



Iron Concentration in the Anterior Pituitary causes Hormone Production

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Introduction

Thalassemia major is a genetic haemoglobin production condition that necessitates frequent blood transfusions. In these patients, a combination of chelation therapy and blood transfusion has significantly increased their life expectancy. Repeated blood transfusions, on the other hand, cause iron accumulation in various organs, including the liver, heart, and endocrine glands, resulting in a high incidence of endocrine dysfunctions in children, teenagers, and young adults. Iron homeostasis is mostly maintained by iron absorption rather than excretion in healthy people. Thalassemia patients who receive a blood transfusion (typically 1 mg of iron per 1 mL of blood) would undoubtedly have considerable iron overload due to a lack of appropriate excretory mechanisms. Non-Transferrin-Bound Iron (NTBI) is detected in excess in the blood of thalassemia individuals as a result of iron overload.

NTBI can potentially cause oxidative damage by acting as a catalyst for the production of reactive oxygen species. Gonadotropes require more iron than other pituitary cell types, according to studies of human anterior pituitary adenomas. As a result, Luteinizing Hormone (LH) and Follicle-Stimulating Hormone (FSH) synthesis declines in these cells (FSH). Hypo gonadotropic hypogonadism in thalassemia is linked to adipose tissue and leptin, as well as iron toxicity on gonadotrope cells. Leptin affects the Hypothalamic-Pituitary-Gonadal (HPG) axis in addition to carbohydrate and fat metabolism and appetite. To our knowledge, various investigations on leptin levels in different age groups of thalassemic patients have been undertaken, and all of them have found low leptin levels. The direct effect of iron, specifically NTBI, on the ovaries and testes is unknown at this time. Even in women who have amenorrhea, the ovarian reserve is preserved in the majority of female thalassemia patients.

Genetic Haemoglobin Production

Hypogonadotropic hypogonadism causes primary amenorrhea when the hypothalamus fails to emit enough Gonadotropin Releasing Hormone (GnRH) or when a pituitary disease causes insufficient production or release of pituitary gonadotropins. Hypopituitarism is a complication of thalassemia major. Hypogonadotropic hypogonadism, an endocrinopathy caused by iron excess, results from hypopituitarism. Iron overload occurs as a result of numerous blood transfusions, which are the most common treatment option for

thalassemia major. Liver problems, persistent hypoxia, diabetes mellitus, and zinc insufficiency are all probable causes of hypogonadism in beta thalassemia major. Increased iron concentration in the anterior pituitary causes hormone production to be disrupted, resulting in hypogonadism, short stature, and acquired hypothyroidism. We present a case of primary amenorrhea in a thalassemic patient to demonstrate that, while hyper transfusion and regular chelation therapy may have improved survival in patients with thalassemia major, growth failure and hypogonadism remain a significant clinical problem in these patients in adolescence, despite medical advances.

Although iron accumulation in the anterior pituitary gland can be seen as early as the first decade of life, clinical signs usually do not appear until puberty. Only a reduced gonadotropin reserve with an intact gonadotropin pulse was seen at an earlier stage. Pituitary siderosis might have an asymptomatic phase before hypogonadism develops. Later, the gonadotropin reserve decreases considerably, with much lower spontaneous pulsatile gonadotropin activity, perhaps leading to irreparable HPG axis injury.

Hypogonadotropic hypogonadism was linked to a variety of hematologic abnormalities. The majority of patients (86.4%) with the 0/0 hematologic phenotype developed hypogonadotropic hypogonadism, whereas only 25% of patients with the 0/+ phenotype acquired hypogonadotropic hypogonadism. During puberty, high serum ferritin levels of more than 2500 ng/mL were also discovered to be a risk factor for hypogonadism, with a 2.75 times increased incidence of hypogonadism compared to patients with serum ferritin levels less than 1000 ng/mL. Splenectomized people with serum ferritin levels less than 2500 ng/mL, on the other hand, had a significant risk of endocrine problems.

Pulsatile Gonadotropin Activity

Delayed puberty, stopped puberty, and hypogonadism are the three main clinical manifestations of the HPG axis disruption in thalassemia major. Delayed puberty is defined as the absence of pubertal symptoms in boys before the age of 14 and in girls before the age of 13. Arrested puberty is defined as the lack of additional pubertal growth once puberty has begun for more than a year. In well-chelated and well-transfused hypogonadism individuals, spontaneous fertility is possible, while others with hypo gonadotropic hypogonadism may require assisted reproductive procedures. Exogenous gonadotropin treatment can stimulate ovulation or spermatogenesis in patients with hypo gonadotropic hypogonadism since gonadal function is usually intact. Hypothyroidism and diabetes mellitus can potentially affect the outcome of reproductive therapies, necessitating specialized care.

Gonadal iron deposition causes primary gonadal failure. Secondary hypogonadism is caused by iron deposition on pituitary gonadotropic cells, as evidenced by a poor response of LH and FSH to GnRH stimulation, or a combination of primary and secondary hypogonadism. According to some research, the failure rate of puberty onset might be as high as 50%, and it can even be as high as 100%. Leptin is a polypeptide hormone produced by fat cells that acts as a puberty-initiating signal. Iron toxicity on adipose tissue has been linked to a decrease in leptin production, which slows sexual development. Menarche is delayed; oligomenorrhea, secondary amenorrhea, reduced testicular size, and breast size are all symptoms

of low gonadotropin levels. These symptoms are most common in significantly elevated serum iron and ferritin levels.