Original Research Paper

Volume - 11 | Issue - 07 | July - 2021 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Internal Medicine

UNEXPLAINED DELIRIUM : THINK OF THYROID STORM

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ABSTRACT **Background:** Thyroid storm is a life-threatening Endocrine emergency with an incidence rate of 1% to 2% all over the world. It is a systemic condition leading to increased production of Thyroid Hormone and its release leading to Thermoregulatory, Adrenergic, Neuropsychiatric, Cardiovascular, and Abdominal Manifestations. Thyroid storm with Malignant Arrhythmia and delirium both together is rare entity, but the mortality rate is very high. The presentation of Malignant Arrhythmias and delirium together in the initial phase of the disease is much less common with only a few isolated cases described in the scientific literature.

Objective: To present a case in which a patient had two simultaneous complication of thyroid storm i.e. delirium and ventricular tachycardia.

Case Study: We report a 65 years-year-old man who came with complaints of Diarrhea, Fever, Breathlessness and psychosis. His serum tsh was <0.015 and anti tpo antibodies was 83. He was diagnosed to be in Thyroid storm and later had complications including Ventricular Tachycardia and delirium in an undiagnosed case of Hyperthyroidism. He was started on anti thyroid medication and slowly as his condition improved he was discharged.

Conclusion: Patients with Thyrotoxicosis need to be closely monitored for complications since its early diagnosis and treatment may save lives.

KEYWORDS : thyroid storm, delirium, ventricular tachycardia, hyperthyroid

INTRODUCTION:

Thyrotoxic crisis also known as thyroid storm , is an acute, life-threatening complication of hyperthyroidism. It is an exaggerated presentation of thyrotoxicosis. It comes with sudden multisystem involvement with symptoms including fever . diarrhea, agitation and irrerular heartbeats. The mortality associated with thyroid storm is estimated to be 8-25% despite modern advancements in its treatment and supportive measures.[1] Thus, it is very important to recognize it early and start aggressive treatment to reduce mortality.

CASE PRESENTATION

Patient DP age 65 years was admitted with history of Fever with Bilateral Pedal edema and breathlessness since 15 days. Fever was of low grade, intermittent with no chills or rigors. It reduced with medication with no exacerbating factor or association with cold, cough, loose motions, burning micturation or rash. Pedal edema was of pitting type which has progressed over 15 days which was present more in evening. He complained of breathlessness which increased on lying down. No history of anginal or pleuritic chest pain ,palpitation, tremor. His appetite was normal. He was non-Hypertensive, non-Diabetic and had no history of Ischemic heart disease in past. No history of tuberculosis. He is a chronic Bidi smoker with no known allergies.

General Examination

Patient was conscious, oriented to time place and person and afebrile at the time of admission.Pulse -120/min irregularly irregular,Blood pressure-110/70mmHg of Mercury, Spo2-99% on room air. Respiratory rate-28/min, mild pallor, no lymphadenopathy, Jugular venous pressure – Raised , Rest general examination was normal.

Systemic Examination

Cardiovascular system- S1 variable,S2 heard, no murmur. No gallop sound. Respiratory system: bilateral basal crepts were heard. Per Abdomen - Soft, with palpable liver and horseshoe shaped dullness. CNS: Higher functions -Normal No neurodeficit.

Investigation

Chest x ray(PA):Right CP angle blunt with Cardiomegaly. ECG: S/O AF (fast ventricular rate),No ST T wave changes. 2D ECHO: Dilated Cardiomegaly with global Hypokinesia with ejection fraction about 40% with mild pulmonary hypertension.

USG abdomen and pelvis :showed presence of Hepatomegaly and mild to moderate ascites and presence of right sided pleural effusion.

Laboratory Investigation

On Admission

Parameters	Normal values	Result
Haemoglobin	12-16g/dl	9.6
Total Leucocyte count	3450-9060/cmm	7900
Platelets	165000-415000/cmm	102000
Urea	7-20mg/dl	21
Creatinine	0.5-1.2mg/dl	0.92
Sodium	136-146meq/L	132
Potassium	3.5-5.5meq/L	4.79
Total Bilirubin	0.3-1.3mg/dl	0.9
Direct	0.1-0.4 mg/dl	0.45
SGOT	12-38U/L	95
SGPT	7-41U/L	45

His Dengue NS1, IgM, IgG was Negative. COVID RTPCR was-Negative Peripheral smear for Malaria Parasite was Negative.

Considering dilated cardiomyopathy with atrial fibrillation with congestive cardiac failure patient was started on B blockers, Diltiazem, Diuretics, and Low molecular weight Heparin and ACE inhibitors. Patient improved with treatment in next two days ,dyspnoea decreased and heart rate controlled but sinus rhythm was not achieved. On 3rd day of admission, on examination patient had coarse postural tremors and some confusion initially. Later progressed to restlessness and agitation to frank hyperactive delirium. Encephalopathy was considered. To rule out any structural cause MRI was done. As MRI was normal, CSF was done which showed normal protein and sugars and no cells. He again had episode of fast ventricular rate and intermittent ventricular premature beat on ECG. The patient received amiodarone bolus followed by infusion. On the same day evening he had ventricular tachycardia which was reverted by cardioversion. TFT report we received suggestive of Hyperthyroidism. Considering clinical features and laboratory parameters patient was diagnosed to have Thyroid strom in long standing Undiagnosed Hyperthyroidism.

His Thyroid Function Test Was:

Parameters	Normal range	Values
Tsh	0.4-4.68	<0.015
Ft3	0.8-1.9	>22.1
Ft4	2.77-5.27	>6.99
Anti TPO Antibodies	<9	81.3

Clinically no sign of Thyroid Eye Disease was seen nor goitre. Ultrasonography of Thyroid- Bulky Thyroid with increased

Vascularity s/o Thyroiditis.

Management:
Patient received Tab Propylthiouracil (PTU) 600mg loading dose and was continued with 200mg QID . For rate control Tab propranolol and diltiazem was added. Inj hydrocort was given along with cholestyramine sachet. Patient was intubated electively in view of drowsiness. During his course due to deranged liver function tests Tab PTU was stopped and carbimazole was started and continued. He was extubated after 7 days after his condition improved.

His repeat labs (after 7 days)

Parameters	Normal range	Values
Tsh	0.4-4.68	<0.015
Ft3	0.8-1.9	4.48
Ft4	2.77-5.27	3.34

Patient improved significantly , became conscious oriented and the heart rate was controlled. Thyroid scan was done which revealed Graves disease. Patient was discharged on Tab Carbimazole 20 mg Tds along with management of Atrial Fibrillation and Congestive Cardiac failure.

Review Of Literature
Even though Thyroid storm is rare, it is a very life-threatening complication of Hyperthyroidism[2] The most common Etiology of Thyroid storm is Graves' disease, Solitary Toxic Adenoma or Toxic Multinodular goiter. Rare causes include Hyper secretory Thyroid Carcinoma, Struma 0varii, Thyrotropin secreting pituitary Tumor and administration Radiocontrast dye or Amiodarone which contains iodine.[3-4]

A precipitating event is usually identified that results in transition from Hyperthyroid state to Thyroid storm like Thyroid surgery during uncontrolled Hyperthyroidism, Infection, Myocardial Infarction, Pulmonary Thromboembolism, Parturition, Surgery, Trauma, Diabetic Ketoacidosis, withdrawal of Antithyroid drugs, and administration of Iodine via Intravenous Radiocontrast dye or amiodarone.[5]

Diagnosis of Thyroid storm is based on clinical presentation and laboratory values of elevated free T3 and free T4 with a suppressed TSH. Since the Clinical symptoms are vague and involve multiple systems, Burch and Wartofsky have created a point system to standardize and help make an objective diagnosis of thyrotoxicosis.(table1)

Criteria	Points
Thermoregulatory dysfunction	
Temperature (˚C)	
37.2–37.7	5
37.8–38.3	10
38.4–38.8	15
38.9–39.4	20
39.4–39.9	25
≥ 40.0	30
Cardiovascular	
Tachycardia (beats per minute)	
100–109	5
110–119	10
120–129	15
13–139	20
≥ 140	25
Atrial fibrillation	
Absent	0
Present	10
Congestive heart failure	
Absent	0
Mild	5
Moderate	10
Severe	20
Gastrointestinal-hepatic dysfunction	
Manifestation	
Absent	0
Moderate (diarrhea, abdominal pain, nausea/vomiting)	10
Severe (jaundice)	15
Central nervous system disturbance	
Manifestation	
Absent	0
Mild (agitation)	10
Moderate (delirium, psychosis, extreme lethargy)	20
Severe (seizure, come)	30
Precipitating event	
Status	
Absent	0
Present	10
Total score	
> 45	Thyroid storm
25–45	Impending storm
< 25	Storm unlikely

TOTAL SCORE	
>45	Thyroid storm
25-45	Impending storm
<25	unlikely

Our patient had a score of 75. Our patient also met the criteria of Thyroid storm (TS1) using the Japanese Thyroid Association criteria[6] as she had elevated Thyroid hormone levels along with Central Nervous System (CNS) Manifestation, Tachycardia, Gastrointestinal symptoms, and Fever.

Thyroid hormone mediates the expression of both structural and regulatory genes in the Cardiac Myocyte. Even though the main thyroid hormone produced by the Thyroid Gland is T4; it is five times less active than T3. The Cardiac cellular actions of Thyroid hormone are mediated by the binding of T3 to nuclear receptors. The subsequent binding of the T3-receptor complexes to DNA regulates the expression of genes, specifically those which control Cardiac Myocyte Contraction.[7-8] Stimulation of Adrenergic receptors by thyroid hormone causes an increase in the intracellular second messenger, CAMP, which increases heart rate. Despite these well-characterized mechanisms, it is not clear how Hyperthyroidism predisposes to Atrial Fibrillation and other potential Arrhythmias like VT.[9]

Hyperthyroidism modulates β-receptor density in the brain as well as their sensitivity to catecholamines; this augmentation of β-receptor-mediated adrenergic activity is thought to be responsible for neuropsychiatric symptoms.[10] Increased stimulation by TSH receptor antibodies in Graves' disease, leading to an excessive local production of T3 is also thought to contribute to psychiatric symptoms.

The standard Medical management of Thyroid storm includes drugs which block the production (Thionamides), release (Inorganic Iodine), and reduce the peripheral effects of Thyroid hormones (β-blockers and Steroids). Only exceptional cases refractory to this conventional treatment need other methods to remove excess Thyroid hormones like Plasmapheresis, Peritoneal Dialysis, Charcoal Hemoperfusion, and resin Hemoperfusion.

Conclusion: Due to severe mortality associated with thyroid storm , it is very important to diagnose and start aggressive treatment to reduce mortality.

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