

## Beta thalassemia revealing hypothyroidism: A case report

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### Abstract

Thalassemia is a condition that affects hemoglobin synthesis and is one of the most Common hereditary illnesses in the world. Patients with thalassemia major require several blood transfusions. Multiple blood transfusions cause thyroid dysfunction, which leads to iron excess. We report the case of 35-year-old, followed in hematology for deep iron deficiency anemia who has benefited from a transfusion repeatedly diagnosed with beta thalassemia. At the interrogation, the patient reports a moderate gain of weight not quantified, hair loss and sensations of chills, chronic constipation, without other associated signs including no other signs of hypersecretion or anterior pituitary hyposecretion, no tumor SD. The physical examination finds a conscious patient, Normocardium, normal build, dysmorphic SD, palpable thyroid not increased in size. Microcytic hypochromic anemia, TSH at 10 mIU/l (0.35 -5.1) mIU / l, T4 at 10.6 pmol / l (10.6-19.4) pmol / l T3 to 2.88 pmol / l (3.8-8.4) pmol / l. Objective cervical ultrasound thyroid gland increased in size at the expense of the thyroid lobe with thyroid nodules classified EU-TIRADS 2 and 3. The patient was put on levothyrox 75ug/d. Thyroid disorders are common in  $\beta$ -thalassemia patients who have been transfused multiple times.

**Keywords:** Ferritin;  $\beta$ -thalassemia; Triiodothyronine (T3); Thyroxine (T4); Thyroid-stimulating hormone (TSH)

### 1. Introduction

Beta-thalassemia is a disease that affects the synthesis of hemoglobin, it is also a hereditary disease. Patients with beta-thalassemia major require multiple blood transfusions [1] causing thyroid dysfunction due to iron overload. It can be classified based on the symptoms it causes or the genes that are impacted. People of various ancestries, especially those from the Mediterranean and Arabian Peninsula, are more likely to have beta-thalassemia. Multiple blood transfusions are the Only way to treat anemia in beta-thalassemia, which causes iron excess and heart, liver, and bone damage [2]. Chelation treatment should reduce the amount of iron in the body, preventing fast clinical deterioration or possibly death. After absorbing iron from various regions of the small intestine, iron transporters bond with transferrin, which is subsequently stored in the reticuloendothelial cells of the spleen, liver, and bone marrow, where it binds with hemosiderin and ferritin

### 2. Case presentation

Patient aged 35-year-old, followed in hematology for deep iron deficiency anemia who has benefited from a transfusion repeatedly diagnosed with beta thalassemia. At the interrogation, the patient reports a moderate gain of weight not quantified, hair loss and sensations of chills, chronic constipation, without other associated signs including no other signs of hypersecretion or anterior pituitary hyposecretion, no tumor SD. The physical examination finds a conscious patient, Normocardium, normal build, dysmorphic syndrome, palpable thyroid not increased in size. Microcytic hypochromic anemia, TSH at 10 mIU/l (0.35 -5.1) mIU / l, T4 at 10.6 pmol / l (10.6-19.4) pmol / l T3 to 2.88 pmol / l

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(3.8-8.4) pmol / l. Objective cervical ultrasound thyroid gland increased in size at the expense of the thyroid lobe with thyroid nodules classified EU-TIRADS 2 and 3. The patient was put on levothyrox 75ug/d.



**Figure 1** Dysmorphic syndrome

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### 3. Discussion

Hypothyroidism is one of the most common endocrinopathy during beta-thalassemia major. Its prevalence is estimated between 6 and 30%. The course of thyroid dysfunction in the disease is variable, hypothyroidism may be reversible in the early phase subject to early intensive chelation<sup>[3]</sup>. On the other hand, replacement therapy is recommended if the FT4 level is low. In addition to the heart and liver, the endocrine glands are another target of post-transfusion iron overload resulting in elevated serum ferritin and hypothyroidism in beta-thalassemia patients. <sup>[4]</sup> Endocrine complications are particularly common in polytransfused beta thalassemia patients. A high level of TSH in beta-thalassemia patients, along with a modest rise in thyroid hormones, compared to healthy levels. It is caused by an increase in iron synthesis, which causes the hypothalamus to generate a lot of thyrotrophic-releasing hormone (TRH). It causes a rise in the pituitary gland's release of the hormone TSH, which aids in the reduction of thyroid hormones or survival at normal levels <sup>[5]</sup>. A mutation or deficiency in the gene responsible for hemoglobin manufacturing arises, especially in infants. Then it causes difficulty with hemoglobin production and red blood cell disintegration, resulting in severe anemia, which allows iron to collect in numerous tissues, glands, and organs. It enhances the toxicity of iron and can be removed by treating the patient with desferrioxamine, which is connected with excess iron and excreted from the body through the kidney when severe anemia occurs <sup>[6]</sup>. Finally, all nations with a high prevalence of thalassemia should fund preventive programs that include minor thalassemia diagnosis, genetic counseling, and bone marrow transplantation for children with significant beta-thalassemia <sup>[7]</sup>.

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### 4. Conclusion

Multiply transfused  $\beta$ -thalassemia patients are prone to metabolic and thyroid problems. Thyroid dysfunction during beta-thalassemia should be evaluated annually from the age of 9 years. It is important to diagnose and establish measures to prevent iron overload in patients with beta-thalassemia.

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### Compliance with ethical standards

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#### *Disclosure of conflict of interest*

The authors declare no conflict of interests.

*Statement of ethical approval*

The present research work does not contain any studies performed on animals/humans subjects by any of the authors'.

*Statement of informed consent*

Informed consent was obtained from all individual participants included in the study.

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