

A case of Thyroid Storm Complicated with Cardiorespiratory Failure Without Obvious Trigger: Early Suspicion is the Key

Abstract: Thyroid storm, also known as thyrotoxic crisis is an acute, rare life-threatening condition of thyroid gland; possessing 100% mortality when it's not treated in time. Thyroid storm coexisting with respiratory failure further can contribute to this mortality. Respiratory collapse can be irreversible. This is an endocrine emergency first described in 1926. This is important to recognize it early and treat to reduce mortality. Herein, we review a case of 75 years of male who developed Cardio-respiratory failure while he was manifesting clinical features thyroid storm.

Key Word: Thyroid storm, Thyrotoxicosis, Atrial Fibrillation, Cardio respiratory failure, Mechanical intubation

Introduction: Thyroid Storm is caused by rapid and uncontrolled release of thyroxines and considered as metabolic outburst with significant mortality that might come with a multisystem involvement. Multiple organs can be fatally affected in this cascade of this clinical entity. Thyroid storm coexisting with cardio-respiratory failure is rare [1]. The incidence of storm ranged from 0.57 to 0.76 cases per 100,000 per year in the normal population and 4.8 to 5.6 cases per 100,000 per year in hospitalized patients. We present a case who was in thyroid storm eventually developed respiratory failure requiring intubation

Case presentation: A 75 years of male who has past medical history of Dementia, Schizophrenia, Depression, Alcohol dependence, Hypertension, Coronary artery

disease, Peripheral vascular disease , Hyperlipidemia, Anemia, Chronic obstructive pulmonary disease, Peripheral neuropathy, Benign Prostatic Hyperplasia was brought to ED the complaints Failure to thrive with underlying protein calorie malnutrition. On examination, Pt appeared frail emaciated and cachectic, atraumatic, normo-cephalic. Respiratory effort and breath sound was normal with tachycardia. On arrival vitals as following: BP 160/98 Pulse 105, Saturation 98%, Respiratory rate 20.

Table 1 : Pathological test report

Test	Ref Range and Units	Values
WBC	4.5-11.0 10 ³ /uL	5.9
Hb	11.0-15.0 g/dL	11.3
MCV	80-100 fL	90.9
Platelets	130-400 10 ³ /uL	136
BUN	7.0-18.7 mg/dL	21.0
Creatinine	0.57-1.11 mg/dL	0.82
eGFR	>=90.0	91.0
Na	136-145 mmol/L	139
K	3.5-5.1 mmol/L	6
CO2	22-29 mmol/L	24
Anion gap	mmol/L	5.0

Total Bilirubin	0.2-1.2 mg/dL	1.2
ALT	10-55 U/L	15
AST	5-34 U/L	42
ALP	40-150 U/L	80.1
Albumin	3.5-5.2 g/dL	3.0
Phosphorous	2.3-4.7 mg/dl	3.6
Ca	8.4-10.2 mg/dL	9.7
BNP	10-100 pg/ml	233.2
Lactate	0.50-1.90 mmol/L	
High sensitivity troponin	0-35 ng/ml	24.8
PT	9.8-13.4 sec	15.1
INR	0.85-1.15	1.32
PTT	24.9-35.9 sec	24.5

In baseline EKG he was found having atrial fibrillation with rapid ventricular response with premature ventricular or aberrantly conducted complexes as well as Nonspecific ST and T wave abnormality.

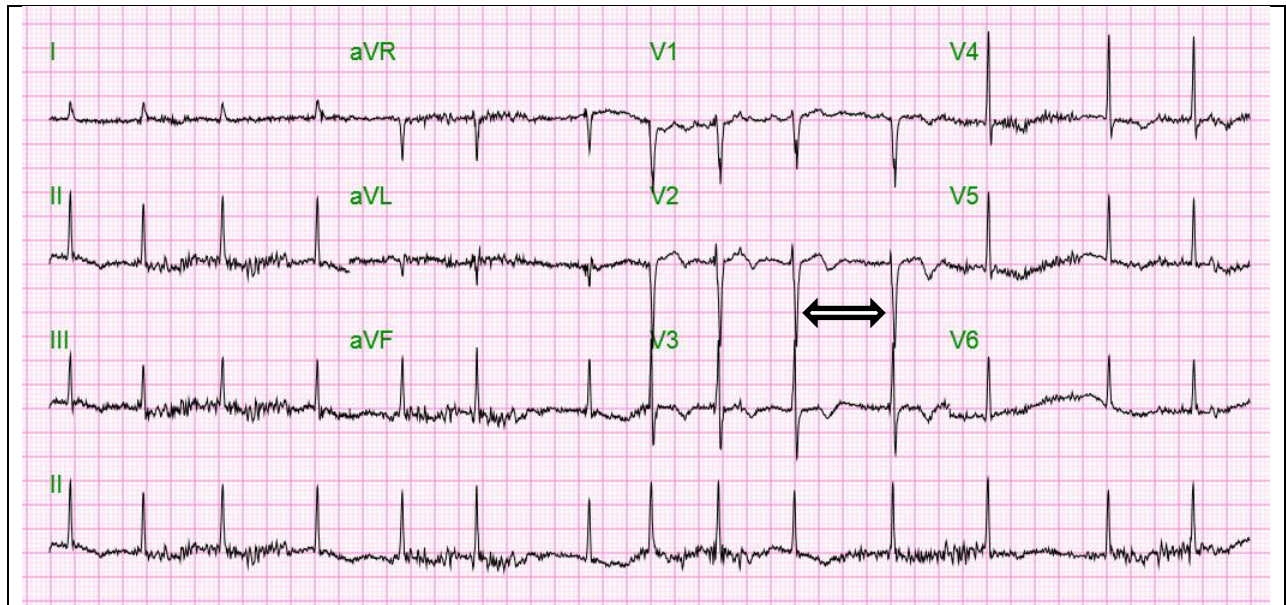


Figure 1: EKG Showing Atrial fibrillation (Arrow - irregular R-R) with no P wave

Chest x ray showed clear lungs with No pleural effusion.

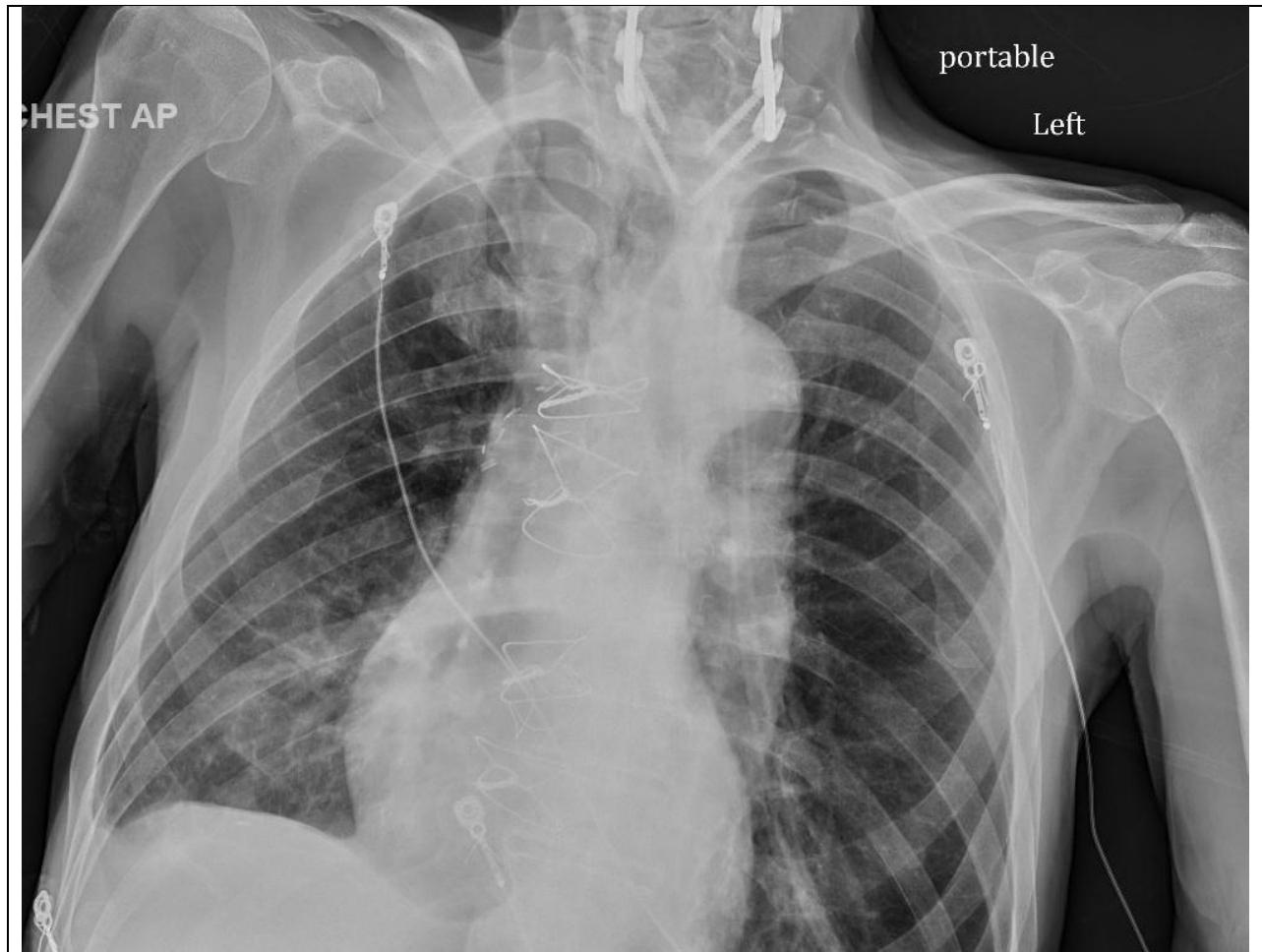


Figure 2 : Normal Chest X ray on admission

It was unknown till this admission whether this patient has any thyroid issue before. On admission T3 level 345 (Normal value 71 - 180 ng/dL), Free T4 was 5.24 (Normal value 0.78 - 2.19 ng/dL), Thyroid stimulating hormone (TSH) was < 0.015 (Normal value 0.465 - 4.680 IU/mL). Pt was diagnosed as Hyperthyroidism with possible thyroid storm. Patient was commenced on Propranolol, Hydrocortisone, Methimazole, Cholestyramine, Strong iodine 5 % oral solution 0.2 mL given an hour after Methimazole was given. Rate control medication was discontinued since Propanol was commenced as a part of the regimen treating Thyrototoxicosis.

On 2nd hospital day Patient started desaturating to 84% so he was placed on Nasal cannula, but saturation did not improve which is why he was placed on

Nonrebreather mask with 10 Liter O₂ . On exam: Patient was alert, awake, could not assess orientation as he was in acute distress. But he was able to follow commands. On exam: Excessive secretions in oropharyngeal area was appreciated but that improved with suction, Arterial Blood Gas result on NRB 100% showed pO₂ 61.4; pCO₂ 36.5; pH 7.4; HCO₃ 22.7, %O₂ Sat 90%.

Lasix 40 mg stat, Methyl prednisone 60 mg IVP and Xopenex nebulization was given.

The thyroid function was repeated.

Table 2: Thyroid test report

Test	Ref Range and Units	Values
TSH	0.465 - 4.680 IU/mL	<0.015
FT4	0.78 - 2.19 ng/dL	6.71
Thyroid peroxidase Antibody	0-34 IU/ml	57

On 3rd hospital day, Patient's ABG showed hypoxemia, gradual increment of all modes oxygen supplementation failed to improve his oxygenation. Patient continued to desaturate so that he was intubated and upgraded to ICU. Blood Pressure dropped suddenly, central venous access was obtained and Norepinephrine was started. His Cardio respiratory failure was attributed by Thyroid storm and Atrial fibrillation was concluded as secondary to overt hyperthyroidism which was likely remained undiagnosed prior to this admission. Post intubation hospital stay was eventful with aspiration pneumonia.



Figure 3 : Ventilator Setting of the Patient on 5th hospital day .

Chest X ray confirmed as bilateral pulmonary infiltrates predominantly in the lower lobes consistent with evolving pneumonias likely from aspiration with No effusion or pneumothorax.

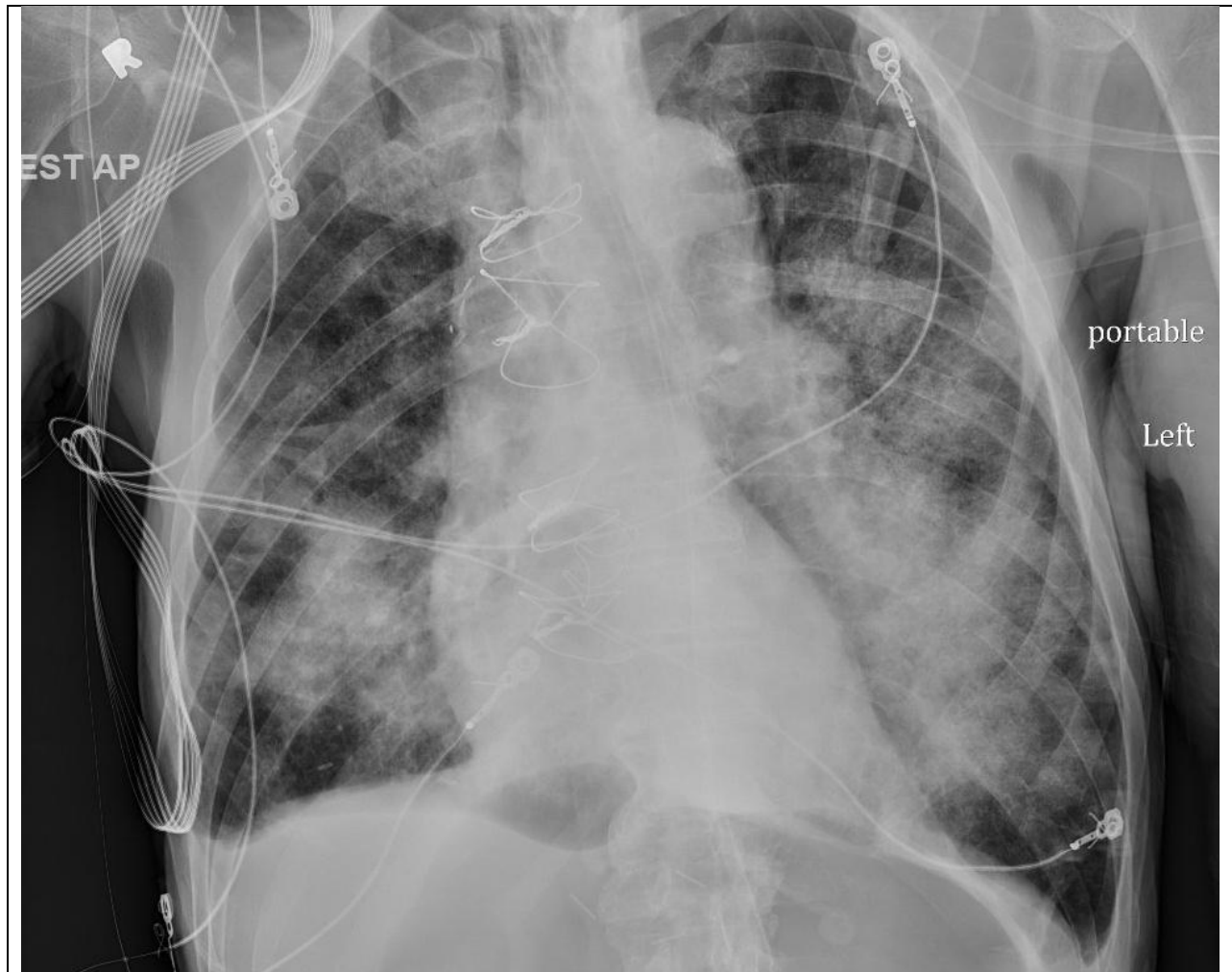


Figure 4: CXR showing Bilateral Pulmonary Infiltrates

Discussion: “Thyroid storm is a life-threatening condition and a medical emergency that usually presents in patients with a known hyperthyroidism disorder. Presentation of thyroid storm can be variable and with atypical symptoms including acute abdominal pain, seizures, heart failure, hepatic failure, and cerebral infarction” [1][2]. The patient manifested features of cardiorespiratory failure which is likely secondary to thyroid crisis. This crisis commonly may manifest with high fever and high heart rate but our patient had high heart rate given to his atrial fibrillation while temperature was normal. This

can be precipitated by recent surgery, infection ,an acute iodine load, ,parturition ,stroke ,traumatic brain injury , COVID-19 ,Acute myocardial infarction, diabetic ketoacidosis , Burns, medication side effects e.g amiodarone, anesthetics, salicylate but in the present case none of those were contributing [2][3]. Another common cause of thyroid storm is a hyperthyroid patient suddenly stopping their anti-thyroid drugs.

“If not managed at the time of presentation multi-organ dysfunction can follow which is one of the severe and life-threatening complications of the thyroid storm, which could lead to mortality” [3]. Multi-organ dysfunction should be considered as a big concern in any patient coming with a thyroid storm. This patient developed respiratory failure and needed intubation and still intubated. Usual treatment plan include beta blocker (if not contraindicated) to control the symptoms and signs induced by increased adrenergic tone, thionamide to block new hormone synthesis, An iodine solution to block the release of thyroid hormone , An iodinated radiocontrast agent to inhibit the peripheral conversion of T4 to T3, Glucocorticoids to reduce T4-to-T3 conversion, promote vasomotor stability, possibly reduce the autoimmune process in Graves' disease, and possibly treat an associated relative adrenal insufficiency, Bile acid sequestrates may also be of benefit in severe cases to decrease enterohepatic recycling of thyroid hormones [4]. We started all these medication after first lab check of Thyroid function test suggestive of overt hyperthyroidism. We preferred Methimazole in this case because it has longer duration of action than Propylthiouracil[6] . However, liver function should be monitored when patient is on Methimazole. When there is evidence of clinical improvement. Beta blockers can be withdrawn, but only after thyroid function tests have returned to normal. Iodine therapy can be discontinued (unless a thyroidectomy is planned in the next 10 to 14 days). Glucocorticoids are tapered and discontinued. The rate of the glucocorticoid taper depends upon the clinical course of the patient; a slower taper is necessary in patients who had a prolonged intensive care unit (ICU) stay with longer duration of glucocorticoid treatment. Some patients may need testing of adrenal function prior to discontinuing glucocorticoids. There are cases reported of thyroid storm

presenting with stroke which should be another monitoring parameter when patient presents with coma [5].

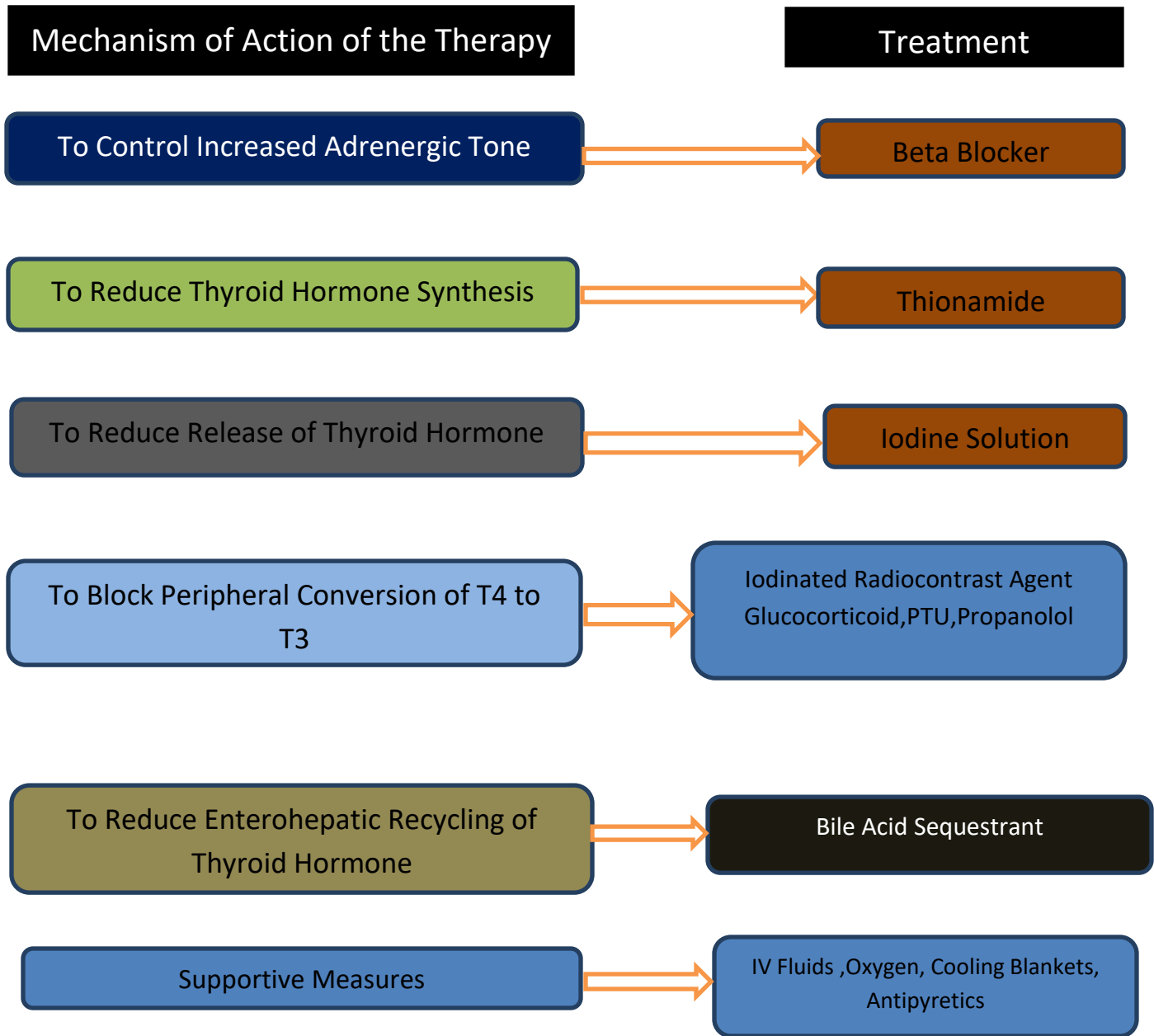


Table 3 : Specific Strategic Steps For Treatment of Thyroid Storm. Ref.[8][9]

Thyroid storm is usually diagnosed clinically while laboratory findings carry very minimal importance as they cannot distinguish with impending thyroid storm and uncomplicated thyrotoxicosis and could not provide a diagnosis. Full ICU support for management is needed and crucial given its high mortality rate. “Risk factors for poor prognosis include advanced age, having neurological issues upon admission to hospital, dialysis, and the need of mechanical ventilation” [7][5][6]. The patient was on mechanical ventilation due to thyroid storm driven respiratory failure till the case was being written. Patient failed multiple spontaneous breathing trial (SBT) and extubation trial.

Conclusion: At the end of this case report, we can conclude that respiratory failure is a rare outcome with poor prognostic value of thyroid storm and a clinician should take it in concern during management.

Ethical Approval:

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

Consent

As per international standard or university standard, patient(s) written consent has been collected and preserved by the author(s).

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