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THYROID STORM: FROM ENDOCRINE CRISIS TO CARDIOVASCULAR COLLAPSE — PATHOPHYSIOLOGY, DIAGNOSIS AND CONTEMPORARY MANAGEMENT

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ABSTRACT

Thyroid storm is a rare but life-threatening endocrine emergency characterized by thyrotoxicosis accompanied by systemic decompensation and end-organ dysfunction. Despite advances in diagnosis and treatment, it remains associated with substantial mortality, cardiovascular failure and multiorgan dysfunction representing major causes of death. This narrative review aims to summarize current knowledge regarding the pathophysiology, clinical presentation, diagnostic approaches, cardiovascular complications, and contemporary treatment strategies for thyroid storm, with particular emphasis on the management of severe cardiovascular manifestations. A narrative literature review was conducted using PubMed and Google Scholar. Recent peer-reviewed studies, systematic reviews, case series, clinical guidelines, and international consensus recommendations were analyzed to provide an up-to-date overview of thyroid storm management. Thyroid storm requires rapid recognition based on clinical manifestations of systemic decompensation in the setting of biochemical thyrotoxicosis. Cardiovascular complications, including tachyarrhythmias, atrial fibrillation, acute heart failure, thyrotoxic cardiomyopathy, myocardial injury, cardiogenic shock, and cardiac arrest, are among the most clinically significant complications. Treatment requires a multimodal approach - antithyroid drugs, iodine, glucocorticoids, β-blockers, and supportive intensive care. Propranolol remains an important therapeutic option; however, β-blockade must be individualized in patients with impaired cardiac function or hemodynamic instability. Alternative and rescue therapies, including lithium, cholestyramine, therapeutic plasma exchange, and thyroidectomy, may be considered in refractory cases. Successful management of thyroid storm requires early diagnosis, prompt treatment, continuous cardiovascular monitoring, and an individualized multidisciplinary approach. Further studies are needed to establish optimal treatment strategies, particularly for patients with cardiovascular complications and those who fail or cannot tolerate conventional therapy.

KEYWORDS

Thyrotoxicosis, Thyroid Storm, Organ Failure, Cardiac Arrest, Tachyarrhythmia

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Introduction

Thyroid storm represents the most severe and life-threatening form of thyrotoxicosis. The diagnosis is established based on biochemical evidence of hyperthyroidism, characterized by suppressed or undetectable serum thyroid-stimulating hormone (TSH) concentrations accompanied by elevated circulating free thyroxine (fT4) and/or triiodothyronine (T3) levels. In patients with Graves' disease the presence of thyroid-stimulating hormone receptor antibodies (TRAb) further supports the underlying etiology. However, laboratory findings alone are insufficient for diagnosis, which also requires the presence of specific clinical manifestations indicating acute systemic decompensation and end-organ dysfunction. [1]

This life-threatening endocrine emergency occurs more frequently in women than in men. Its estimated incidence ranges from 0.2 to 1.1 per 100,000 individuals in general population. In spite of advances in medical management, thyroid storm continues to be associated with a substantial mortality rate of approximately 5-25%, with cardiovascular failure and multiorgan dysfunction representing the leading causes of death. [3]

The diagnosis of thyroid storm may be supported by several validated diagnostic scoring systems such as the Burch-Wartofsky Point Scale (BWPS), the Japanese Thyroid Association (JTA) scale and the Akamizu criteria. These diagnostic tools integrate both clinical manifestations and biochemical evidence of thyrotoxicosis which are crutial to identificate thyroid storm. [2] Furthermore, elevated levels of the inflammatory markers, such as C-reactive protein (CRP) and interleukin-6 (IL-6), may also contribute to diagnosis of thyroid storm. [2]

The primary objective of thyroid storm management is the prompt restoration of euthyroidism. Once metabolic stabilization has been achieved, therapeutic efforts should be directed toward the identification and correction of the underlying precipitating cause of the hypermetabolic state.

Methodology

A narrative literature review was conducted to summarize the current evidence regarding the pathophysiology, diagnosis and contemporary management of thyroid storm. A comprehensive search of the literature was performed using the PubMed and Google Scholar databases. Priority was given to recent peer-reviewed publications, high-quality review articles and international clinical guidelines. The retrieved literature was critically evaluated and the findings were synthesized to provide an up-to-date overview of the current understanding of thyroid storm with particular emphasis on its pathophysiology, diagnostic criteria and emerging therapeutic approaches.

Discussion

Symptoms and diagnosis

Thyroid storm may be caused by a variety of factors, including infections (especially sepsis), surgical procedures and discontinuation or improper use of antithyroid medication. [3] In addition, cases associated with the administration of immune checkpoint inhibitors, such as pembrolizumab, have been reported [4]. Such a severe condition may be also induced by triggers leading to significant level of physiological stress – extensive burns, major trauma, childbirth and acute cardiovascular events. Furthermore, excessive iodine exposure may provoke thyrotoxicosis, especially in individuals with underlying thyroid disease [5]. Nevertheless, in a proportion of patient, no indetifiable precipitating factor can be determined despite meticulous clinical evaluation. [3]

Although the precipitating factors of thyroid storm are well recognized and preoperative optimization is routinely recommended to reduce perioperative risk, current evidence suggests that no preventive strategy can completely eliminate the occurrence of thyroid storm. Antithyroid drugs, β-blockers, iodine, corticosteroids, lithium, and combination regimens have all been used successfully, yet thyroid storm has been reported with each approach. Interestingly, retrospective cohort studies reported no cases of perioperative thyroid storm among patients prepared solely with β-blockers despite persistent biochemical hyperthyroidism, suggesting that adequate control of adrenergic manifestations may be sufficient in carefully selected patients. However, isolated cases of thyroid storm have also been described following β-blocker monotherapy, and the available evidence remains limited by the retrospective design of most studies and the rarity of the condition. Consequently, perioperative management should be individualized according to the patient's clinical status, disease severity, and tolerance to available therapies, as no preoperative regimen has been shown to completely abolish the risk of thyroid storm. [18]

A particularly unusual precipitating factor for thyroid storm is direct manipulation of the thyroid gland, as illustrated by a case report of thyroid storm following fine-needle aspiration (FNA) of a thyroid nodule. The patient had previously untreated thyrotoxicosis and a large multinodular goiter and developed clinical deterioration shortly after undergoing FNA. She subsequently presented with tachycardia, hyperthermia, gastrointestinal symptoms and other manifestations consistent with thyroid storm, requiring intensive medical treatment and ultimately emergency thyroidectomy. The authors suggest that mechanical manipulation of hyperfunctioning thyroid tissue may contribute to the acute release of thyroid hormones and trigger systemic decompensation in susceptible patients. This case highlights that, although FNA is generally considered a minimally invasive and safe procedure, thyroid manipulation may rarely precipitate thyroid storm in patients with uncontrolled thyrotoxicosis. Therefore, thyroid function and the clinical indication for invasive thyroid procedures should be carefully assessed before FNA, particularly in patients with suspected hyperfunctioning nodules. Achieving adequate control of thyrotoxicosis before elective thyroid manipulation may reduce the risk of this potentially life-threatening complication. [25]

Thyroid storm is frequently connected to Graves' disease and it may also be the first symptom of this condition. Other conditions associated with this disease include toxic multinodular goiter, toxic adenoma, or amiodarone-induced thyrotoxicosis. [3]

The clinical manifestations of thyroid storm are extremely variable, with the severity of symptoms differing significantly among affected individuals. The primary features include hyperthermia above 38.5°C, tachycardia disproportionate to the elevated body temperature, cardiac arrhythmias such as atrial fibrillation, congestive heart failure and pulmonary edema. Neurological disorders range from agitation and altered mental status to lack or impaired consciousness, including coma. Gastrointestinal manifestations may present with diarrhea, nausea and vomiting, or jaundice. Also reversible dilated cardiomyopathy has been observed, however occuring more frequently in younger individuals than in elderly patients. Additionally, stress-induced cardiomyopathy (Takotsubo) has been reported as a potential complication connected to thyroid storm. [3]

Methods of treatment

Antithyroid drugs

First-line pharmalogical treatment of thyroid storm includes antithyroid drugs such as propylthiouracil (PTU), carbimazole, and methimazole (MMI), which inhibit thyroid hormone synthesis by blocking thyroid peroxidase. Additionally, PTU reduces the peripheral conversion of thyroxine (T4) to triiodothyronine (T3) through inhibition of type 1 deiodinase. This dual mechanism of action makes PTU the preferred initial therapy, as it has been shown to produce a greater reduction in serum T4 and T3 concentrations within the first 24 hours compared with MMI. However, methimazole may be preferred in some clinical settings due to evidence suggesting more rapid normalization of thyroid hormone levels and its more favorable safety profile, particularly regarding the risk of hepatotoxicity. [1] The recommended loading dose od PTU is 500-1000 mg, followed by 250 mg every 4 hours (maximum 1600 mg per day), administered orally or intravenously. MMI is typically prescribed at 60-80 mg/day with a maximum dose of 100 mg.

Recent evidence from a large multicenter comparative effectiveness study including 1,383 patients with thyroid storm demonstrated no significant differences between propylthiouracil and methimazole with respect to in-hospital mortality, adverse events, duration of organ support, or hospitalization costs. In-hospital mortality was 8.5% in the PTU group and 6.3% in the MMI group, with an adjusted risk difference of 0.6% (95% CI, -1.8% to 3.0%; P = 0.64), suggesting that both medications have comparable clinical effectiveness in the maganement of thyroid storm. [7]

Antithyroid drugs are the most commonly administered orally. However, alternative routs of administration, including intravenous, rectal, and enteral delivery (nosogastric tube) may be used in selected clinical situations, particularly in individuals with thyroid storm or when oral administration is not possible. Available evidence suggests that these alternative methods can achieve therapeutic efficacy, although current literature is limited to case reports and small case series. Consequently, the evidence base remains insufficient to draw definitive conclusions regarding the comparative efficacy, safety, and optimal administration protocols of non-oral antithyroid drug delivery. Well-designed clinical studies are needed to establish standardized recommendations for use of alternative routes of administration in different clinical settings. [8]

Alternative therapeutic strategies are primarily required in patients with contraindications or intolerance to antithyroid drugs, such as agranulocytosis or hepatotoxicity, as well as in those who fail to achieve adequate biochemical control despite maximal medical therapy or require urgent thyroidectomy. In these situations, adjunctive therapies including glucocorticoids, iodine, lithium, cholestyramine, β-blockers, and therapeutic plasma exchange have been successfully used to achieve rapid biochemical stabilization and facilitate definitive surgical treatment. Across the available studies, these approaches were associated with substantial reductions in serum thyroid hormone concentrations and enabled safe thyroidectomy without reported cases of perioperative thyroid storm. Nevertheless, the current evidence is based predominantly on observational studies and case series, highlighting the need for prospective studies to establish standardized treatment algorithms for patients in whom conventional antithyroid therapy is contraindicated or ineffective. [13]

Iodine

Potassium iodine (KI) remains an important adjunctive therapy in the management of thyroid storm due to its rapid inhibition of thyroid hormone release (the Wolff-Chaikoff effect). Recent observational evidence suggests that early administration of KI, in combination with antithyroid drugs, may improve clinical outcomes in patients with Graves' disease presenting with thyroid storm. Although KI use was not associated with a significant reduction in overall in-hospitality mortality, a noticeable mortality benefit was observed in the subgroup of patients with Graves' disease. In addition, KI administration was associated with shorter hospital stays and lower hospitalization costs. Nevertheless, these findings are derived from observational data, and prospective studies are still needed to confirm the clinical benefits of KI and to better define its role in the management of thyroid storm across different etiologies of thyrotoxicosis. [9]

In the preoperative setting, iodine administration has been associated with reduced thyroid gland vascularity and decreased intraoperative blood loss, thereby facilitating safer thyroidectomy. In addition, the combination of potassium iodide with methimazole may enable more rapid achievement of euthyroidism while allowing lower thionamide doses, potentially reducing treatment-related adverse effects. However, iodine should be used cautiously in patients with autonomous thyroid nodules or iodine deficiency, as it may paradoxically increase thyroid hormone synthesis. Furthermore, current international guidelines differ regarding the optimal timing of iodine administration in relation to thionamide therapy, reflecting the limited evidence available and the need for further studies to establish standardized treatment protocols. Although

potassium iodide is generally well tolerated when administered for short periods, adverse effects may occur and include gastrointestinal disturbances, dermatological reactions, metallic taste, salivary gland inflammation, esophageal ulceration, and, rarely, hypersensitivity reactions such as anaphylaxis. Patients should therefore be monitored for potential adverse events, particularly when higher doses or prolonged treatment are required. [1]

Lithium

Lithium may be considered a second-line therapeutic option in the management of thyroid storm, particularly in patients with contraindications or intolerance to thionamides. By inhibiting thyroid hormone release through suppression of TSH-mediated cyclic adenosine monophosphate (cAMP) signaling, lithium can rapidly reduce circulating thyroid hormone concentrations. Clinical studies have demonstrated that therapeutic doses of lithium decrease serum thyroxine levels within several days, while the combination of lithium and propylthiouracil has been associated with faster achievement of euthyroidism in patients with amiodarone-induced thyrotoxicosis compared with propylthiouracil alone. Despite these potential benefits, the use of lithium is limited by its narrow therapeutic index and the risk of adverse effects, including gastrointestinal symptoms, tremor, neurological toxicity, and altered mental status. Consequently, careful monitoring of serum lithium concentrations and renal function is required throughout treatment, and its use should be reserved for selected patients in whom conventional antithyroid therapy is not feasible. [1,10]

Potassium perchlorate

Perchlorate may be used as an adjunctive treatment in selected patients with thyroid storm, particularly in cases of amiodarone-induced thyrotoxicosis, where excess iodine plays a key role in disease pathogenesis. By competitively inhibiting the sodium–iodide symporter, perchlorate reduces thyroidal iodine uptake and promotes the release of intrathyroidal iodide, thereby decreasing the intrathyroidal iodine pool. It is typically administered in combination with thionamides to enhance the control of thyrotoxicosis. However, its clinical use is limited by the potential for serious adverse effects, including dermatological reactions, drug fever, nephrotic syndrome, agranulocytosis, and, rarely, aplastic anemia. Consequently, perchlorate should be reserved for carefully selected patients and used under close clinical and hematological monitoring. [1]

Clucocorticoids

Glucocorticoids remain an important component of multimodal therapy for thyroid storm. In addition to reducing the peripheral conversion of thyroxine (T4) to triiodothyronine (T3), they suppress thyroid hormone production in patients with Graves' disease and may help prevent the exacerbation of the relative adrenal insufficiency frequently associated with severe thyrotoxicosis. Consequently, glucocorticoids are recommended as adjunctive therapy, particularly in critically ill patients, with hydrocortisone and dexamethasone being the most commonly used agents. Although short-term treatment is generally well tolerated, potential adverse effects include hyperglycemia, hypertension, and immunosuppression. Nevertheless, the available evidence supporting glucocorticoid use in thyroid storm is based predominantly on observational studies and expert consensus, with limited high-quality clinical data demonstrating a clear mortality benefit. Therefore, further prospective studies are required to better define their impact on clinical outcomes and to optimize treatment protocols. [1,11]

Cholestyramine

Cholestyramine may be considered an adjunctive therapy in thyroid storm, particularly in patients with severe thyrotoxicosis or when conventional antithyroid treatment is insufficient or contraindicated. By binding thyroid hormones within the intestinal lumen and interrupting their enterohepatic circulation, cholestyramine accelerates the reduction of circulating thyroid hormone concentrations. Available evidence suggests that its addition to standard multimodal therapy results in a more rapid biochemical improvement without significant safety concerns during short-term use. However, the current evidence is derived primarily from small clinical studies, case reports, and observational data, and cholestyramine should therefore be regarded as an adjunct rather than a substitute for established therapies. Further prospective studies are required to better define its impact on clinical outcomes in patients with thyroid storm. [12]

In patients with refractory thyroid storm complicated by multiorgan dysfunction or contraindications to conventional antithyroid therapy, the combination of therapeutic plasma exchange (TPE) and cholestyramine may provide an effective rescue strategy. While TPE rapidly reduces circulating thyroid hormones, thyroid hormone-binding proteins, and inflammatory mediators, cholestyramine complements this effect by interrupting the enterohepatic circulation of thyroid hormones, thereby limiting their reabsorption. The combined use of these therapies has been associated with rapid biochemical and clinical improvement in patients who fail to respond adequately to standard medical treatment, potentially providing a bridge to definitive therapy such as thyroidectomy. [12]

Beta Adrenergic Blockers

β-blockers remain the cornerstone of symptomatic management in thyrotoxicosis and thyroid storm by controlling adrenergic manifestations. Among them, propranolol is generally preferred because, in addition to β-adrenergic blockade, it inhibits the peripheral conversion of thyroxine (T4) to triiodothyronine (T3), thereby providing an additional antithyroid effect. However, β-blocker selection should be individualized according to the patient's hemodynamic status and underlying cardiovascular comorbidities, particularly in the presence of heart failure or circulatory instability. Further studies are warranted to optimize β-blocker selection across different clinical scenarios of severe thyrotoxicosis. [14]

Compared with metoprolol, propranolol offers the additional advantage of inhibiting the peripheral conversion of thyroxine (T4) to triiodothyronine (T3) through inhibition of type 1 deiodinase, thereby reducing circulating T3 concentrations in addition to controlling adrenergic symptoms. This dual mechanism makes propranolol the preferred β-blocker in patients with severe thyrotoxicosis and thyroid storm when no contraindications exist. Nevertheless, caution is warranted in patients with impaired cardiac function or hemodynamic instability, in whom alternative β-blockers may be more appropriate. [14].

Although propranolol is widely recommended in thyroid storm due to its ability to control adrenergic symptoms and inhibit peripheral conversion of T4 to T3, its use requires caution in patients with underlying thyrotoxic cardiomyopathy. In individuals with impaired cardiac reserve or low-output heart failure, β-adrenergic blockade may precipitate severe hemodynamic deterioration, including hypotension, cardiogenic shock, and cardiac arrest. Therefore, assessment of cardiac function and hemodynamic stability is essential before initiating β-blocker therapy, and treatment should be individualized according to the cardiovascular status of the patient. [15]

Despite their established role in controlling adrenergic manifestations of thyroid storm, β-blockers may occasionally precipitate severe hemodynamic deterioration, particularly in patients with unrecognized thyrotoxic cardiomyopathy or limited cardiac reserve. Several case reports and case series have described circulatory collapse and cardiac arrest shortly after administration of long-acting β-blockers, most commonly propranolol, highlighting the need for careful patient selection and cardiovascular assessment before initiation. In high-risk patients, short-acting agents such as esmolol may offer a safer alternative due to their rapid onset and ability to be promptly discontinued in the event of adverse hemodynamic effects. However, current evidence is limited to case reports and observational studies, and further research is required to define the optimal β-blocker strategy in patients with severe thyrotoxicosis and thyroid storm. [16]

Thyroid storm is a rare but life-threatening endocrine emergency representing the most severe form of thyrotoxicosis and is associated with significant cardiovascular morbidity and mortality. Excess thyroid hormones increase heart rate, myocardial contractility and cardiac output while reducing systemic vascular resistance, placing substantial stress on the cardiovascular system. Consequently, patients may develop severe tachycardia, atrial fibrillation and other tachyarrhythmias, acute heart failure, thyrotoxic cardiomyopathy, myocardial ischemia or infarction, myocarditis and, in the most severe cases, cardiogenic shock and cardiac arrest. Cardiovascular complications are therefore among the most important determinants of clinical outcome. Diagnosis is primarily clinical, as thyroid hormone concentrations alone cannot reliably distinguish uncomplicated thyrotoxicosis from thyroid storm. Treatment requires rapid, simultaneous management of thyrotoxicosis, cardiovascular complications and the precipitating cause, with supportive intensive care, antithyroid drugs, iodine and glucocorticoids forming the basis of therapy. β-blockers remain the cornerstone of symptomatic management because they control the excessive adrenergic manifestations of thyrotoxicosis, particularly tachycardia. Among them, propranolol is generally preferred because, in addition to β-adrenergic blockade, it inhibits type 1 deiodinase and therefore reduces the peripheral conversion of thyroxine (T4) to triiodothyronine (T3), providing an additional antithyroid effect. Compared with metoprolol, this dual mechanism makes propranolol particularly attractive in severe thyrotoxicosis and thyroid storm when no contraindications are present.

However, β-blocker therapy must be individualized according to the patient's cardiovascular and hemodynamic status. This is particularly important in patients with thyrotoxic cardiomyopathy, impaired cardiac reserve, acute heart failure or circulatory instability. Although β-adrenergic blockade can effectively control tachycardia and adrenergic symptoms, excessive reduction in heart rate and myocardial contractility may significantly decrease cardiac output and precipitate hypotension, cardiogenic shock or even cardiac arrest. Several case reports and case series have described severe hemodynamic deterioration shortly after administration of long-acting β-blockers, most commonly propranolol, particularly in patients with previously unrecognized thyrotoxic cardiomyopathy. Therefore, careful assessment of cardiac function and hemodynamic

stability is essential before initiating treatment. In high-risk or hemodynamically unstable patients, short-acting agents such as esmolol may be a safer alternative, as their effects can be rapidly titrated and discontinued if cardiovascular deterioration occurs. Overall, the management of thyroid storm should balance the benefits of controlling the adrenergic effects of thyroid hormones against the risk of worsening cardiac function. Despite the established role of β-blockers, evidence regarding the optimal agent and dosing strategy in patients with severe thyrotoxicosis and cardiovascular complications remains limited, highlighting the need for further research and emphasizing that β-blocker selection should be guided by the individual patient's cardiac function and hemodynamic status. [20]

A case report by Herzallah et al. highlights the potential risk of β-blocker therapy even when using a short-acting agent such as esmolol in patients with thyroid storm and atrial fibrillation. A 37-year-old woman presented with thyroid storm complicated by atrial fibrillation with a rapid ventricular response and borderline low blood pressure, but without overt clinical signs of heart failure. Because of the short half-life and rapid titratability of esmolol, an infusion was initiated for rate control. However, approximately 15 minutes after treatment initiation, the patient developed respiratory distress, followed by profound bradycardia and cardiac arrest. After resuscitation, echocardiography revealed reduced left ventricular systolic function with an ejection fraction of approximately 40%, dilated cardiomyopathy and moderate-to-severe mitral regurgitation, suggesting previously unrecognized cardiac dysfunction. Despite subsequent treatment with propranolol, carbimazole and hydrocortisone, together with intensive supportive therapy, the patient developed multiorgan failure and ultimately died. This case emphasizes that the short-acting nature of esmolol does not eliminate the risk of severe cardiovascular deterioration in thyroid storm. Importantly, apparently compensated patients may have underlying low-output heart failure that is not evident on initial clinical examination. Therefore, careful assessment of cardiac function and hemodynamic status, including echocardiography when available, should precede β-blocker therapy. In patients with evidence of heart failure or impaired cardiac reserve, management should prioritize treatment of thyrotoxicosis and adequate hemodynamic support rather than immediate β-adrenergic blockade. [26]

Plasmapheresis

Therapeutic plasma exchange (plasmapheresis) was first reported as a one of treatment option for thyroid storm in 1970. It reduces the amount of circulating thyroid hormones, cytokines and thyroid-related antibodies contibuting to clinical stabilization. According to the current recommendations of the American Society of Apheresis, plasmapheresis should be performed every 1-3 days until the patient's condition has stabilized. In patients with severe thyroid storm who fail to respond to standard therapy within 48 hours, or in whom conventional treatment is contraindicated, plasmapheresis is recommended. Although the procedure is considered safe and weel tolerated, there may occur some potential complications including anaphylactic reactions, coagulation disorders vascular injury and seizures. [6] Therapeutic plasma exchange serves as an effective bridging therapy to thyroidectomy. Clinical studies have demonstrated that plasmapheresis results in a progressive reduction in circulating thyroid hormone levels, with an average decrease od approximately 21% after each session and a cumulative decline of up to 55% following four treatment sessions. [7]

Therapeutic plasma exchange (TPE) may represent a valuable rescue therapy in patients with severe or refractory thyroid storm, particularly when conventional medical treatment is ineffective, contraindicated, or complicated by multiorgan dysfunction. By removing protein-bound thyroid hormones and other circulating factors, TPE enables a rapid reduction in thyroid hormone concentrations and may provide clinical stabilization before definitive treatment, such as thyroidectomy. Available evidence suggests that TPE can be effective in achieving biochemical improvement and facilitating surgical management in critically ill patients. However, its use remains based mainly on case reports and small observational studies, and therefore it should be reserved for selected patients in experienced tertiary centers with multidisciplinary support. [17]

Thyroidectomy

A large retrospective national study by Seo et al. evaluated the role of thyroidectomy during hospitalization for thyroid storm using data from more than 16,000 admissions in the United States between 2016 and 2020. Thyroidectomy was performed in only 3.9% of patients, reflecting its role as a rescue strategy rather than routine treatment. Patients who underwent surgery more frequently presented with severe systemic complications, including supraventricular tachycardia, acute decompensated heart failure, acute liver failure and acute renal failure. Notably, acute decompensated heart failure was independently associated with a higher likelihood of undergoing thyroidectomy (adjusted OR 1.66), suggesting that severe cardiovascular deterioration may contribute to the decision to pursue definitive surgical treatment. However, thyroidectomy

was associated with a 30% perioperative complication rate, and the study did not demonstrate a significant reduction in mortality compared with nonoperative management. These findings support the current approach of reserving thyroidectomy for patients who fail or cannot tolerate maximal medical therapy or require rapid definitive control of thyrotoxicosis. Importantly, the study highlights the difficulty of performing surgery during an acute thyroid storm, particularly in patients with cardiovascular and multiorgan dysfunction, and suggests that thyroidectomy should be considered a carefully selected rescue intervention rather than a standard first-line treatment. [27]

Cardiovascular Consequences

Thyroid storm can lead to severe and potentially life-threatening cardiovascular complications. The cardiovascular system is one of the most commonly affected organ systems, and patients may develop marked sinus tachycardia, atrial fibrillation, atrial flutter, supraventricular and ventricular arrhythmias, myocardial injury and acute myocardial infarction, including cases related to coronary artery spasm or an imbalance between myocardial oxygen supply and demand. Thyroid storm may also result in acute heart failure, high-output heart failure, thyrotoxic dilated cardiomyopathy, Takotsubo cardiomyopathy and, less frequently, pericardial effusion. In severe cases, progressive myocardial dysfunction can lead to pulmonary edema, cardiogenic shock, circulatory collapse and cardiac arrest. Importantly, these cardiovascular complications may occur simultaneously and contribute substantially to the high mortality associated with thyroid storm. Therefore, early recognition and appropriate management of cardiovascular complications are essential to prevent hemodynamic deterioration and improve outcomes in patients with thyroid storm. [19]

The treatment of thyroid storm requires an urgent, multimodal approach aimed at controlling excessive thyroid hormone production and action, stabilizing the cardiovascular system, and treating the underlying precipitating factor. Patients should receive intensive supportive care, including continuous cardiac and hemodynamic monitoring, oxygen or ventilatory support when required, correction of fluid and electrolyte disturbances, and treatment of hyperthermia. Specific therapy includes antithyroid drugs, such as propylthiouracil (PTU) or methimazole, to inhibit new thyroid hormone synthesis. Iodine should be administered after antithyroid therapy to inhibit the release of preformed thyroid hormones, while glucocorticoids, such as hydrocortisone, reduce peripheral conversion of T4 to T3 and help prevent or treat relative adrenal insufficiency.

β-blockers play a central role in controlling the cardiovascular manifestations of thyroid storm, particularly tachycardia and excessive sympathetic activity. Propranolol is traditionally preferred because it also inhibits peripheral T4-to-T3 conversion; however, its use requires caution in patients with heart failure, thyrotoxic cardiomyopathy or hemodynamic instability. In such patients, short-acting agents such as esmolol may be preferable because their effects can be rapidly titrated and discontinued if cardiovascular deterioration occurs. Additional cardiovascular treatment should be tailored to the specific complication, including management of atrial fibrillation, acute heart failure and cardiogenic shock. If conventional therapy is insufficient, cholestyramine, therapeutic plasma exchange (plasmapheresis), dialysis or emergency thyroidectomy may be considered in refractory cases. Once the acute crisis has resolved, definitive treatment of the underlying hyperthyroidism, such as radioactive iodine therapy or thyroidectomy, should be considered to prevent recurrence. Overall, successful management of thyroid storm depends on early recognition, simultaneous control of thyrotoxicosis and cardiovascular complications, and individualized treatment according to the patient's hemodynamic status. [19]

Overall, the current consensus emphasizes that thyroid storm should be managed as a medical emergency requiring early recognition, prompt initiation of treatment and close monitoring in an intensive care setting. Management should be individualized according to the severity of thyrotoxicosis, the presence of multiorgan dysfunction and, particularly, the patient’s cardiovascular status. Since robust randomized evidence is limited, many therapeutic recommendations are based on expert consensus and clinical experience. The treatment should therefore focus not only on reducing thyroid hormone synthesis and action, but also on maintaining adequate tissue perfusion and preventing or treating cardiovascular and other organ complications. Early identification and treatment of precipitating factors, continuous cardiovascular monitoring and timely escalation to advanced supportive therapies in refractory cases are essential. Ultimately, successful management of thyroid storm depends on a structured, multidisciplinary and individualized approach, with particular attention to hemodynamic stability and the potential for rapid clinical deterioration. [21]

A case report by Bokhari et al. illustrates the severe cardiovascular consequences that may occur during thyroid storm and highlights the potentially reversible nature of thyrotoxic cardiomyopathy. The patient

developed supraventricular tachycardia followed by atrial flutter with 2:1 conduction and severe left ventricular systolic dysfunction, with a left ventricular ejection fraction (LVEF) of only 15%. The cardiovascular deterioration was attributed to the combination of prolonged untreated hyperthyroidism, thyroid storm, and persistent tachyarrhythmia. Management included antithyroid therapy with propylthiouracil, potassium iodide, hydrocortisone and cholestyramine, together with β-blockade using esmolol and subsequently propranolol. Because of persistent atrial flutter, electrical cardioversion was performed, resulting in restoration of sinus rhythm and subsequent improvement in left ventricular function, with LVEF increasing to approximately 25–30%. This case demonstrates that thyroid storm may cause severe tachyarrhythmias and acute myocardial dysfunction, potentially progressing to heart failure and hemodynamic instability. Importantly, the improvement in cardiac function following control of thyrotoxicosis and restoration of sinus rhythm suggests that thyrotoxic cardiomyopathy may be at least partially reversible when the underlying endocrine disorder and cardiovascular complications are treated promptly and appropriately. [23]

A case report by Zayour et al. further illustrates the potentially catastrophic cardiovascular consequences of thyroid storm. A previously healthy 24-year-old man presented with cardiac arrest and required prolonged cardiopulmonary resuscitation, including defibrillation for pulseless ventricular tachycardia, followed by vasopressor and mechanical ventilatory support. After return of spontaneous circulation, he was found to have severe sinus tachycardia, hyperthermia and marked left ventricular systolic dysfunction, with an ejection fraction of only 25%. Laboratory testing revealed severe thyrotoxicosis, and the clinical presentation was consistent with thyroid storm. Although the patient developed a significant rise in troponin levels, coronary angiography showed no obstructive coronary artery disease, suggesting that the cardiovascular deterioration was related to the thyroid storm rather than acute coronary occlusion. Treatment with methimazole, Lugol's iodine, propranolol and subsequently hydrocortisone, together with intensive supportive care, resulted in rapid clinical improvement. Left ventricular function improved to 50–55% by the third day and subsequently normalized, with an LVEF of approximately 60–65% during follow-up. This case highlights that thyroid storm may present initially as cardiac arrest and severe acute heart failure, even in a young patient without previously known thyroid or cardiovascular disease. It also demonstrates that thyrotoxic cardiomyopathy may be largely reversible following restoration of euthyroidism, emphasizing the importance of considering thyroid storm in the differential diagnosis of unexplained cardiac arrest or acute heart failure, particularly when other common causes have been excluded. [24]

Conclusions

Thyroid storm is a rare but potentially fatal endocrine emergency in which cardiovascular complications play a central role in determining clinical outcome. Tachyarrhythmias, atrial fibrillation, acute and high-output heart failure, thyrotoxic cardiomyopathy, myocardial injury, cardiogenic shock, and cardiac arrest may occur as a consequence of severe thyrotoxicosis and can contribute substantially to mortality. Therefore, early recognition of cardiovascular involvement and continuous assessment of hemodynamic status are essential components of management.

Treatment should be initiated promptly and should combine control of thyroid hormone synthesis and release with aggressive supportive and cardiovascular therapy. Antithyroid drugs, iodine, glucocorticoids and β-blockers remain the main components of pharmacological treatment, while the choice and intensity of therapy should be adapted to the patient's clinical condition. Although propranolol provides the additional benefit of reducing peripheral T4-to-T3 conversion, β-blockade may be hazardous in patients with thyrotoxic cardiomyopathy, low-output heart failure or circulatory instability; in selected high-risk patients, short-acting agents such as esmolol may therefore be preferable. Alternative therapies, including lithium, cholestyramine and therapeutic plasma exchange, may provide useful adjunctive or rescue treatment, particularly when conventional therapy is contraindicated or ineffective, while thyroidectomy may be required as definitive treatment in refractory cases.

Overall, there is currently insufficient evidence to determine which alternative or rescue treatment is superior in thyroid storm. Most available evidence is derived from observational studies, case reports and expert consensus, highlighting the need for prospective, high-quality studies. Future research should focus particularly on optimizing cardiovascular management, β-blocker selection, rescue therapies, and individualized treatment strategies in patients with severe or refractory thyroid storm. [22]

A case report by Mansoor et al. highlights the potential cardiovascular risks associated with β-blocker therapy in patients with thyroid storm and underlying thyrotoxic cardiomyopathy. A 47-year-old man with untreated Graves' disease and atrial fibrillation presented with thyroid storm and symptoms of heart failure.

Shortly after administration of propranolol, the patient developed pulseless electrical activity cardiac arrest, requiring cardiopulmonary resuscitation. Echocardiography subsequently revealed severe biventricular enlargement and markedly reduced left ventricular systolic function, with an ejection fraction of 15–20%, compared with 60–65% one year earlier. The patient was treated with supportive intensive care, diuretics and guideline-directed therapy for heart failure, while β-blockade was cautiously reintroduced using metoprolol after stabilization. An ischemic work-up revealed normal coronary arteries, supporting thyrotoxic cardiomyopathy as the underlying cause of cardiac dysfunction. Importantly, cardiac function subsequently recovered completely, with LVEF returning to 60–65% after 90 days. This case emphasizes that thyroid storm may present with overt or subclinical low-output heart failure and that conventional β-blockade, particularly with longer-acting agents such as propranolol, may precipitate severe hemodynamic deterioration in patients with impaired cardiac reserve. Therefore, early assessment of cardiac systolic function with transthoracic echocardiography should be considered in patients with thyroid storm before initiating β-blocker therapy. In patients with significant cardiac dysfunction or hemodynamic instability, short-acting agents such as esmolol may offer a safer alternative because their effects can be rapidly titrated and discontinued if cardiac function deteriorates.

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