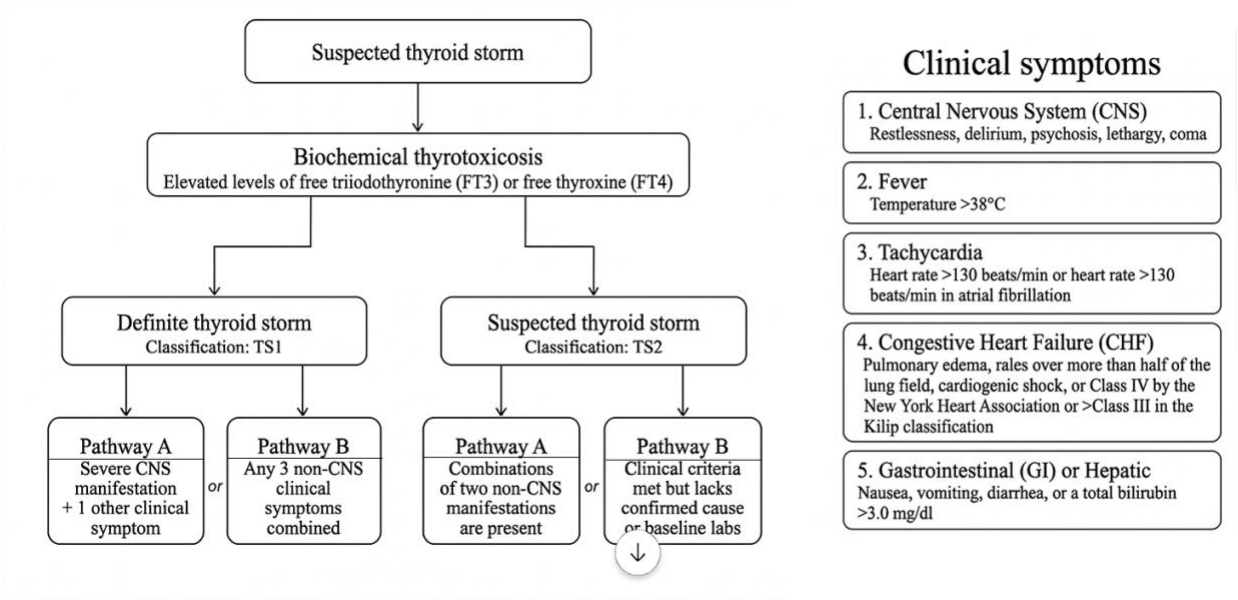

Fig. 1. *Diagram of Japan Thyroid Association diagnostic criteria.*

Beyond formalized scoring systems, several auxiliary laboratory markers provide valuable diagnostic support and insight into disease severity. Historically, an extreme elevation of thyroid hormones, often exceeding three times the upper limit of normal (ULN), has been noted as a supportive biochemical indicator (Satoh et al., 2016). More recently, emerging diagnostic focus has shifted toward systemic inflammatory markers, such as C-reactive protein (CRP) and interleukin-6 (IL-6), which may reshape future prognostic stratification entirely (Elendu et al., 2024). In addition to these acute-phase reactants, case-based reports increasingly highlight markers of metabolic collapse, specifically elevated lactate levels and profound metabolic acidosis, as critical signals of impending multi-organ failure (Bhuiya et al., 2025; IZUMI et al., 2009; Prosser & Quan, 2015; Yoshino et al., 2010). Although definitive baseline thresholds for serum lactate or arterial blood pH in a thyroid storm have not yet been established through large-scale cohort or registry data, these metabolic parameters represent a promising frontier in research, serving as vital 'red flag' indicators for early risk stratification.

While objective scoring frameworks provide essential diagnostic scaffolding, they cannot entirely supplant independent clinical acumen, particularly when managing atypical presentations of a thyroid storm. In these highly nuanced clinical scenarios, the experience of the treating clinician remains the definitive tool for interpreting subtle deviations that standardized protocols inherently overlook. This diagnostic tension was elegantly highlighted in a comparative review by Elendu et al. (2023), which juxtaposed these established diagnostic criteria and highlighted several shared limitations. Notably, existing scales often lack diagnostic sensitivity in milder or early-stage cases of thyroid storm. Furthermore, they frequently fail to adequately account for variations across different age groups, the presence of complex multimorbidities, or the challenge of differentiating a thyroid crisis from other critical systemic illnesses (Elendu et al., 2024). Consequently, these scoring systems still rely heavily on the clinician's subjective assessment, which can lead to diagnostic variability if the examiner lacks extensive experience. Ultimately, while diagnostic criteria are invaluable tools, they must be applied flexibly on a case-by-case basis with a clear awareness of their inherent limitations.

7. Therapeutic Management

7.1 Proactive Outpatient Intervention and Risk Mitigation

The therapeutic management of a thyroid storm does not begin exclusively upon emergency department presentation. Rather, it encompasses a preventative continuum initiated in the outpatient setting. When a patient with underlying hyperthyroidism encounters before mentioned acute physiological stressors, such as an unmanaged infection, severe physical trauma, or a pending surgical intervention, the primary care practitioner must act preemptively to avert a precipitous descent into systemic crisis.

First, the clinician must meticulously verify medical compliance. Because the abrupt discontinuation of or non-adherence to antithyroid drug (ATD) therapy stands out as the leading iatrogenic catalyst for a thyroid storm, the immediate re-establishment of compliance is paramount. Second, if the patient exhibits early, emerging signs of systemic or autonomic decompensation characterized by unexplained tachycardia, low-grade hyperthermia, or escalating psychomotor agitation, the clinician should aggressively titrate the ATD upward to its maximum safe clinical dose (Kahaly et al., 2018; Ross et al., 2016). Simultaneously, the preemptive addition of a beta-blocker, such as propranolol, is heavily indicated to counteract hyperadrenergic cardiotoxicity and safeguard the cardiovascular system (Kahaly et al., 2018; Ross et al., 2016). When these early prophylactic interventions fail to stabilize the patient, or if the clinical presentation breaches these outpatient defenses, immediate escalation to acute care is required.

7.2 Acute Emergency Stabilization and Supportive Care

Treatment in the emergency department relies on stabilizing and providing supportive care to organ systems currently exhibiting, or vulnerable to developing, clinical manifestations of a thyroid storm. This requires a dual approach balancing general supportive care with definitive antithyroid treatment, while any accompanying triggers, such as systemic infections, must be identified and treated simultaneously.

Beginning with the management of hyperthermia, clinicians should promptly implement temperature-lowering therapy. Acetaminophen is the preferred antipyretic because, unlike nonsteroidal anti-inflammatory drugs (NSAIDs), it does not displace thyroid hormones from thyroid-binding globulin, which would otherwise increase freely circulating free T4 levels (De Almeida et al., 2022; Farooqi et al., 2023; Kopp et al., 2026; Satoh et al., 2016). Additionally, aggressive physical cooling techniques, including the application of cold compresses or cooling blankets, are recommended (De Almeida et al., 2022; Farooqi et al., 2023; Kopp et al., 2026; Satoh et al., 2016). Supplemental oxygen therapy should be initiated immediately in patients exhibiting signs of dyspnea or respiratory distress (De Almeida et al., 2022; Farooqi et al., 2023; Kopp et al., 2026; Satoh et al., 2016). Furthermore, the administration of intravenous (IV) fluids must be conducted with extreme caution. Clinicians must strike a careful balance between providing adequate rehydration, required by excessive fluid loss from diarrhea and vomiting, and avoiding fluid overload, which carries a severe risk of precipitating circulatory congestion, pulmonary edema, and acute heart failure (De Almeida et al., 2022; Farooqi et al., 2023; Kopp et al., 2026; Satoh et al., 2016).

7.3 Multi-Targeted Pharmalogical Approach

The definitive management of a thyroid storm requires a multi-targeted pharmacological approach aimed at every stage of thyroid hormone synthesis, release, and peripheral action. Immediate suppression of the hyperadrenergic state is accomplished via beta-blockers, typically propranolol, which uniquely provides the dual benefit of reducing peripheral T4-to-T3 conversion (De Leo et al., 2016; Ross et al., 2016). However, some authors advocate for the use of short-acting, cardioselective beta-blockers, such as esmolol, citing a lower risk of overall mortality (El-Menyar et al., 2025; Satoh et al., 2016). In contrast, a recent retrospective cohort study by Matsuo et al. found that no specific beta-blocker was associated with a higher risk of death, even in the presence of acute heart failure (Matsuo et al., 2024). To avoid precipitating further hemodynamic decompensation, performing an echocardiogram to evaluate the patient's ejection fraction (EF) should ideally precede beta-blocker administration, particularly when severe heart failure or a low EF is clinically suspected (Farooqi et al., 2023; Satoh et al., 2016). Ultimately, the immediate therapeutic goal of beta-blocker therapy is to reduce and maintain the patient's heart rate below 130 beats per minute (Farooqi et al., 2023; Satoh et al., 2016).

Concurrently, thyroid hormone synthesis must be halted using thionamides, with methimazole (MMI) and propylthiouracil (PTU) serving as the two primary agents. Recent studies show no significant differences in mortality, disease severity, organ support duration, or adverse events between PTU and MMI users (Lee et al., 2023). However, PTU is traditionally preferred and recommended by the American Thyroid Association

(ATA) due to its additional property of inhibiting peripheral T4-to-T3 conversion (Ross et al., 2016). Conversely, methimazole is often favored in Japanese guidelines due to its favorable hepatic safety profile and longer half-life (Satoh et al., 2016). Patient demographics also dictate selection: during the first trimester of pregnancy, PTU is the preferable option to avoid embryopathy, while methimazole is favored during the remainder of the gestational period (Alexander et al., 2017; Lee & Pearce, 2021).

Inorganic iodine therapy is utilized to exploit two distinct, powerful intrathyroidal physiological mechanisms. First, it induces a transient autoregulatory shutdown of thyroid hormone organification and synthesis via the Wolff–Chaikoff effect (Kopp et al., 2026; Ross et al., 2016). Second, it directly and rapidly inhibits the proteolytic release of preformed thyroid hormones from the colloid into the bloodstream, a phenomenon known as the Plummer effect (Kopp et al., 2026; Ross et al., 2016).

According to Western guidelines established by the American Thyroid Association (ATA), the administration of iodine compounds, such as Lugol's solution, a saturated solution of potassium iodide (SSKI), or potassium iodide tablets, must be strictly delayed until at least one hour after thionamides therapy has been initiated (Ross et al., 2016). Administering iodine prematurely introduces an immediate substrate influx that can accelerate thyroid hormone synthesis, risking a severe, paradoxical aggravation of the thyroid crisis (Ross et al., 2016). Conversely, guidelines from the Japan Thyroid Association (JTA) note that simultaneous administration of iodine alongside thionamides yields highly positive clinical outcomes in thyroid storms secondary to Graves' disease, without demonstrating an increased risk of worsening thyrotoxicosis (Satoh et al., 2016).

In scenarios where a patient presents with a documented, severe iodine hypersensitivity, lithium carbonate serves as a valuable alternative therapeutic agent to block glandular thyroid hormone release (Kopp et al., 2026; Sharma, 2022). However, its clinical utility is heavily constrained by a narrow therapeutic index and significant risks of severe nephrotoxicity and neurotoxicity, necessitating rigorous serum concentration monitoring (Kopp et al., 2026; Sharma, 2022).

Glucocorticoids represent another critical therapeutic class utilized during acute stabilization. Beyond their ability to reduce the peripheral conversion of T4 to T3, corticosteroids play an indispensable protective role against relative adrenal insufficiency – a life-threatening complication driven by the hypermetabolic acceleration of cortisol clearance during a crisis (Kopp et al., 2026; Ross et al., 2016).

Additionally, bile acid sequestrants, predominantly cholestyramine, can be introduced as adjuncts to lower circulating thyroid hormone levels (Kopp et al., 2026; Ross et al., 2016). These agents work by binding thyroid hormones within the gastrointestinal lumen, effectively disrupting enterohepatic recirculation and preventing their reabsorption (Kopp et al., 2026; Ross et al., 2016).

7.4 Neuropsychiatric Management

Because neuropsychiatric manifestations are strictly secondary to severe thyrotoxicosis rather than a primary psychiatric disorder, frontline therapeutic efforts must focus entirely on the rapid, aggressive suppression of the underlying thyroid storm rather than conventional antipsychotic regimens. However, if severe psychomotor agitation, hallucinations, or delusions persist despite anti-thyroid therapy, standard atypical antipsychotic medications, such as risperidone or olanzapine, should be implemented (Sharp et al., 2016). In the event of acute thyrotoxic seizures, intravenous benzodiazepines remain the standard initial first-line intervention (Sharp et al., 2016).

7.5 Hemodynamic Collapse and Advanced Rescue Systems

When progressive hemodynamic instability occurs, therapeutic management follows a strict, rapid escalation pathway meticulously adjusted to clinical severity. In the presence of acute cardiac rhythm disturbances, immediate synchronized electrical cardioversion is indicated for unstable tachyarrhythmias, whereas standard pharmacological rate-control protocols are maintained for hemodynamically stable presentations (El-Menyar et al., 2025; Farooqi et al., 2023; Satoh et al., 2016). Should the clinical picture worsen into formal shock, active pharmacological resuscitation is initiated. This requires the preemptive introduction of tailored inotropic agents and vasopressors designed specifically to combat early-stage cardiogenic shock and high-output cardiac failure (El-Menyar et al., 2025; Farooqi et al., 2023; Satoh et al., 2016). Finally, for patients facing refractory circulatory collapse that fails to respond to conventional medical therapy, standard protocols dictate rapid escalation to advanced extracorporeal rescue systems. This includes the implementation of therapeutic plasma exchange (TPE) to mechanically strip excess thyroid hormones from

their binding proteins (Osorio-Toro et al., 2025), utilized alongside mechanical circulatory support systems such as extracorporeal membrane oxygenation (ECMO) (Lim et al., 2021).

Given the imminent risk of multi-organ collapse, immediate admission to an intensive care unit (ICU) is mandatory to ensure continuous hemodynamic monitoring, aggressive metabolic stabilization, and potential invasive ventilatory support (Farooqi et al., 2023; Kopp et al., 2026; Satoh et al., 2016).

Table 3. Dosage of medications used to treat thyroid storm.

Class of medication	Medication	Dosage	Special remarks
Beta-blockers	Propranolol	PO: 60–80 mg every 4h or 80–120 mg every 6h IV: 0.5–1 mg slow push, then 1–2mg every 15 min	Preemptive echocardiography should be considered when CHF is suspected.
	Esmolol	IV: 250–500 μg/kg of bolus, then an infusion rate of 50–100 μg /kg/min	Preemptive echocardiography should be considered when CHF is suspected.
Thionamides	Methimazole	PO: loading dose of 40 mg, then 20 mg every 4h PR: 20–40 mg every 6h p.r IV: 10–30 mg every 6h	Fewer adverse effects.
	Propylothiouracil	PO: loading dose of 500–1000 mg followed by 250 mg every 4 h PR: 400–600 mg every 6 h	Preferred during first trimester of pregnancy.
Iodine	Potassium iodine (SSKI)	PO: 5 drops every 6 h PR: 250–500 mg every 6 h	To be given 1 h after administration of thionamides to prevent its use as a substrate for new thyroid hormone production.
	Lugol's solution	PO: 8 drops every 6 h PR: 80 drops per day/5–10 drops every 6 h	To be given 1 h after administration of thionamides to prevent its use as a substrate for new thyroid hormone production.
	Lithium	PO: 300 mg every 6–8 h	Alternative for patients allergic to iodine.
Steroids	Hydrocortisone	IV: 300 mg loading dose, followed by 100 mg every 8 h	
	Dexamethasone	IV: 2 mg every 6 h up to 8 mg/day	
Bile acid sequestrants	Cholestyramine	PO: 4 mg every 6 h	

Abbreviations used: PO – per os, PR – per rectum, IV – intravenous, CHF – congestive heart failure.

8. Conclusions

Thyroid storm is a rare, life-threatening endocrine emergency characterized by decompensated thyrotoxicosis and precipitous multi-organ failure. Because serum thyroid hormone levels correlate poorly with clinical severity, diagnosis relies entirely on frontline clinical vigilance and validated scoring frameworks rather than objective laboratory values. Optimizing patient outcomes depends on a seamless, interprofessional continuum of care that begins with primary care physicians, who form the first line of defense through the longitudinal management of hyperthyroidism. In the outpatient setting, these practitioners must proactively identify potential precipitating factors of thyroid storm and adjust the dosage of antithyroid medications accordingly to prevent stable thyrotoxicosis from escalating into a metabolic crisis.

When a crisis does breach these outpatient defenses, emergency department physicians serve as the critical frontline against acute systemic decompensation. Emergency clinicians must maintain a low threshold for diagnostic consideration, applying clinical criteria dynamically while prioritizing clinical acumen over rigid algorithms, particularly in atypical presentations. Once a crisis is suspected, the immediate role of the emergency provider is to initiate aggressive stabilization protocols and secure prompt intensive care

containment. Ultimately, mitigating the high mortality of a thyroid storm depends on this tightly coordinated sequence, where early, proactive outpatient risk management transitions seamlessly into rapid, life-saving intervention in the emergency department.

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