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Thyroid Storm Precipitated by Medication Non-adherence and Infection Presenting with Proximal Myopathy: A Case Report

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Abstract

Thyroid storm is life-threatening endocrine emergency. It requires early recognition and aggressive management. A 59-year-old female with long-standing multinodular goitre, presented with complaints of fever, diarrhoea, palpitations, leg swelling and progressive weakness & poor compliance to antithyroid medications. On examination she was febrile, tachycardiac, with bilateral proptosis and multinodular goitre. Laboratory investigations revealed severe thyrotoxicosis. Burch -wartofsky score >45 (65) supported the diagnosis of thyroid storm.

She was managed with beta-blockers, antithyroid drugs, corticosteroids, insulin infusion, and broad-spectrum antibiotics. she initially showed clinical improvement with treatment and developed sudden respiratory distress requiring mechanical ventilation. With intensive care support, she subsequently improved and recovered. Our case highlights the critical importance of medication adherence in hyperthyroid patients and demonstrates that thyroid storm may deteriorate despite apparent biochemical improvement, necessitating vigilant monitoring and timely escalation of care.

Keywords: Thyroid storm, multinodular goitre, hyperthyroidism, non-compliance, cardiorespiratory failure.

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Introduction

Thyroid storm is rare and life-threatening exacerbation of hyperthyroidism and is associated with high (4-17%) mortality rate even with treatment.[1] It remains a clinical diagnosis characterized fever, tachycardia, delirium, seizures, coma, vomiting, diarrhoea and multiorgan involvement and scoring system such as Burch -wartofsky score Point Scale are commonly used to support the diagnosis and assess severity.[2,3,4] Thyrotoxicosis the state of thyroid hormone excess and hyperthyroidism is the result of excessive thyroid function. Thyroid storm is usually precipitated by acute illness, e.g., Infection, stroke, trauma, surgery, and discontinuation or poor adherence to antithyroid medications.[1] We present a case of thyroid storm in multinodular goitre precipitated by medication non-compliance and infection, complicated by tachyarrhythmia leading to acute decompensated left heart failure with pulmonary oedema.

Case Presentation

A 59-years-old female brought to emergency department with neck swelling for 9-years, progressive inability to stand and walk with leg swelling palpitations for 2 months, which had worsened over last 2 days. She also had fever, abdominal pain, vomiting and diarrhoea for last 5 days. Her history was significant for a diagnosis of long-standing multinodular goitre and hyperthyroidism and had been started on treatment with tab. carbimazole 20 mg once daily for 1.5 years; however, she was noncompliant to medications.

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At presentation she was conscious, anxious, agitated and emotionally labile had episodes of cry. she had fever with a temperature of 99 °F, tachycardia with a pulse rate of 150 beats per minute, respiratory rate of 18 beats/minute, had a blood pressure of 118/74 mm Hg with normal O_2 saturation on room air.

On her examination pallor, warm and moist skin, fine tremors, large multinodular goitre (Figure1) with an audible thyroid bruit, severe bilateral proptosis with lid retraction and a characteristic staring appearance (Figure 2) and bilateral pitting pedal oedema were noted. Systemic examination revealed severe proximal lower limb weakness with power of 1/5 at the hip and knee joints, marked tachycardia, bilateral clear chest with no adventitious sounds heard.

Based on the clinical history and rapid bedside assessment using the Burch–Wartofsky Point Scale (score 65), a diagnosis

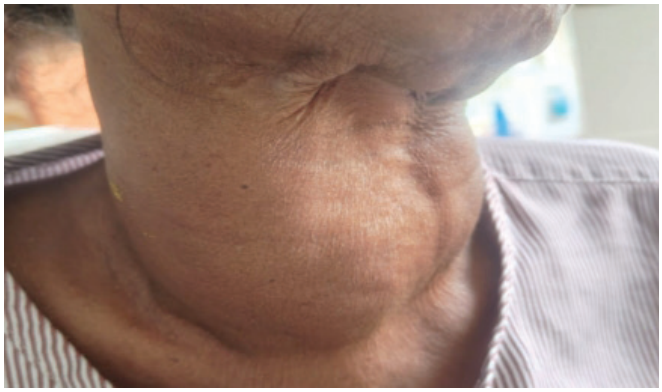

Figure 1: Multinodular Goitre with Neck swelling

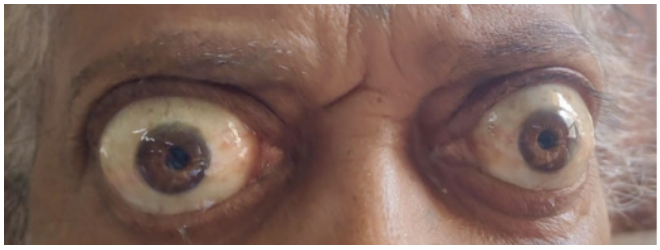

Figure 2: Severe bilateral proptosis with lid retraction

of thyroid storm in multinodular goitre precipitated by medication nonadherence and infection was made. An electrocardiogram showed marked sinus tachycardia with a heart rate of 150 beats per minute. On admission laboratory investigations revealed anaemia, neutrophilic leucocytosis, Platelet count WNL.

Thyroid Function Tests (17/02/2026) revealed thyrotoxicosis with depressed TSH level and markedly raised T3, T4 levels. Neck ultrasonography & Colour Doppler revealed an enlarged thyroid gland with heterogeneous echotexture and multiple nodules, increased vascularity in thyroid gland consistent with multinodular goitre.

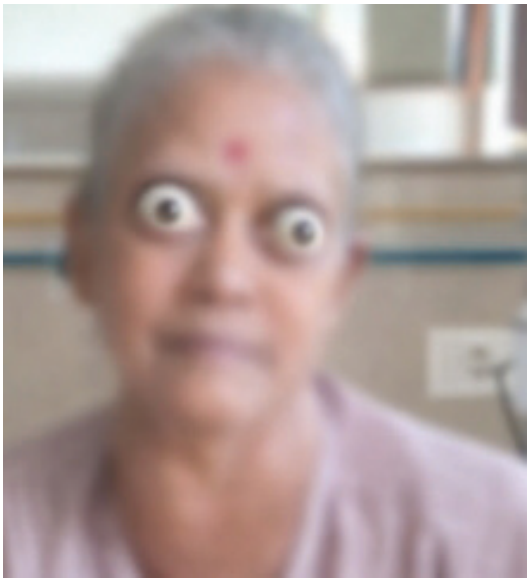

Figure 3: Facial appearance.

Table 1: Burch–Wartofsky Point Score (BWPS)[3] for diagnosis of thyroid storm.

Burch–Wartofsky Point Score (BWPS)

Diagnostic parameter	Scoring points	Score in case
1. Temperature		
99-99.9 °F	5	5
100–100.9 °F	10	
101-101.9 °F	15	
102-102.9 °F	20	
103-103.9 °F	25	
>104 °F	30	
2. Central nervous system effects		
Absent	0	
Mild (Agitation)	10	10
Moderate (delirium, psychosis & extreme lethargy)	20	
Severe (seizures and coma)	30	
3. Gastrointestinal -hepatic dysfunction		
Absent	0	
Moderate (diarrhoea, nausea/ vomiting, and abdominal pain)	10	10
Severe (unexplained jaundice)	20	
4. Cardiovascular dysfunction		
Tachycardia (beats per minute)		
90 – 109	5	
100- 119	10	
120 – 129	15	
>140	25	25
5. Congestive Heart failure		
Absent	0	
Mild (Pedal oedema)	5	5
Moderate (bibasilar rales)	10	
Severe (Pulmonary oedema)	15	
6. Atrial fibrillation		
Absent	0	0
Present	10	
7. Precipitating event		
Absent	0	
Present	10	10
Total score		65
> 45	Thyroid storm	
25 – 45	Impending storm	
<25	Storm Unlikely	

She was initiated on comprehensive management for thyroid storm oral propranolol 40 mg (beta blocker) 12 hourly for control of adrenergic symptoms and heart rate, antithyroid drug Carbimazole 20 mg 12 hourly doses escalated from a once-daily regimen, intravenous steroids, iv dexamethasone 4 mg 12 hourly, preferred over hydrocortisone to prevent further hypokalaemia, IV antibiotics injection ceftriaxone to address suspected precipitating infection was started. Insulin infusion started for glycaemic control along with Intravenous potassium (KCl) and magnesium supplementation for hypokalaemia and hypomagnesemia correction.

The patient showed clinical improvement within initial 24 hours, reduction in symptoms and vitals stable pulse rate decreased to 100 beats/min. Subsequent intravenous steroids were gradually tapered, while other therapies were continued. On fourth day of admission, she had sudden onset severe palpitations with shortness of breath at rest suggesting possible cardiovascular& respiratory failure.

On examination she was conscious but agitated with Glasgow Coma Scale: E4V5M6. Her important clinical findings were fever (T-100.4°F), marked tachycardia (Pulse was 180 beats/min) (marked tachycardia) with Blood Pressure: 100/70 mmHg, hypoxia (SpO_2 66% on room air and 76% on 10 L/min oxygen support via face mask) bilateral pitting pedal oedema. Her systemic examination revealed marked tachycardia, bilateral basal crackles heard on auscultation, suggestive of pulmonary congestion which were not there on day of admission initially. In view of clinical deterioration, she was shifted to intensive care unit. She developed acute cardiorespiratory distress, likely secondary to acute left ventricular failure due to thyroid storm. She was intubated and mechanically ventilated for worsening respiratory failure, continued medical therapy.

In ICU, Electrocardiogram showed sinus tachycardia with a heart rate of 180 beats/min, Chest Radiography revealed Cardiomegaly, Bilateral perihilar opacities & Mild pleural effusion suggestive of pulmonary oedema, correlating with the patient's acute respiratory deterioration laboratory evaluation revealed improved biochemical parameters despite clinical deterioration, hypokalaemia & hypomagnesemia was persistent despite iv correction.

Transthoracic Echocardiography revealed structurally normal cardiac chambers with preserved biventricular systolic function (LVEF ~60%), mild mitral regurgitation, mild tricuspid regurgitation, mild pulmonary hypertension (RVSP ~35 mm Hg), No evidence of cardiomyopathy or diastolic dysfunction.

Diagnosis of Thyroid storm (Burch–Wartofsky Point Score: 65)[3] in a case of multinodular goitre complicated by tachyarrhythmia leading to acute decompensated left heart failure with pulmonary oedema precipitated by medication non-compliance and infection.

In intensive care unit, she was managed for thyroid storm with acute cardiopulmonary decompensation with intravenous metoprolol infusion, Intravenous hydrocortisone 50mg 6hourly, Antithyroid therapy Carbimazole 20 mg

Table 2: Thyroid Function Tests done on admission and during hospitalisation revealed.

	17/02/2026	27/02/2026	03/03/2026
TSH(µIU/mL)	<0.005 µIU/mL	0.01	0.01 µIU/mL
T3(ng/dL)	651 ng/dL	0.57	2.90 pg./ml (F T3)
T4(µg/dL)	24.9 µg/dL	3.52	0.94 ng/dL (FT4)

Table 3: Laboratory investigations.

Laboratory Parameters	On Day of admission	In ICU
Haemoglobin	9 gm/dL	8.6 gm/dL
WBC count	14000/mm^3	10,200/ mm^3
Platelets	187,000/mm^3	227,000/mm^3
Sr. Creatinine/ Sr. Urea	0.8mg/dL/ 20mg/dL	0.6mg/dL/ 22mg/dL
Sr. Sodium/ Sr. potassium/sr. Mg	142 mmol/L /2.4 mmol/L/1.30mg/dL	145 mmol/L/2.8 mmol/L/1.37 mg/dL
Sr. SGOT/Sr. SGPT	25 U/L/29U/L	60 U/L/32 U/L
Sr. Albumin	2.1gm/dL	2.5gm/dL
Sr. Bilirubin(T)/(D)	1.01 mg/dL/ 0.59 mg/dL	1.03mg/dL/ 0.6mg/dL
LDL/HDL ratio	1.1	
Urine analysis	Glucose: 2+, Ketones: 2+, Pus cells: 10-12/ HPF	Glucose: 1+, Ketones: 1+, Pus cells: 4-6/HPF
Blood sugar (R)	565mg/dL	190 mg/dL
Sr. calcium	8.69mg/dL	8.0mg/dL
CRP	125 mg/L	57.94 mg/L
HHH	negative	

12 hourly, iv diuretics furosemide 40mg bolus doses for management of pulmonary oedema. Aggressive correction of hypokalaemia and hypomagnesemia, Empirical intravenous antibiotics continued for suspected infection. She was on ventilatory support for 2 days, and which was gradually tapered and she was successfully weaned off ventilatory support and extubated subsequently maintained on oxygen via face mask later weaned off oxygen support within 24 hours. After stabilization she was shifted to the ward, where she remained hemodynamically stable and was continued on oral beta-blockers propranolol 40 mg 12 hourly, oral prednisolone (tapering regimen), antithyroid medication (carbimazole), ongoing potassium supplementation. Physiotherapy continued for proximal muscle weakness.

Further planned investigations serum TRAb, nerve conduction studies, and electromyography could not be performed due to financial constraints. Radioactive iodine uptake (RAIU) was not done in view of the acute clinical presentation and need for urgent treatment.

Follow-up and Outcomes

The Patient was hospitalised for prolonged period and showed progressive clinical improvement with stabilization of cardiovascular and respiratory status and improvement in muscle strength. She was discharged in a stable condition on tab carbimazole 20 mg once in 24 hours, tab propranolol 40 mg once in day. She was advised regular follow-up, medication adherence, and evaluation for surgery. Timely intensive care management with multidisciplinary intervention resulted in favourable clinical recovery in this life-threatening presentation of thyroid storm.

DISCUSSION

Thyroid storm is rare and life-threatening exacerbation of thyrotoxicosis and is associated with high (4-17%) mortality rate even with treatment.[1] In our case diagnosis was supported by a Burch–Wartofsky Point Score of 65 in the setting of classical systemic features, consistent with established diagnostic criteria.[3,4]

Two major precipitating factors identified in our case were poor adherence to antithyroid therapy and urinary tract infection. These triggers are well documented and frequently act synergistically to precipitate thyroid storm.[1,3,5] Graves' disease, toxic multinodular goitre remains major cause of thyrotoxicosis in older patients, often presenting insidiously and increasing the risk of delayed diagnosis and severe decompensation.[1,4]

The metabolic profile in our case revealed marked hyperglycaemia with ketonuria, hypoalbuminemia reflecting the hypermetabolic state induced by excess thyroid hormones. Thyroid hormones enhance hepatic gluconeogenesis, increase gastrointestinal glucose absorption, and promote peripheral insulin resistance increase degradation and synthesis of proteins.[5] Significant neuromuscular involvement with profound lower limb weakness was notable. Thyrotoxic proximal myopathy is a recognized manifestation,[1,4,5] in our case weakness was likely multifactorial, with contributions from catabolic state, electrolyte disturbances (notably hypokalaemia and hypomagnesemia), and systemic illness.[1,5]

A key clinical insight from our case is the occurrence of delayed cardiopulmonary decompensation despite initial biochemical improvement. This highlights an important clinical principle: biochemical improvement does not necessarily indicate resolution of the underlying pathophysiological processes, and patients remain at risk for sudden deterioration.[2,5] Excess thyroid hormone increases cardiac output, myocardial oxygen demand, and adrenergic sensitivity, predisposing patients to tachyarrhythmias, high-output cardiac failure, and pulmonary oedema. Notably, even in the presence of preserved ejection fraction, patients may develop acute decompensation due to impaired physiological reserve and functional cardiac dysfunction, as observed in this case.[1,2] Our case reinforces the importance of early, aggressive, and sustained therapy including beta-blockers, antithyroid drugs, corticosteroids, and supportive care with close monitoring as patients may deteriorate even after apparent stabilization. In retrospect, premature tapering of corticosteroids may have contributed to delayed deterioration, emphasizing the need for cautious and individualized de-escalation of therapy.[4,5]

Case highlights unpredictable nature of thyroid storm, importance of early recognition, strict treatment adherence, and vigilant monitoring for delayed complications.

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Consent for publication:

A Written informed consent was obtained from the patient for publication of this case report and accompanying images.