



An Interesting Case of Short Stature

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Abstract

Hypothyroidism is very common problem in developing part of the world. Hypothyroidism in children can produce various skeletal manifestations one of them is short stature and it can also cause delayed puberty. This is case report of 15 years female patient who presented with short stature, investigations revealed primary hypothyroidism, hyperprolactinemia and pituitary enlargement. Complete evaluation and treatment showed primary hypothyroidism was the cause for pituitary enlargement.

Keywords: Short stature, Delayed puberty, Hypothyroidism, Hyperprolactinemia, Pituitary enlargement.

Introduction

Hypothyroidism is very common problem in developing part of the world. Hypothyroidism in children can produce various skeletal manifestations one of them is short stature and it can also cause delayed puberty. It needs special attention as the treatment with thyroxine replacement can significantly improve the outcome.

Case Report

Miss surenya 15 years female patient presented with complaints of easy fatiguability, failure to gain height, decreased physical activity for past 5 years. She has not attained menarche and there is develop secondary sexual characters. No h/o loss of weight, no history suggestive of malabsorption, cardiovascular, renal disorders, liver cell failure and bony deformity. Past history revealed that she was diagnosed to have hypothyroidism at the age of 10 years but she was not on any treatment. Antenatal and birth was nil significant. She had normal developmental milestones. Now she is studying 10th standard and her school performance is good.



Examination

General examination showed mild pallor, dry skin, no skeletal deformity. There was no development of secondary sexual characters Tanner staging- stage 1. Vitals and systemic examination was normal. Anthropometry - height 117 cm which was less than 3rd centile, height age was 9 years, weight for height was normal, upper segment/lower segment ratio was 0.9.

Investigations

CBC showed Hb of 8.6 gm/dl . peripheral smear study was suggestive of dimorphic anemia. Renal and liver function tests, lipid profile, urine and stool routine, chest x ray, ECG were normal. USG abdomen showed rudimentary uterus and normal ovaries. Bone age of the patient was < 12 years. In skeletal survey there was no skeletal dysplasia. HORMONAL ASSAYS FT3 – 0.41ng/dl (2 – 4.4), FT4- 0.10µg/dl (0.93 – 1.7), TSH >100IU/L . FSH,LH were below normal. GH stimulative test was not performed as patient had hypothyroidism . ACTH and serum cortisol was normal. Serum ANA was negative. **Anti TPO antibodies** were elevated 193 IU/ml. USG NECK showed altered echotexture of both lobes of thyroid. FNAC OF THYROID- scattered follicular cells. MRI BRAIN showed pituitary enlargement with homogenous enhancement with contrast, features were suggestive of pituitary hyperplasia related to hypothyroidism.

KARYOTYPING -NORMAL .Xray Forearm with wrist joint



MRI BRAIN showing pituitary hyperplasia



Diagnosis- Primary Hypothyroidism with Secondary Hyperprolactinemia.

Pituitary Hyperplasia Related to Hypothyroidism

TREATMENT – T levothyroxine 50µg OD, T FST 1 BD, T BCT 1 OD.

FOLLOW UP- Patient is under regular follow up. Height of the patient significantly increased. She has attained menarche. Repeat MRI BRAIN has been planned after 6 months.

Discussion

Short stature is defined as height less than 3rd centile or below 2 standard deviation for that corresponding age in a population. Causes may be systemic illnesses like renal failure, liver failure, malabsorption, endocrine causes like hypothyroidism, hypoparathyroidism, Cushing's disease, rickets, growth hormone deficiency and idiopathic short stature. Hypothyroidism in children can have varied presentation like short stature, spondylolisthesis, delayed bone age, and irregular ossification of epiphysis. Prompt recognition of findings can lead to easy and effective treatment.

In this type of pituitary hyperplasia, the thyrotropes cells become enlarged by the lack of negative feedback from thyroid gland. The increase in TRH also stimulates lactotrophes leading to secondary hyperprolactinemia. Treatment with thyroid hormone normalizes the serum prolactin and TSH levels. Growth hormone production also decreases in patients with hypothyroidism as the stimulatory effect of thyroxine is lost. Incidence of pituitary hyperplasia in patients with hypothyroidism varies from 25 to 81%. Secondary hyperprolactinemia occurs in three fourths of patients. Rarely patients present with neurological symptoms secondary to sellar expansion. Their complaints at presentation include that of hypothyroidism as occurred in our case. In literature it has been suggested that the presence of a pituitary mass in MRI BRAIN with markedly elevated TSH, with absence of clinical features of hyperthyroidism and low thyroid hormones, one should suspect pituitary enlargement is due to primary hypothyroidism. This illustrates the importance of doing thyroid function tests in patients with pituitary mass and this avoids unnecessary surgical intervention. This hyperplasia of pituitary regresses after treatment with thyroxine.

In our case patient had short stature, delayed bone age, and delayed menarche. Short stature can be due to the following mechanisms occurring due to hypothyroidism

1. Impaired general protein synthesis,
2. Growth hormone is not directly regulated by thyroid hormone in humans, but thyroid status influences the growth hormone axis. Hypothyroid children have delayed growth and the response of growth hormone to provocative stimuli may be subnormal.
3. Reduced insulin-like growth factor 1
4. Delay in development of epiphyseal ossification centres.

Delayed puberty can be explained by the following mechanisms

1. Hypothyroidism affects GH-IGF axis. GH and IGF are essential for pubertal breast development.
2. Hyperprolactinemia occurring before puberty has led to primary amenorrhoea. High prolactin affects the release of FSH and LH leading to hypogonadism and delayed puberty.

Conclusion

In hypothyroidism occurring in a child clinical features are intermediate between those of cretinism and adult hypothyroidism in that developmental retardation is not as severe as in cretinism and the manifestations of full blown adult myxedema are rarely seen. Growth and sexual development are affected predominantly. If left untreated, linear growth is severely retarded and sexual maturation and onset of puberty are delayed. So early detection and treatment can have good prognosis in such patients.

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