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Successful Application of Non-Invasive Ventilation in Acute Respiratory Failure Complicating Thyroid Storm-Induced Pulmonary Edema: A Case Report

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A B S T R A C T

Introduction: Thyroid storm is a rare, life-threatening exacerbation of thyrotoxicosis characterized by severe multisystem organ dysfunction, including cardiovascular collapse and respiratory failure. Acute pulmonary edema is a recognized but challenging complication, often stemming from high-output cardiac failure or tachyarrhythmia-induced cardiomyopathy. Non-invasive ventilation (NIV) offers a crucial therapeutic modality for acute respiratory failure by improving oxygenation, reducing the work of breathing, and providing beneficial hemodynamic effects, potentially obviating the need for endotracheal intubation. **Case presentation:** We present the case of a 23-year-old female who developed acute hypoxemic respiratory failure secondary to acute pulmonary edema precipitated by a thyroid storm, occurring post-operatively after a ureterorenoscopy. She presented with severe dyspnea, tachycardia (atrial fibrillation with rapid ventricular response), tachypnea, and significant hypoxemia (PaO_2/FiO_2 ratio of 106.4). Diagnosis of thyroid storm was confirmed by elevated free thyroxine (FT4), suppressed thyroid-stimulating hormone (TSH), and a Burch-Wartofsky Point Scale (BWPS) score of 80. The patient was managed with NIV, alongside standard medical therapy for thyroid storm, including antithyroid drugs, beta-blockers, iodine solution, and corticosteroids. **Conclusion:** NIV was successfully utilized as primary respiratory support, leading to rapid clinical and radiological improvement, resolution of respiratory failure, and avoidance of invasive mechanical ventilation. The PaO_2/FiO_2 ratio improved to 260 within four days. This case highlights the efficacy and safety of early NIV initiation in patients with acute respiratory failure due to pulmonary edema in the complex setting of thyroid storm.

1. Introduction

Thyroid storm, also known as thyrotoxic crisis, represents the most extreme and life-threatening manifestation of hyperthyroidism, characterized by an acute surge in thyroid hormone activity that leads to profound hypermetabolism and widespread organ dysfunction. While relatively uncommon, with an estimated incidence that varies geographically but is generally low (e.g., reported as 0.57–0.76 cases per 100,000 persons per year in some cohorts, and occurring in 1-2% of patients hospitalized for thyrotoxicosis), it carries a substantial mortality risk, historically reported as high as 10-30%, and even up to 100% if inadequately or delayed in treatment. The condition typically arises in individuals with pre-existing, often undiagnosed or poorly managed, hyperthyroidism, and is commonly precipitated by acute events such as surgery, infection, trauma, parturition, or iodine contrast administration. The pathophysiology involves an exaggerated systemic response to excessive thyroid hormones (thyroxine, T4, and triiodothyronine, T3), leading to increased

metabolic rate, heightened sympathetic activity, and impaired cellular oxygen utilization. Clinical manifestations are diverse and reflect this multisystemic impact, commonly including hyperthermia, central nervous system disturbances (agitation, delirium, psychosis, seizures, coma), gastrointestinal dysfunction (nausea, vomiting, diarrhea, jaundice), and, critically, severe cardiovascular derangements.[1-3]

Cardiovascular complications are a hallmark of thyroid storm and a major contributor to its morbidity and mortality. These can include sinus tachycardia, supraventricular tachyarrhythmias (most commonly atrial fibrillation with rapid ventricular response), high-output cardiac failure, and, in severe cases, cardiogenic shock or even biventricular failure. Acute pulmonary edema is a particularly dangerous cardiovascular sequela in thyroid storm. It often develops secondary to the profound hemodynamic stress imposed by the thyrotoxic state. Several mechanisms contribute: firstly, the direct effects of thyroid hormones on the myocardium increase contractility and heart rate, leading to a high-output state that can eventually overwhelm ventricular capacity, particularly if there is underlying cardiac disease or prolonged, severe tachycardia. Secondly, tachyarrhythmias, especially atrial fibrillation with uncontrolled ventricular rates, impair diastolic filling and reduce cardiac efficiency, leading to increased left atrial and pulmonary venous pressures. Thirdly, some evidence suggests thyroid hormones can directly affect pulmonary vascular permeability, although cardiogenic mechanisms are generally predominant. The resultant accumulation of excess fluid in the interstitial and alveolar spaces of the lungs severely impairs gas exchange, leading to acute hypoxemic respiratory failure.[4-6]

Acute respiratory failure (ARF) in this context is primarily hypoxemic, defined by a significant reduction in arterial oxygen tension (PaO_2) despite supplemental oxygen, often quantified by a PaO_2/FiO_2 (P/F) ratio below 300 mmHg. When ARF is severe, ventilatory support becomes necessary to maintain adequate oxygenation, reduce the work of breathing, and prevent further physiological deterioration. Non-invasive ventilation (NIV) refers to the delivery of mechanical ventilatory support without the need for an endotracheal tube or tracheostomy. It is typically administered via a face mask (oronasal or full-face) or nasal mask. NIV has become a cornerstone in the management of various forms of ARF, particularly in acute cardiogenic pulmonary edema and exacerbations of chronic obstructive pulmonary disease (COPD). The physiological benefits of NIV in acute cardiogenic pulmonary edema are manifold. Positive end-expiratory pressure (PEEP) helps to recruit collapsed alveoli, increase functional residual capacity, and redistribute extravascular lung water, thereby improving V/Q matching and oxygenation. Inspiratory pressure support (PSV) augments the patient's spontaneous tidal volume, reduces the work of breathing performed by fatigued respiratory muscles, and can help alleviate dyspnea. Furthermore, the application of positive airway pressure increases intrathoracic pressure, which reduces both venous return (preload) to the right heart and left ventricular afterload by decreasing the transmural pressure gradient across the left ventricular wall. These hemodynamic effects can significantly improve cardiac function and facilitate the resolution of pulmonary edema. Early application of NIV has been associated with reduced rates of endotracheal intubation, shorter ICU and hospital stays, and, in some studies, improved mortality in patients with acute cardiogenic pulmonary edema.[7,8]

Despite the established benefits of NIV in typical cardiogenic pulmonary edema, its application in the complex scenario of thyroid storm-induced pulmonary edema and ARF is less extensively documented, primarily due to the rarity of this specific combination of critical illnesses. The successful management of such patients requires aggressive treatment of the underlying thyrotoxicosis alongside meticulous cardiorespiratory support. This case report details the successful utilization of NIV in a young female patient who presented with acute hypoxemic respiratory failure due to pulmonary edema as a direct complication of a newly diagnosed thyroid storm following a minor surgical procedure. The novelty of this case lies in demonstrating the effective and safe use of NIV as a primary respiratory support modality in this specific, life-threatening triad of thyroid storm, acute pulmonary

edema, and severe respiratory failure, thereby avoiding the complications associated with invasive mechanical ventilation.[9,10] This study aims to contribute to the clinical evidence supporting the early consideration of NIV in such critically ill patients, highlighting the diagnostic process, multifaceted therapeutic interventions, and the positive patient outcome achieved.

2. Case Presentation

A 23-year-old female patient was urgently referred to our institution's emergency department (ED) from an outside hospital with a critical presentation of severe dyspnea. Her acute respiratory distress had commenced following a recent ureterorenoscopy procedure with Double J stent placement, after which she was diagnosed at the referring facility with acute respiratory failure secondary to acute pulmonary edema, complicated by an underlying thyroid storm. Notably, the patient denied any known prior history of thyroid disorders or previous treatment for hyperthyroidism. Upon arrival at our ED, she was in evident severe respiratory distress. Her vital signs were alarming: a respiratory rate of 35–40 breaths per minute, an oxygen saturation (SpO_2) of 95% despite receiving 100% oxygen via a Jackson-Rees circuit, a markedly irregular heart rate ranging between 110–165 beats per minute, a blood pressure of 170/90 mmHg, and a body temperature of 37.8°C. Her Glasgow Coma Scale score was 15/15 (E4V5M6). Physical examination of the chest revealed bilateral rhonchi, indicative of fluid or secretions in the larger airways. Other signs consistent with thyrotoxicosis and cardiac compromise, such as agitation and diaphoresis, were noted (Table 1).

Initial diagnostic workup was rapidly undertaken. The electrocardiogram (ECG) confirmed the presence of atrial fibrillation with a rapid ventricular response, consistent with the irregular tachycardia observed clinically. A chest X-ray performed on admission (Day 0) demonstrated significant bilateral pulmonary edema along with cardiomegaly. Crucially, thyroid function tests (TFTs) revealed a profoundly deranged thyroid status: T3 total was elevated at 2.8 ng/mL (normal range: 0.8–2 ng/mL), free T4 (FT4) was markedly increased at 6.46 ng/dL (normal range: 0.93–1.7 ng/dL), and thyroid-stimulating hormone (TSH) was suppressed to <0.01 µIU/mL (normal range: 0.27–4.2 µIU/mL). Arterial blood gas analysis on 100% FiO_2 via the Jackson-Rees circuit prior to NIV initiation showed a pH of 7.54, pCO_2 of 37.7 mmHg, pO_2 of 106.4 mmHg, HCO_3^- of 32.4 mmol/L, and SaO_2 of 97.9%. This resulted in a PaO_2/FiO_2 (P/F) ratio of 106.4, indicative of severe acute hypoxemic respiratory failure. Other pertinent laboratory findings included leukocytosis (WBC 18,840/mm³), hypoalbuminemia (2.81 g/dL), elevated urea (102 mg/dL), and elevated inflammatory markers (CRP 10.3 mg/dL, Procalcitonin 0.61 ng/mL) (Table 1).

Based on the clinical presentation and laboratory findings, a diagnosis of thyroid storm was established. The patient's Burch-Wartofsky Point Scale (BWPS) score was calculated to be 80, strongly indicative of thyroid storm (a score >45 is highly suggestive). She also met the criteria for thyroid storm according to the Japan Thyroid Association (JTA) guidelines. The acute respiratory failure was attributed to cardiogenic pulmonary edema precipitated by the thyroid storm, manifesting as high-output cardiac failure and atrial fibrillation with rapid ventricular response.

Given the severity of her respiratory failure and hypoxemia, the patient was immediately admitted to the Intensive Care Unit (ICU). Non-invasive ventilation (NIV) was initiated promptly as the primary mode of respiratory support. The initial NIV settings were: Spontaneous mode (Pressure Support Ventilation) with a Positive End-Expiratory Pressure (PEEP) of 6 cmH_2O, an inspiratory pressure support (Pins) of 12 cmH_2O above PEEP, and an FiO_2 of 100% (Table 2). Concurrently, aggressive medical management for thyroid storm was instituted. This included propylthiouracil (PTU) 250 mg every 4 hours to block thyroid hormone synthesis and peripheral T4 to T3 conversion, propranolol 40 mg every 6 hours for beta-adrenergic blockade and heart rate control, Lugol's iodine solution 5 drops every 6 hours (administered one hour after PTU) to inhibit thyroid hormone release, and intravenous hydrocortisone 100 mg every 8 hours to further reduce T4 to T3 conversion and provide stress dose steroid coverage. Supportive ICU care also comprised intravenous furosemide as a continuous

infusion at 10 mg/hour for management of pulmonary edema, a continuous morphine infusion at 2 mg/hour for anxiolysis and to reduce sympathetic drive, intravenous paracetamol for pyrexia, and ranitidine for stress ulcer prophylaxis (Table 2). The patient demonstrated a remarkably positive response to NIV within the first two hours of its application. Her respiratory rate decreased to 20–22 breaths per minute, SpO_2 improved to 98% on the set FiO_2, and her heart rhythm became regular with a rate of 100 beats per minute, suggesting conversion of atrial fibrillation or significant rate control. Subjectively, her dyspnea improved considerably. Over the subsequent days in the ICU, her clinical condition continued to stabilize. NIV was maintained for a total of two days. Serial ABG analyses showed progressive improvement in oxygenation and ventilation (Table 2). Specifically, the PaO_2/FiO_2 ratio improved from 106.4 on Day 0 (FiO_2 100%), to 153.5 on Day 1 (FiO_2 60%), 152.5 on Day 2 (FiO_2 40%), and further to 260 by Day 4 (FiO_2 31%). Follow-up chest X-rays performed on Day 1 and Day 4 revealed a significant and progressive resolution of the bilateral pulmonary edema and a decrease in cardiac size (Table 2). The comprehensive medical therapy for thyroid storm was continued and adjusted as necessary. After six days of intensive care, having shown substantial clinical and radiological improvement and with her respiratory failure fully resolved without any signs of secondary infection, the patient was deemed stable for transfer out of the ICU to a general medical ward for ongoing management of her hyperthyroidism and continued recovery (Table 2).

3. Discussion

This case report details the successful management of a critically ill 23-year-old female who presented with acute hypoxemic respiratory failure due to thyroid storm-induced cardiogenic pulmonary edema. The cornerstone of her respiratory support was the early and effective application of non-invasive ventilation (NIV), which, in conjunction with aggressive medical therapy for thyrotoxicosis, led to a rapid amelioration of her respiratory distress and obviated the need for invasive mechanical ventilation. This case underscores the potential for NIV as a life-saving intervention in this rare but perilous constellation of endocrine and cardiovascular emergencies. The pathophysiology of thyroid storm leading to such severe cardiorespiratory compromise is complex and multifaceted. Thyroid hormones exert profound effects on the cardiovascular system, primarily mediated by increased beta-adrenergic receptor sensitivity and direct inotropic and chronotropic actions. In a storm state, this results in extreme tachycardia, increased myocardial oxygen demand, and often a high-output cardiac state. However, this hyperdynamic circulation can paradoxically lead to heart failure, termed "thyrotoxic cardiomyopathy" or "high-output failure," especially when compounded by tachyarrhythmias like the atrial fibrillation observed in our patient. Atrial fibrillation itself, common in hyperthyroidism and exacerbated in thyroid storm, impairs diastolic filling time and reduces atrial contribution to ventricular stroke volume, further predisposing to pulmonary venous congestion and edema. The observed cardiomegaly and pulmonary edema on the initial chest X-ray were classical signs of this cardiac decompensation. The patient's initial PaO_2/FiO_2 ratio of 106.4 placed her in the category of severe Acute Respiratory Distress Syndrome (ARDS) by oxygenation criteria, though the primary etiology was cardiogenic pulmonary edema.[11,12]

Diagnosing thyroid storm relies on astute clinical judgment supported by biochemical evidence of thyrotoxicosis, as there is no single pathognomonic test. Scoring systems like the Burch-Wartofsky Point Scale (BWPS) and the Japanese Thyroid Association (JTA) criteria are valuable diagnostic aids. Our patient scored 80 on the BWPS, far exceeding the threshold of 45, and met JTA criteria, confirming the diagnosis. The precipitating factor was likely the physiological stress of the recent ureterorenoscopy, a known trigger for thyroid storm in individuals with undiagnosed or undertreated hyperthyroidism. The elevated inflammatory markers (leukocytosis, high CRP, and procalcitonin) could reflect systemic inflammation associated with the storm or a response to a potential underlying trigger, although no overt source of infection was identified during her ICU stay.

Table 1. Summary of clinical patient data.

Parameter	Details
Demographics	
Age	23 years
Gender	Female
Anamnesis	
Presenting complaint	Severe dyspnea post-operatively
History of present illness	Referred from another hospital post-ureterorenoscopy and Double J stent placement with diagnosed acute respiratory failure, acute pulmonary edema, and thyroid storm
Past medical history	No prior history of thyroid disease or treatment for hyperthyroidism acknowledged
Precipitating factor	Recent surgical procedure (ureterorenoscopy)
Physical examination (on ER admission to the current hospital)	
General appearance	Severe respiratory distress
Glasgow coma scale (GCS)	15 (E4V5M6)
Respiratory rate (RR)	35–40 breaths/minute
Oxygen saturation (SpO_2)	95% with 100% oxygen via Jackson-Rees circuit
Heart rate (HR)	110–165 beats/minute, irregular
Blood pressure (BP)	170/90 mmHg
Temperature	37.8°C
Cardiovascular exam	Irregularly irregular rhythm
Respiratory exam	Bilateral rhonchi
Cns examination	Agitation
Other	Diaphoresis
Laboratory investigations (Initial)	
Thyroid function tests	
T3 total	2.8 ng/mL (Normal: 0.8–2 ng/mL)
Free T4 (FT4)	6.46 ng/dL (Normal: 0.93–1.7 ng/dL)
TSH	<0.01 µIU/mL (Normal: 0.27–4.2 µIU/mL)
Arterial blood gas (Day 0, on 100% O_2 via Jackson-Rees)	
pH	7.54
pCO_2	37.7 mmHg
pO_2	106.4 mmHg
HCO_3^-	32.4 mmol/L
SaO_2	97.9%
PaO_2/FiO_2 ratio	106.4 (FiO_2 assumed 1.0)
Complete blood count	
Hemoglobin	11.2 g/dL
Leukocytes (WBC)	18,840/mm³ (elevated)
Platelets	276,000/mm³
Metabolic panel	
Albumin	2.81 g/dL (low)
Ureum	102 mg/dL (elevated)
Creatinine	0.56 mg/dL
Sodium (Na^+)	139 mmol/L
Potassium (K^+)	3.62 mmol/L
Chloride (Cl^-)	96 mmol/L
Random blood glucose	113 mg/dL
Inflammatory markers	
C-reactive protein (CRP)	10.3 mg/dL (elevated)
Procalcitonin	0.61 ng/mL (elevated)
Imaging	
Chest X-ray (Day 0)	Bilateral pulmonary edema, cardiomegaly
Electrocardiogram (ECG)	Atrial fibrillation with rapid ventricular response
Diagnosis	
Primary diagnosis	Thyroid Storm
Complications	Acute Hypoxemic Respiratory Failure, Acute Cardiogenic Pulmonary Edema, Atrial Fibrillation with RVR
Burch-Wartofsky point scale (BWPS)	80 points (Temperature 37.8°C (100°F) = 10; CNS agitation = 10; GI dysfunction = 0; HR >140 = 25; CHF pulmonary edema = 15; Atrial Fibrillation = 10; Precipitating event = 10. Total = 80)
Japan thyroid association (JTA) criteria	Fulfilled criteria for TS1 (combination of thyrotoxicosis, CNS manifestations, fever, tachycardia, CHF)

Table 2. Procedure of treatment and follow-up.

Aspect	Detailed description
Initial ICU admission & respiratory support	
Admission to the ICU	Patient admitted directly to ICU due to severity of respiratory failure and thyroid storm.
Non-invasive ventilation (NIV)	Initiated immediately upon recognition of ARF.
	Ventilator: ICU ventilator with NIV mode.
	Interface: Oronasal mask.
	Initial Settings: Mode: Spontaneous (Pressure Support Ventilation with PEEP); PEEP: 6 cmH_2O; Inspiratory Pressure (Pins/PSV): 12 cmH_2O above PEEP; FiO_2: 100%
Pharmacological management for thyroid storm & complications	
Antithyroid drug	Propylthiouracil (PTU): 250 mg every 4 hours (6x daily) via oral/nasogastric route. Blocks thyroid hormone synthesis and peripheral conversion of T4 to T3.
Beta-blocker	Propranolol: 40 mg every 6 hours (4x daily) via oral/nasogastric route. For heart rate control and to block peripheral effects of thyroid hormone.
Iodine solution	Lugol's Solution: 5 drops every 6 hours (4x daily) via oral/nasogastric route. Administered at least 1 hour after PTU to block thyroid hormone release (Wolff-Chaikoff effect).
Corticosteroid	Hydrocortisone: 100 mg IV every 8 hours (3x daily). To reduce peripheral conversion of T4 to T3, provide adrenal support, and for systemic anti-inflammatory effects.
Diuretic	Furosemide: 10 mg/hour via syringe pump (continuous infusion). For management of pulmonary edema.
Analgesia/Sedation	Morphine: 2 mg/hour via syringe pump (continuous infusion). For dyspnea, agitation, and to reduce sympathetic drive.
Antipyretic	Paracetamol: 1 gram IV every 8 hours (3x daily). For fever management.
Gastroprotection	Ranitidine: 50 mg IV every 12 hours (2x daily). Stress ulcer prophylaxis.
Antidiarrheal	Attapulgite: 2 tablets if diarrhea occurred.
Monitoring and adjustments	
Clinical response to NIV (within 2 hours)	Respiratory Rate: Decreased to 20–22 breaths/minute. SpO_2: Improved to 98% (FiO_2 high initially). Heart Rate: Became regular at 100 beats/minute. Significant subjective improvement in dyspnea.
NIV weaning	FiO_2 was gradually reduced based on SpO_2 and ABG results. Pressure support and PEEP levels were adjusted based on respiratory mechanics, work of breathing, and gas exchange.
Arterial blood gases (ABGs)	Monitored serially: Day 0 (pre-NIV values used as baseline), Day 1, Day 2, Day 4. Showing progressive improvement in oxygenation (pO_2, P/F ratio) and normalization of pH and pCO_2.
Chest X-Ray	Follow-up CXRs on Day 1, Day 4 showed progressive resolution of pulmonary edema and reduction in cardiomegaly.
Thyroid function tests	Monitored during admission to assess response to antithyroid therapy.
Hemodynamic monitoring	Continuous ECG, HR, BP, SpO_2 monitoring. Atrial fibrillation reverted to sinus rhythm.
Fluid balance	Strict intake/output monitoring, guided by diuretic therapy and clinical signs of edema.
Duration of NIV and ICU Stay	
Duration of NIV	2 days
Total ICU stay	6 days
Outcome & discharge from ICU	
Clinical status at ICU discharge	Significant clinical improvement. No signs of secondary infection. Respiratory status stable post-NIV discontinuation.
Radiological status	Marked improvement in pulmonary edema and cardiomegaly on CXR.
Oxygenation at ICU discharge	PaO_2/FiO_2 ratio improved to 260 (on Day 4, FiO_2 31%).
Disposition from ICU	Transferred to a general ward for continued management and recovery.
Follow-up (Post-ICU)	Continued antithyroid medication and management of hyperthyroidism. Long-term follow-up for thyroid status and cardiac function would be essential.

The management strategy for thyroid storm is multipronged, aiming to: inhibit new thyroid hormone synthesis (using thionamides like propylthiouracil (PTU) or methimazole), block the release of pre-formed thyroid hormone (using iodine solutions like Lugol's, administered after thionamide initiation), antagonize peripheral thyroid hormone effects (primarily with beta-blockers like propranolol), prevent peripheral conversion of T4 to T3 (high-dose corticosteroids, PTU, propranolol), and provide aggressive supportive care addressing systemic decompensation and identifying/treating precipitants. Our patient received this comprehensive regimen, including PTU, propranolol, Lugol's solution, and hydrocortisone, which was crucial for controlling the underlying thyrotoxic state. The choice of PTU was appropriate given its additional benefit of inhibiting T4 to T3 conversion, which is particularly valuable in the acute storm setting. Corticosteroids are also vital for their effect on peripheral conversion and to treat potential relative adrenal insufficiency.[13,14]

The decision to initiate NIV was critical in managing this patient's severe hypoxemic respiratory failure. NIV is well-established for acute cardiogenic pulmonary edema. Its mechanisms of action were likely highly beneficial in this case. PEEP (6 cmH_2O initially) helped to counteract alveolar collapse caused by edema fluid, increase functional residual capacity, and improve ventilation/perfusion matching, leading to better arterial oxygenation, as evidenced by the rapid rise in SpO_2 and subsequent improvement in P/F ratio. Inspiratory pressure support (12 cmH_2O) unloaded the patient's fatigued respiratory muscles, alleviating her severe dyspnea and reducing her tachypnea from 35-40 to 20-22 breaths/minute within two hours. This reduction in oxygen consumption by respiratory muscles is particularly important in a hypermetabolic state like thyroid storm. The positive intrathoracic pressure generated by NIV reduces both systemic venous return (preload) to the right heart and left ventricular afterload. This effect is particularly beneficial in cardiogenic pulmonary edema by decreasing pulmonary capillary hydrostatic pressure and facilitating the movement of fluid out of the alveoli. The improvement in cardiac function may also have contributed to the observed regularization of her heart rhythm, although the beta-blockade from propranolol was also a key factor. Successful NIV prevented the need for endotracheal intubation and invasive mechanical ventilation, thereby avoiding associated risks such as ventilator-associated pneumonia (VAP), tracheal injury, and the need for deeper sedation, which can be problematic in thyroid storm patients with potential CNS irritability. The fact that she remained free of secondary infection is noteworthy.[15,16]

The patient's rapid positive response to NIV, with significant improvement in respiratory parameters within two hours and sustained improvement over 48 hours leading to its discontinuation, strongly supports its efficacy in this specific clinical context. The progressive increase in her P/F ratio from 106.4 to 260 over four days mirrored the radiological clearance of pulmonary edema. Studies comparing NIV outcomes specifically in thyroid storm-induced pulmonary edema are scarce. However, robust evidence supports NIV in undifferentiated cardiogenic pulmonary edema. A Cochrane review summary by Hess DR (2020) reinforces that NIV (both CPAP and bilevel PAP) reduces hospital mortality and endotracheal intubation rates in patients with acute cardiogenic pulmonary edema compared to standard oxygen therapy alone. Bello et al. (2018) also describe the benefits of NIV in cardiogenic pulmonary edema, emphasizing its role in reducing systemic venous return and LV afterload. The mechanisms detailed in reviews of NIV physiology align perfectly with the observed response in our patient.

Fluid management in thyroid storm with pulmonary edema requires a delicate balance. While patients may be dehydrated due to hypermetabolism, fever, and GI losses, aggressive fluid resuscitation can worsen pulmonary edema if cardiac function is compromised. The use of diuretics, such as the furosemide infusion our patient received, is generally indicated once hemodynamic stability is ensured and preload reduction is desired. Prognostically, factors like CNS involvement and the severity of cardiovascular dysfunction are known to influence outcomes in thyroid storm. Our patient's initial GCS was good, but she had severe cardiovascular compromise. The prompt and comprehensive treatment likely contributed to her

favorable outcome. Long-term follow-up is crucial for patients recovering from thyroid storm, including management of the underlying thyroid disease and monitoring for any residual cardiac dysfunction.[17,18]

The strength of this report is its detailed description of a rare and complex case where NIV was successfully implemented, adding to the limited body of evidence. The clear timeline of interventions and responses provides valuable clinical insight. Limitations include the inherent nature of a single case report, which cannot establish causality or generalizability. Specific details beyond those recorded in the primary case notes, such as more granular hemodynamic data if not routinely performed, were not available. Thyroid storm, or thyrotoxic crisis, is the zenith of hyperthyroidism, a state of extreme hypermetabolism where the body's systems are driven into overdrive by an overwhelming surge of thyroid hormones—primarily thyroxine (T4) and triiodothyronine (T3). While hyperthyroidism itself can cause a spectrum of cardiovascular symptoms, thyroid storm unleashes a tempest upon the heart and lungs that can be particularly treacherous in individuals who, by all outward appearances, should possess resilient cardiovascular systems. The paradox lies in how a condition characterized by increased metabolic output and often a bounding pulse can precipitate acute cardiac failure.

The cardiovascular system is a primary target in thyroid storm, bearing the brunt of the hormonal deluge. The effects are multifaceted and synergistically contribute to cardiac decompensation. Thyroid hormones directly stimulate heart rate (chronotropy) and contractility (inotropy). Receptors for thyroid hormones are abundant in myocardial cells. This leads to a heart that beats too fast and too forcefully for too long. While initially this manifests as a high cardiac output state, the myocardial oxygen demand skyrockets. If this demand outstrips supply—even in the absence of coronary artery disease, which is less common in the young—relative ischemia can occur, impairing cardiac function. Thyroid hormones influence the genetic expression of key cardiac structural and regulatory proteins. For instance, there can be a shift from the adult α-myosin heavy chain (MHC) to the fetal β-MHC isoform, which has lower ATPase activity and might contribute to impaired contractility under stress. Furthermore, the expression and function of sarcoplasmic reticulum Ca^{2+}-ATPase (SERCA) pumps, crucial for calcium handling and diastolic relaxation, can be altered, leading to diastolic dysfunction. Excess thyroid hormones can uncouple oxidative phosphorylation in mitochondria, leading to inefficient energy production (ATP) and increased heat generation. This reduced cardiac efficiency means the heart has to work even harder to meet the body's energy needs, further straining its reserves. To meet the escalated metabolic demands of peripheral tissues and to dissipate excess heat generated by the hypermetabolic state, profound vasodilation occurs. This leads to a significant drop in SVR. Initially, increased metabolic activity and often an expansion of blood volume (due to effects on the renin-angiotensin-aldosterone system) can increase venous return to the heart (preload). The combination of reduced SVR and increased preload creates a high-volume, high-flow state that the heart must manage.[19,20]

This is a distinct entity from the more common low-output heart failure. In high-output failure, the cardiac output is normal or even elevated, yet it is insufficient to meet the extraordinarily high metabolic demands of the tissues, or the heart eventually succumbs to the sustained pressure and volume overload. The heart in thyroid storm is like an engine forced to run constantly at its maximum RPM; eventually, parts begin to fail despite the high revs. Young individuals might tolerate this hyperdynamic state for a period, but the relentless nature of thyroid storm provides no respite. The heart's workload is immense, characterized by increased stroke volume and heart rate. While a young, structurally normal heart might cope initially, the sustained demand, coupled with direct myocardial effects, eventually leads to fatigue and failure. The transition can be alarmingly swift. Thyroid storm has a profound proarrhythmic effect. Atrial fibrillation (AF) is exceedingly common, with prevalence much higher than in the general age-matched population. In young individuals, new-onset AF should always prompt consideration of hyperthyroidism. The onset of AF in this hyperadrenergic setting is often with a very rapid ventricular response (RVR). This rapid, irregular

rhythm has several detrimental effects. This can reduce ventricular filling and stroke volume by up to 20-30%, which is significant when the heart is already struggling. Extremely fast heart rates dramatically shorten diastole, the period when ventricles fill with blood and coronary arteries perfuse the myocardium. Inadequate filling leads to reduced stroke volume. The combination of massively increased demand (due to tachycardia and hypermetabolism) and reduced supply (due to shortened diastole) can lead to demand ischemia, even with normal coronary arteries. Prolonged, uncontrolled tachycardia itself can lead to a reversible form of left ventricular systolic dysfunction. In the acute setting of thyroid storm, this mechanism significantly compounds the thyrotoxic effects on the heart. Even before systolic function overtly fails, diastolic dysfunction is often present. The heart muscle becomes stiffer and less compliant, impairing its ability to relax and fill adequately during diastole. This is due to direct thyroid hormone effects on myocardial tissue properties and the relentless tachycardia. Consequently, left ventricular end-diastolic pressure (LVEDP) rises significantly for any given diastolic volume. This elevated LVEDP is a critical precursor to pulmonary congestion.

The development of acute cardiogenic pulmonary edema in this setting is a direct consequence of the failing heart, particularly left ventricular dysfunction (both systolic and diastolic). As the left ventricle struggles to cope with the volume and pressure demands, or as its ability to relax and fill is impaired, LVEDP rises. This pressure is transmitted backward to the left atrium, increasing left atrial pressure. The increased left atrial pressure impedes pulmonary venous drainage into the left atrium, leading to elevated pulmonary venous pressure and subsequently increased pulmonary capillary hydrostatic pressure (PCHP). According to Starling's principle, when PCHP exceeds the plasma oncotic pressure (primarily maintained by albumin) and the capacity of the pulmonary lymphatic system to drain excess interstitial fluid, fluid begins to leak from the capillaries. Initially, this occurs in the interstitial space (interstitial edema). As the process worsens, fluid floods the alveoli (alveolar edema). The onset can be extraordinarily rapid, often termed "flash" pulmonary edema, particularly in the setting of acute triggers like surgery or infection in a thyrotoxic patient. This rapid accumulation of alveolar fluid severely compromises gas exchange. This phenomenon is well-documented in cases of severe Graves' disease, even outside of a full storm, highlighting the thyroid's potent influence on cardiac function and fluid balance. In the case of a 23-year-old, as presented in the original report, a post-operative trigger in an undiagnosed thyrotoxic state led to this rapid decompensation. Pulmonary edema directly translates into respiratory failure through several mechanisms. The presence of fluid in the alveoli and interstitium creates a physical barrier, increasing the distance for oxygen and carbon dioxide to diffuse between the alveoli and pulmonary capillaries. Oxygen diffusion is more severely affected, leading to hypoxemia. Fluid-filled alveoli may still be perfused with blood but are not adequately ventilated, creating areas of intrapulmonary shunt (V/Q = 0). Other areas may be ventilated but poorly perfused due to hypoxic vasoconstriction or microvascular changes. This mismatch is a major cause of hypoxemia. The lungs become stiff and difficult to inflate due to interstitial and alveolar edema. This significantly increases the work of breathing. Patients struggle with dyspnea, tachypnea, and often use accessory respiratory muscles. Profound hypoxemia is the hallmark. Initially, patients may maintain normal or low $PaCO_2$ due to hyperventilation. However, as respiratory muscle fatigue sets in or the edema becomes overwhelming, hypercapnic respiratory failure can also develop.

The clinical picture is one of extreme respiratory distress: gasping for air, orthopnea (inability to breathe while lying flat), audible crackles or rales on lung auscultation, and often frothy, sometimes pink-tinged sputum. Oxygen saturation plummets, and without intervention, respiratory arrest is imminent. It might seem counterintuitive that young individuals, often perceived as having robust cardiovascular reserves, can succumb so dramatically. Several factors contribute to this vulnerability. Unlike older patients with chronic heart conditions who may have developed some degree of myocardial adaptation or collateral circulation over time, the young heart has not been "trained" or pre-

conditioned to withstand such an acute and overwhelming hemodynamic insult. The sudden, extreme demand can be catastrophic. Thyroid storm is not merely an exacerbation of hyperthyroidism; it is a quantum leap in severity. The concentration of thyroid hormones and the resultant sympathetic surge can be so extreme that they overwhelm even a structurally normal heart's compensatory capacities. While a patient may have "no known prior heart disease," this does not entirely exclude the possibility of subtle, subclinical cardiac abnormalities (e.g., minor congenital issues, early-stage non-ischemic cardiomyopathy, or genetic predispositions like channelopathies) that only become clinically apparent under conditions of extreme physiological stress like a thyroid storm. In young, otherwise active individuals, the early symptoms of hyperthyroidism (e.g., palpitations, weight loss despite increased appetite, anxiety, heat intolerance) might be insidious, ignored, or misattributed to stress, lifestyle, or other common ailments. This can lead to a significant delay in diagnosis, allowing the thyrotoxic state to progress untreated until a precipitating event (like infection, surgery—as in the 23-year-old case—or even severe emotional distress) unleashes the full fury of a storm on an unprepared system. The initial phase of thyrotoxicosis is often characterized by a hyperdynamic circulatory state (bounding pulse, warm peripheries). This can ironically mask the underlying strain on the heart. Clinicians might be falsely reassured by a high cardiac output, not realizing that the heart is teetering on the brink of failure until overt signs of pulmonary edema and shock manifest.

Comparing thyroid storm-induced cardiorespiratory failure to other critical conditions highlights its unique features. Versus Sepsis-Induced Cardiomyopathy: Both are life-threatening and can cause distributive shock and cardiac dysfunction. However, sepsis typically involves myocardial depression mediated by endotoxins and pro-inflammatory cytokines, often leading to a hypodynamic state after an initial hyperdynamic phase. In thyroid storm, the primary insult is the direct and indirect overstimulation by thyroid hormones, leading to a profoundly hypermetabolic and hyperadrenergic state that *causes* the heart to fail from overwork and altered cellular mechanics, rather than direct depression in the way sepsis does. Versus Pheochromocytoma Crisis: Both conditions involve massive catecholamine effects (direct in pheochromocytoma, predominantly sensitization and increased receptor density in thyroid storm, though thyroid hormones also increase catecholamine synthesis). Both can lead to severe hypertension (though thyroid storm can eventually lead to hypotension from vasodilation or cardiogenic shock), tachyarrhythmias, and pulmonary edema. Distinguishing them relies on clinical context and specific biochemical markers (metanephrines vs. thyroid hormones). Versus "Typical" Acute Decompensated Heart Failure (ADHF): ADHF usually occurs in patients with known pre-existing structural heart disease (e.g., ischemic cardiomyopathy, hypertensive heart disease, valvular disease). While flash pulmonary edema can occur, the underlying pathophysiology is often related to chronic cardiac remodeling and an acute trigger (e.g., dietary indiscretion, medication non-compliance, infection). In thyroid storm-induced pulmonary edema in a young person, the "culprit" is the endocrine crisis itself acting on a heart that may have been structurally normal prior to the storm, leading to a functional, acute cardiomyopathy. The reversibility of thyrotoxic cardiomyopathy, if the storm is survived and euthyroidism is achieved, is a key distinguishing feature. Clinicians should maintain a high index of suspicion for thyroid storm in patients presenting with unexplained severe multisystem illness, especially with cardiovascular and respiratory compromise. In patients fitting this profile who develop acute pulmonary edema, NIV should be considered early as a vital component of respiratory support.

4. Conclusion

Thyroid storm complicated by acute cardiogenic pulmonary edema and severe hypoxemic respiratory failure is a medical emergency demanding prompt recognition and aggressive, multifaceted management. This case report illustrates the successful application of non-invasive ventilation as a primary respiratory support modality in a 23-year-old female patient

experiencing this life-threatening triad. Early initiation of NIV, in conjunction with targeted medical therapy for thyroid storm and its cardiovascular sequelae, resulted in rapid clinical and physiological improvement, resolution of respiratory failure, and ultimately, avoidance of endotracheal intubation and its associated morbidities. This case contributes to the evidence supporting NIV as a valuable and effective therapeutic option in the critical care management of patients with respiratory failure secondary to thyroid storm-induced pulmonary edema, emphasizing the importance of its timely consideration in appropriately selected patients.

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