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THYROID STORM IN A PATIENT WITH GRAVES' DISEASE AND TUBO-OVARIAN ABSCESS: A CLINICAL CASE REPORT

Ni Nyoman Gita Kharisma Dewi*, Dewi Catur Wulandari
Department of Internal Medicine, Wangaya Regional General Hospital, Jl. Kartini No.133, Dauh Puri Kaja, Denpasar, Bali 80231 Indonesia
*gitakharismadewi67@gmail.com

ABSTRACT
Thyroid storm is a rare, life-threatening complication of thyrotoxicosis, involving multi-organ dysfunction. This study aims to identify risk factors for thyroid storm and evaluate effective management strategies to reduce mortality. This study is a descriptive case report. Data were collected through patient interviews, physical examinations, and supporting tests at Wangaya Hospital. The data were analyzed qualitatively and presented in a narrative format based on case report guidelines. This paper reports the case of a 24-year-old female with a history of hyperthyroidism on methimazole and an intrauterine device (IUD) presented with abdominal pain that started in the epigastric region and migrated to the lower quadrants. She developed ocular prominence, vomiting, palpitations, generalized weakness, and non-bloody diarrhea. She also experienced persistent lower abdominal pain (pain score: 6/10), dysuria, and fever. A Burch-Wartofsky Point Scale (BWPS) score > 45 confirmed thyroid storm. She was admitted to the ICU and treated with propylthiouracil (PTU), hydrocortisone, digoxin, propranolol, and antibiotics for suspected infection. Her condition improved, and she was discharged after seven days. Thyroid storm is commonly triggered by Graves' disease. The diagnosis is clinical, based on BWPS or the Japanese Thyroid Association criteria, especially in patients with a history of hyperthyroidism and a precipitating factor. PTU is preferred due to its rapid onset and ability to inhibit the conversion of T4-to-T3. Successful management of thyroid storm requires prompt, comprehensive therapy to suppress thyroid hormone levels and treat triggering factors such as infection.

Keywords: hyperthyroidism; infection; thyroid storm

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INTRODUCTION

Thyroid crisis, or thyroid storm, is a severe manifestation of thyrotoxicosis (De Almeida et al., 2022). It is essential to distinguish hyperthyroidism from thyrotoxicosis. Hyperthyroidism refers to a condition characterized by excessive synthesis and secretion of thyroid hormones by the thyroid gland. In contrast, thyrotoxicosis encompasses the clinical manifestations resulting from elevated levels of triiodothyronine (T3) and thyroxine (T4) in peripheral tissues (De Leo et al., 2016; Farooqi et al., 2023). The severity of thyrotoxicosis ranges from subclinical to overt forms, and in extreme cases, it can progress to multi-organ dysfunction, a life-threatening state commonly referred to as thyroid storm or thyroid crisis (Farooqi et al., 2023; Pearce, 2006).Thyroid storm is a life-threatening condition characterized by multi-organ dysfunction, affecting the cardiovascular, thermoregulatory, gastrointestinal-hepatic, and central nervous systems. Although its incidence is rare, occurring in only 1–2% of hospitalized patients, the mortality rate remains high, ranging from 10–30% (Chiha et al., 2015; Klubo-Gwiezdzinska & Wartofsky, 2012; Lim et al., 2021). Diagnosing thyroid storm presents a significant challenge due to the nonspecific nature of laboratory findings. However, a combination of thyroid function tests, clinical signs, and evidence of end-organ damage can aid in establishing the diagnosis.

The clinical manifestations of thyroid storm include tachycardia, atrial fibrillation, congestive heart failure, agitation, delirium, psychosis, stupor, nausea, vomiting, diarrhea, hepatic failure, and hyperpyrexia (De Almeida et al., 2022; Swee et al., 2015). The Burch-Wartofsky Point Scale (BWPS) and the Japanese Thyroid Association (JTA) scoring system can be utilized to support the diagnosis of thyroid storm (De Almeida et al., 2022). Thyroid function tests typically reveal markedly suppressed thyroid-stimulating hormone (TSH) levels (<0.01 mU/L) with elevated free thyroxine (fT4) and/or free triiodothyronine (fT3). The presence of thyroid receptor antibodies (TRAb) may indicate Graves' disease as the underlying cause (De Almeida et al., 2022). The highly aggressive nature of thyroid storm necessitates a multimodal treatment approach immediately after diagnosis. Management should focus on inhibiting thyroid hormone synthesis, release, and action through pharmacological agents while providing hemodynamic support in an intensive care setting (De Almeida et al., 2022; Swee et al., 2015). Based on this premise, this study presents a case of thyroid storm in a reproductive-age woman with a history of IUD use, which triggered a tubo-ovarian abscess, and a history of poor adherence to antithyroid therapy. The study aims to explore the risk factors contributing to thyroid storm and to evaluate the appropriate management strategies to reduce mortality in this critical condition.

METHOD

This article presents a case report focusing on the diagnosis, clinical managements, and follow-up care of a patient. Data were obtained through patient anamnesis, physical examination, and supporting investigations conducted at Wangaya Hospital, Bali. The information was analyzed qualitatively and is presented in a narrative format. The case involves a female patient diagnosed with thyroid storm. This report outlines the identified risk factors, analyzes the case in detail, and aims to offer valuable insights for clinical practice, particularly in the management of thyroid storm. The findings are compared with relevant literature to evaluate the accuracy of the diagnosis and the appropriateness of the treatment provided.

RESULT

A 24-year-old female presented to the emergency department of Wangaya General Hospital with abdominal pain. The pain initially developed two days prior to admission, originating in the epigastric region before migrating to the left lower quadrant and subsequently to the right lower quadrant, with an intensity of 6/10. She also reported fever since the previous day, accompanied by chills. Although she experienced a decrease in appetite, she denied any history of nausea or vomiting. On the morning of admission, she developed watery diarrhea without mucus or blood. Additionally, she reported spotting that had started four days prior. The patient has a history of hyperthyroidism and has been receiving regular follow-up care at the endocrinology clinic of Wangaya General Hospital. She was diagnosed with Graves' disease–induced hyperthyroidism based on clinical symptoms and supporting investigations. Clinically, she reported dyspnea, easy fatigability, excessive sweating even in cold environments, hand tremors, palpitations, and an increased appetite. Based on the Wayne scoring system, her condition was classified as toxic hyperthyroidism (total score >19). Laboratory findings revealed a suppressed TSH level (<0.005 mU/L) and elevated free T4 (>6 ng/dL). A neck ultrasound demonstrated thyroiditis with multiple bilateral cervical lymphadenopathies. Fine-needle aspiration biopsy (FNAB) showed follicular hyperplasia with focal toxic changes. These findings confirmed the diagnosis of Graves' disease–induced hyperthyroidism, for which she was prescribed methimazole (thyrozol) 10 mg three times daily. Additionally, she received regular follow-ups at the cardiology clinic for a history of sinus tachycardia and thyroid-related heart disease. As part of her management, she was prescribed propranolol 10 mg three times daily. However, she reported inconsistent adherence to her hyperthyroidism medication, frequently forgetting to take the prescribed treatment.

The patient was previously hospitalized at Wangaya General Hospital in November with a diagnosis of a urinary tract infection (UTI). During that admission, her methimazole (Thyrozol) dosage was increased to 20 mg three times daily. She has a history of intrauterine device (IUD), which remains in place. Laboratory investigations revealed leukocytosis with neutrophil predominance. Urinalysis showed leukocyte esterase at 250 with a sediment leukocyte count of 4–5 per high-power field. Chest X-ray findings were within normal limits, and abdominal radiography did not reveal any abnormalities. Abdominal ultrasound showed free fluid in the left abdominal cavity and pelvic cavity, raising suspicion of colitis. The patient was referred to a cardiology specialist for further evaluation and management of palpitations associated with thyroid-related heart disease. Additionally, a consultation with an obstetrics and gynecology specialist was conducted due to the patient's history of intrauterine device (IUD) use, to assess its positioning. A transvaginal ultrasound (TVS) was performed, revealing a complex mass measuring 9.55 × 7.25 × 5.74 cm, characterized by hyper- and hypoechoic features, air-fluid levels, and incomplete septations, suggestive of a tubo-ovarian abscess.

During hospitalization, the patient's family reported that her eyes suddenly became more prominent (exophthalmos), more severe than usual. The patient also experienced two episodes of vomiting, palpitations, generalized weakness, and five episodes of diarrhea with solid stool content, without blood or mucus. She continued to report moderate left lower and mid-abdominal pain (pain score: 6/10), dysuria, and fever. Based on the patient's history and physical examination, the Burch-Wartofsky Point Scale (BWPS) score was greater than 45, indicating a high probability of thyroid storm. The specific BWPS criteria met in this case are detailed in Table 1. A precipitating factor for the onset of thyroid storm was also identified in this patient. The detailed precipitating factors contributing to thyroid storm in this case are listed in Table 2.

Table 1.
Bursch Wartofsky' criteria for thyroid storm

Thermoregulatory Dysfunction		Cardiovascular Dysfunction	
Temperature		Tachycardia	
37.2-37.7 °C	5	99-109	5
37.8-38.3 °C	10	110-119	10
38.4-38.8 °C	15	120-129	15
38.9-39.4 °C	20	130-139	20
39.5-39.9 °C	25	≥ 140	25
≥ 40 °C	30		
Central nervous System Effects		Congestive Heart failure	
• Absent	0	• Absent	0
• Mild - Agitation	10	• Mild -Pedal edema	5
• Moderate - Delerium -Psuchosis -Extreme lethargy	20	• Moderate -Bibasilar rales	10
• Severe -Seizure -Coma	30	• Severe -Pulmonary edema	15
Gastrointestinal-Hepatic Dysfunction		Atrial fibrilation	
• Absent	0	• Absent	0
• Moderate -Diarrhea -Nausea/vomiting -Abdominal pain	10	• Present	10
• Severe -Unexplained jaundice	20	Precipitant History	
		• Negative	0
		• Positive	10

A score of 45 or greater is highly suggestive of thyroid storm
A score of 25-44 is suggestive of impending thyroid storm
A score below 25 is unlikely to present thyroid storm
* The bolded criteria indicate the specific BWPS components that were met by the patient in this case.

Table 2.
Trigger factors of thyroid storm

Common	Rare
Infection	Vigorous palpitation of thyroid gland
Acute medical illness	Subacute thyroiditis
Acute psychosis	Thyroxine overdosage
Nonthyroidal surgery	Aspirin intoxication
Parturition	Hydatidiform mole
Trauma	OP poisoning
Discontinuation of antithyroid drugs	Neurotoxins
After radioactive iodine therapy	Cytotoxic chemotherapy
Post-thyroidectomy	
After high dose of iodine administration	
Iodinated radiographic cintrast agents	

*The bolded criteria indicate the triggering factors that were present in this patient.

The diagnosis of thyroid storm was supported by clinical findings according to Burch-Wartofsky criteria, a known history of hyperthyroidism, and the presence of triggering factors, including infection (tubo-ovarian abscess) and poor adherence to antithyroid therapy. This condition necessitated admission to the ICU for further management. She was admitted to the ICU with the following diagnoses: 1. Grave disease -induced Thyroid storm; 2.Thyroid heart disease; 3. Valvular heart disease with tachycardia; 4.Tubo-ovarian abscess; 5. Urinary tract infection (UTI); 6. Acute gastroenteritis (GE). During her ICU admission, the patient received multidisciplinary care involving the endocrinology team from the internal medicine department, as well as the cardiology and obstetrics-gynecology teams. During ICU management, the patient received: 1) fluid resucitation, 2) Methimazole (Thyrozol) was discontinued and replaced with propylthiouracil (PTU) 200 mg five times daily, 3) Hydrocortisone 100 mg twice daily, to inhibit peripheral conversion of T4 to T3, 4) Lugol's iodine was planned as part of thyroid storm management, but it was unavailable at Wangaya General Hospital, 5) Ranitidine 40 mg twice daily, was administered for gastric protection, and 6) Diagit 2 tablets three times daily as needed for diarrhea, and 7) paracetamol 1 g three times daily for treating fever.

The cardiology team initiated therapy with digoxin 0.25 mg once daily for rate control in thyroid heart disease and increased propranolol dosage to 20 mg three times daily, up from 10 mg, due to persistent tachycardia (heart rate 130–140 bpm), with an option to crush the tablet for easier administration. For infection management, antibiotics therapy was adjusted based on obstetric-gynecologic recommendations, consisting of Ampicillin 1 g every 6 hours, Gentamicin 80 mg every 12 hours, and Metronidazole 500 mg every 8 hours for anaerobic coverage. After three days of PTU 200 mg five times daily, the dose was reduced to 200 mg four times daily and subsequently to 200 mg three times daily the following day due to clinical improvement. Hydrocortisone was discontinued after five days of administration. Cardiac management adjustments were made based on ECG findings, which showed second-degree AV block type 2: 1. digoxin 0.25 mg once daily was withheld, 2. propranolol 20 mg three times daily was also withheld if HR <40 bpm. After six days later, echocardiography showed normal cardiac dimensions, no left ventricular hypertrophy (LVH), normal ejection fraction (EF), normal tricuspid annular plane systolic excursion (TAPSE), mild mitral regurgitation (MR), and trace tricuspid regurgitation (TR). Propranolol dose was adjusted, reduced to 10 mg three times daily when HR <70 bpm. Increased to 20 mg three times daily if HR >70 bpm, withheld if HR <60 bpm.

After seven days, the patient showed significant improvement and was discharged. She was advised to continue outpatient follow-up and prescribed: Cefixime 200 mg every 12 hours, Metronidazole 500 mg every 8 hours, Paracetamol 500 mg every 8 hours, Propranolol 10 mg every 8 hours (to be withheld if HR <60 bpm), PTU 200 mg every 8 hours. The patient was referred to Prof. Ngoerah General Hospital via the obstetrics and gynecology clinic for planned laparotomy to manage the tubo-ovarian abscess.

DISCUSSION

Thyroid storm, also known as thyrotoxic crisis, is a life-threatening syndrome resulting from severe hyperthyroidism with multisystem involvement. Although rare, thyroid storm carries a high risk of mortality if not promptly recognized and treated. In the United States, the incidence of thyroid storm is estimated at 0.57–0.76 cases per 100,000 people per year (Elmenyar et al., 2023; Galindo et al., 2019). The mortality rate ranges from 8–25%, emphasizing the critical need for rapid and effective management to reduce fatal outcomes. Thyroid storm is most commonly caused by Graves' disease, but it can also occur in conditions such as toxic multinodular goiter and solitary toxic adenomas. It may be triggered by factors such as thyroid or non-thyroid surgery, trauma, infection, acute iodine load, parturition, amiodarone use, and non-compliance with anti-thyroid medication (Pandey et al., 2020; Pokhrel et al., 2025).In this case, the patient had a history of hyperthyroidism managed with methimazole. However, she reported inconsistent adherence to methimazole therapy due to episodes of forgetting to take the medication. Additionally, she had a history of intrauterine device (IUD) use, which was previously associated with a urinary tract infection (UTI). In this instance, she was diagnosed with a tubo-ovarian abscess based on transvaginal ultrasound findings. From a pathophysiological perspective, the thyroid storm in this patient was triggered by two primary factors: discontinuation of thyroid medication and infection.

The diagnosis of thyroid storm is based on clinical findings, supported by a history of hyperthyroidism and the presence of precipitating factors. Key symptoms indicative of thyroid storm include fever (temperature >38° C), tachycardia (≥130 beats per minute), central nervous system manifestations, congestive heart failure, and gastrointestinal or hepatic involvement (Pandey et al., 2019). Two scoring systems are commonly used to assess thyrotoxicosis severity: the BWPS and JTA scale. According to these systems, systemic decompensation necessitates aggressive treatment if the BWPS score exceeds 45 or if the condition is classified as thyroid storm 1 (TS1) or thyroid storm 2 (TS2) under the JTA criteria ((Akamizu et al., 2012; Burch & Wartofsky, 1993; Pandey et al., 2020).In this case, the diagnosis of thyroid storm was established based on clinical findings consistent with the Burch-Wartofsky Score. The patient had a total BWPS score > 45, meeting the criteria for thyroid storm classification. This diagnosis was further supported by a history of hyperthyroidism and the presence of precipitating factors. In hyperthyroid states, laboratory findings typically include severely suppressed or undetectable thyroid-stimulating hormone (TSH) levels (<0.01 mU/L), along with elevated free thyroxine (fT4) and/or triiodothyronine (fT3) concentrations. If Graves' disease is the underlying etiology, thyroid receptor antibodies (TRAb) may also be positive. Patients diagnosed with thyroid storm require intensive care unit (ICU) admission for aggressive treatment and continuous monitoring to prevent

life-threatening complications.

The treatment of thyroid storm should be initiated immediately upon diagnosis. The goals of therapy in thyroid storm are to reduce thyroid hormone synthesis and release, decrease peripheral action of thyroid hormones, and address the underlying cause of the thyroid storm (Pandey et al., 2020) The therapeutic strategies include:

Inhibition of Thyroid Hormone Synthesis

The first-line therapy for thyroid storm includes inhibiting thyroid hormone production. Medications that can be used for this purpose are thionamides, such as propylthiouracil (PTU) and imidazoles (methimazole and carbimazole). These therapies inhibit the enzyme thyroid peroxidase, preventing the formation of T3 and T4 from thyroglobulin. PTU is preferred in the management of thyroid storm due to its advantages over carbimazole and methimazole, such as a faster onset of action and its ability to inhibit the peripheral conversion of T4 to T3, mediated by peripheral deiodinase. Additionally, PTU is considered safe for use in pregnant women (De Almeida et al., 2022; Pandey et al., 2020) The recommended PTU dose is 600-1500 mg per day, divided into doses every 4-6 hours, starting with an initial dose of 600 mg. The recommended dose of methimazole is 80-120 mg per day, divided into doses every 4-6 hours. According to the American Association of Clinical Endocrinologists/American Thyroid Association, the initial PTU dose should be 500-1000 mg, followed by 250 mg every 4 hours, while methimazole should be given at 60-80 mg per day in divided doses. The route of administration can be intravenous, enteral, or rectal (in suppository or retention enema form). However, PTU is relatively insoluble in physiological pH, making intravenous administration challenging.

Non-radioactive iodine can also reduce thyroid hormone synthesis by inhibiting the incorporation of organic iodide into thyroglobulin when plasma iodide levels reach a critical threshold. This phenomenon is known as the Wolff-Chaikoff effect. This effect lasts for approximately 50 hours, after which the thyroid gland adapts to prolonged excess iodide. Organic iodine can be administered orally in the form of saturated solution of potassium iodide (SSKI), with a dose of five drops (0.25 mL or 250 mg) every 6 hours, or as Lugol's solution (eight drops every 6 hours). The route of administration can be enteral, rectal, or intravenous. Iodine should be administered at least 30 minutes after thionamides to prevent iodide from serving as a substrate for new thyroid hormone production, which could exacerbate hyperthyroidism. Thionamides should continue to be given during iodine therapy to prevent iodide organification and increased thyroid hormone production. Lithium inhibits the synthesis of T4 and T3 by preventing the coupling of iodotyrosine residues. It can be used as an alternative to iodine. Lithium is administered at a dose of 300 mg every 6-8 hours (De Almeida et al., 2022; Pandey et al., 2020)

Inhibition of Thyroid Hormone Release

Iodine administration inhibits the release of thyroid hormones by preventing the release of iodothyronines (T3 and T4) from thyroglobulin. The combination of thionamide therapy and iodine effectively reduces serum T4 levels to the normal range within 4–5 days (De Almeida et al., 2022; Pandey et al., 2020)

Inhibition of Peripheral Thyroid Hormone Effects

Propranolol, a non-selective β-adrenergic antagonist, reduces the peripheral effects of thyroid hormones by blocking β-adrenergic receptors and inhibiting the peripheral conversion of T4 to T3. The recommended oral dose is 60–120 mg every 6 hours (Pandey et al., 2020; Stathatos & Wartofsky, 2002). Intravenous propranolol or short-acting β-blockers, such as esmolol, may also be used. The initial intravenous dose of propranolol is 0.5–1.0 mg administered via slow infusion, followed by 1–2 mg every 15 minutes while monitoring heart rate. Esmolol can be initiated with a bolus dose of 0.25–0.5 mg/kg, followed by continuous infusion at a rate of 0.05–0.1 mg/kg per minute (Pandey et al., 2020; Sosa, 2006)

Inhibition of Enterohepatic Circulation of Thyroid Hormones

This therapy is intended for patients with severe and refractory thyroid storm. Thyroid hormones are metabolized in the liver through conjugation into glucuronides and sulfates.

These conjugated products are excreted into the intestines via bile, where free thyroid hormones are subsequently released, reabsorbed, and recirculated. Cholestyramine binds to these conjugated products, promoting their excretion and thereby reducing circulating thyroid hormone levels. The recommended dose is 1–4 g twice daily (Kaykhaei et al., 2008; Mercado et al., 1996; Pandey et al., 2020; Tsai et al., 2005)

Other Therapies

Oral iodinated contrast agents act as inhibitors of deiodinase enzymes D1 and D2, reducing T3 levels. This therapy also suppresses the synthesis of new thyroid hormones and the release of preformed hormones from the thyroid gland. The recommended dosage is a 2 g loading dose followed by 1 g per day (Pandey et al., 2020; Stathatos & Wartofsky, 2002)). Lower doses are used as preoperative preparation for thyroid surgery and as an adjunct to thionamide therapy in Graves' disease (Langley & Burch, 2003; Pandey et al., 2020; Panzer et al., 2004)

Supportive and Resuscitative Measures

Resuscitation therapy should be initiated immediately in the intensive care unit (ICU). The urgent management of systemic decompensation includes correcting hyperthermia, dehydration, congestive heart failure (CHF), and dysrhythmia while preventing adrenal crisis (Pandey et al., 2020; Sosa, 2006). Hyperthermia should be managed through peripheral cooling and the use of antipyretics. Acetaminophen is preferred over salicylates, as salicylates increase free thyroid hormone levels by reducing T4-binding globulin affinity, thereby exacerbating thyroid storm (Pandey et al., 2020; WANG et al., 1999) Peripheral cooling can be achieved with ice packs, cooling blankets, or alcohol sponges. Fluid losses due to hyperpyrexia, diarrhea, and vomiting must be promptly corrected. Corticosteroids serve as an important adjunctive therapy. Thyrotoxicosis disrupts the hypothalamic-pituitary-adrenal axis, leading to decreased adrenal reserve. Increased cortisol production compensates for the elevated glucocorticoid metabolism seen in hyperthyroidism; however, the adrenal response to adrenocorticotropic hormone (ACTH) is often suboptimal. Corticosteroids help prevent adrenal insufficiency in thyroid storm and reduce peripheral conversion of T4 to T3. The recommended dose is an initial intravenous loading dose of 300 mg hydrocortisone, followed by 100 mg every 8 hours. Evaluation for infections as potential triggers of thyroid storm is essential. If an infection is identified, appropriate antibiotic therapy should be considered. Additionally, other metabolic conditions, such as diabetic ketoacidosis, stroke, or pulmonary embolism, should be managed according to established protocols.

Therapeutic Plasma Exchange (TPE)

In refractory thyroid storm with no clinical improvement, therapeutic plasma exchange (TPE) is an effective intervention for rapidly reducing circulating thyroid hormone levels (Pandey et al., 2020; Szczepiorkowski et al., 2010) This procedure involves extracting the patient's plasma from the blood components and replacing it with albumin or fresh frozen plasma. Müller et al. suggest that early initiation of TPE is indicated in cases of severe symptoms (such as cardio-thyrotoxicosis, neurological manifestations, and severe myopathy), rapid clinical deterioration, contraindications to conventional therapies, or failure of standard treatment (Muller et al., 2011; Pandey et al., 2020). The American Society for Apheresis (ASFA) recommends performing TPE at a frequency of daily or every 2–3 days until clinical improvement is achieved (Pandey et al., 2020)

Surgical Management

Achieving a euthyroid state is the primary prerequisite before surgical intervention, typically accomplished through the aforementioned medical therapies. In certain cases, particularly in patients with autonomous thyroid function, iodine contamination can lead to resistance to thionamides or iodine therapy due to a large intrathyroidal iodine pool (Pandey et al., 2020)

The patient in this case received treatment in accordance with established management protocols for thyroid storm. Propylthiouracil (PTU) 200 mg five times daily was administered to suppress thyroid hormone synthesis. Additionally, hydrocortisone 100 mg twice daily was given as adjunctive therapy to prevent adrenal insufficiency and inhibit the peripheral conversion of thyroxine (T4) to triiodothyronine (T3), a critical mechanism in thyroid storm management. Lugol's iodine was considered as part of the therapeutic regimen; however, it was unavailable at Wangaya General Hospital. Iodine therapy plays a key role in thyroid storm management by inhibiting the release of thyroid hormones, thereby preventing the secretion of T3 and T4 from thyroglobulin.

Further treatment included propranolol 20 mg three times daily and antibiotic therapy. Propranolol, a non-selective β-adrenergic antagonist, was used to attenuate the peripheral effects of excess thyroid hormones by blocking β-adrenergic receptors and reducing the conversion of T4 to T3. Antibiotics were initiated to target the underlying infection, which served as a precipitating factor for thyroid storm. Following ICU admission, the patient demonstrated progressive clinical improvement, permitting transfer to a general ward. After a total hospitalization period of seven days, the patient was discharged with the following medications: PTU 200 mg every 8 hours, Propranolol 10 mg every 8 hours (adjusted based on heart rate), Cefixime 200 mg every 12 hours, and Metronidazole 500 mg every 8 hours

CONCLUSION

Thyroid storm is a rare but life-threatening condition that requires rapid and appropriate management. Effective treatment necessitates a combination of therapies that work synergistically to reduce thyroid hormone levels and alleviate life-threatening symptoms. The administration of propylthiouracil (PTU), propranolol, hydrocortisone, and antibiotics has been shown to lead to clinical improvement in patients with thyroid storm triggered by infection.

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