

## Thyrotoxicosis Crisis as an Acute Clinical Presentation in a Child: One Case Report and Literature Review

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### Abstract

Thyrotoxic crisis also known as thyroid storm is an acute, life-threatening, hypermetabolic state in children, this is fortunately extremely rare but can be an initial presentation in previously undiagnosed children, Tabora region is located in central Tanzania, with limited resources. In the case presented, delayed diagnosis was made, hence some complications had ensued, ophthalmopathy and cardiomyopathy. This condition when rapid diagnosis is not made, mortality is high.

**Keywords:** Thyrotoxic Crisis; Hyperthyroidism; Hashitoxicosis; Graves' Disease; Thyrotoxicosis; Anti-Thyroid Treatment

### Abbreviations

AST: Aspartate Aminotransferase; ATD: Anti-Thyroid Drugs; BP: Blood Pressure; ECG: Echocardiogram; FBG: Fasting Blood Glucose; GALT: Gut-Associated Lymphoid Tissue; GD: Graves' Disease; SGOT: Serum Glutamic-Oxaloacetic Transaminase; SGPT: Serum Glutamic Pyruvic Transaminase; ICS: Intercostal Space; IOP: Intraocular Pressure; JVP: Jugular Venous Pressure; LVH: Left Ventricular Hypertrophy; OD: Once a Day; TRAb: Thyrotropin Receptor Antibodies; TSH: Thyroid-Stimulating Hormone

### Introduction

Thyrotoxicosis is the hyper metabolic clinical syndrome that occurs when there is elevated serum level of T3 and/or T4 irrespective of source of thyroid hormone [3]. This leads to a higher energy demand and tissue oxygen consumption, resulting in symptoms such as lethargy, hyperphagia, weight loss and heat intolerance [1]. Although it is rare in paediatrics rapid diagnosis is essential to ensure early treatment since delayment in treatment is associated with high mortality rate [8]. In severe cases the following are observed as complication of the disease ophthalmopathy and cardiomyopathy [2]. In the present study the complication were highly influenced with delayment in diagnosis and treatment which was a result of limited resources in Tabora region.

### Case Presentation

A 10-year-old boy presented with 8 weeks history of heart beat awareness chest pain, Protrusion of eyes with increased appetite, weight loss, tiredness, sleep difficulty, excessive sweating, and anterior neck swelling. He is a 10<sup>th</sup> child among twelve children of this pastoral family. No history of similar condition in the family.

On examination, the child had resting tremor, tachycardia, hypertension (BP 160/80 mmhg), warm peripheries, while the thyroid was diffusely enlarged to World Health Organization grade 2 had a soft texture on palpation but with no nodules and bilateral exophthalmos with no lid lag. No hepatomegaly, splenomegaly or abnormalities of the chest or abdomen were found.

He was review by assistant medical officer ophthalmology, Visual acuity -both right and left was 6/6 and an Intra-ocular pressure of 14/20 mmHg.

Laboratory studies revealed a normal hemoglobin concentration of 11.0 g/dL, hematocrit of 33.1%, a normal WBC  $5.5 \times 10^3$ /UL, platelet of  $228 \times 10^3$ /UL.

Thyroxine (Free T4) 244.23 nmol/L (66 - 1811 nmol/L), Total triiodothyronine pediatric (T3) > 9.22 nmol/l (1.3 - 3.1 nmol/L) Thyroid-stimulating hormone (TSH) < 0.1 mIU/L (0.5 - 5.0 mIU/L), FBG- 4.0 mmol/dl, (ALT) - 184.5U/L, reference < 40.0), AST - 224.2 U/L, reference < 40.0), Creatinine low 0.5 mg/dl (0.6- 1.4).

ECG (sinus tachycardia with left ventricular hypertrophy (LVH)) and hypertension.

Echocardiogram result - severe LVH.

Thyroid Ultrasound revealed heterogeneous enlarged thyroid gland with reduced echogenicity, right lobe 8.5 cm, LT lobe 8.2 cm, thymus is 0.85 cm, no focal mass seen, no neck adenopathy seen. Thyroid peroxides antibody or further radio-isotope studies were not done as they are not available.

The child was commenced on the antithyroid drug carbimazole (20 mg once daily) and beta-blocker propranolol (10 mg three times a day). After 7 days results was as follows - BP 130/79 mmhg, PR-88 b/m Thyroxine (T4) 135.62 nmol/L (66 - 1811 nmol/L) Total triiodothyronine pediatric (T3) 4.52 nmol/l (1.3 - 3.1 nmol/L) Thyroid-stimulating hormone (TSH) 0.29 mIU/L (0.5 - 5.0 mIU/L) current on a maintenance dose of carbimazole (5 mg OD). Propranolol 20 mg OD.

Due to expenses of the thyroid function test we didn't continue checking the levels instead we monitor the patient clinically.

Sign and symptoms were gradually improved as times goes; cardiovascular symptoms improved after day 4. Day 7 child was doing fine and being discharged, although the prognosis of the patient required an extensive follow up.



**Figure 1:** (A) Shows anterior neck swelling thyroid gland hypertrophy or goiter. (B) Shows Protrusion of eye or exophthalmos.

### Discussion

Thyrotoxicosis is the hyper metabolic clinical syndrome that occurs when there is elevated serum level of T3 and/or T4 irrespective of source of thyroid hormone [3]. This leads to a higher energy demand and tissue oxygen consumption, resulting in symptoms such as lethargy, hyperphagia, weight loss and heat intolerance [1]. Thyrotoxicosis is less common in children than in adults, affecting only 0.02% of all children. Recent work has estimated the incidence of thyrotoxicosis at 80/100,000 per year in women and 8/100,000 per year in men [4,5].

Hyperthyroidism is a condition where the thyroid gland produces and secretes inappropriately high amounts of thyroid hormone which can lead to thyrotoxicosis [6]. The prevalence of hyperthyroidism in the United States is 1.2% with overt hyperthyroidism accounting for 0.5% and subclinical hyperthyroidism accounting for 0.7% [6].

Graves' disease (GD) is the most common cause of thyrotoxicosis in patients younger than 18 years of age and accounts for 10 - 15% of all paediatric thyroid diseases. GD is rare under the age of 5 years; it has a peak incidence at age 10 - 15 years and more commonly affects female patients [5]. The transient toxic phase of chronic lymphocytic thyroiditis (Hashimoto's thyroiditis), also known as 'Hashitoxicosis', is also cited as a significant cause of thyrotoxicosis in children [4].

GD is an autoimmune condition that occurs with the loss of immunotolerance causing thyrotropin receptor antibodies (TRAb) to form, bind and subsequently stimulate the thyroid stimulating hormone (TSH) receptors. This causes increased thyroid hormone synthesis and secretion [6]. However, in our case the diagnosis was mostly based on T4, T3 and TSH the lack of TRAb diagnostic test which is cited to be more specific and has been shown to predict the risk for relapse, thus helping to guide future management of patient show drawbacks [1].

### Conclusion

One of the most severe complications of thyrotoxicosis is thyrotoxicosis crisis, also known as thyroid storm [7]. The clinical manifestations this life-threatening complication usually include acute onset, hyperthermia, severe tachycardia, and heart failure. Also, thyroid storm is associated with neurological manifestations such as emotional lability, restlessness, anxiety, agitation, confusion, and even seizure [7].

Anti-thyroid medications are commonly used as the first-line therapy in children [5] and on the case discussed above the patient responded well to antithyroid medication carbimazole. However, because of the low rates of spontaneous remission, most children eventually require permanent treatment with radioactive iodine or surgery [8]. Of the available antithyroid medications, current guidelines recommend use of methimazole and not propylthiouracil because of the unacceptable risk of hepatotoxicity associated with propylthiouracil [8].

### Conflict of Interest

We declare that there is no conflict of interest.

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