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Case Report

Hypothyroidism presenting with development delay, failure to thrive, and pituitary adenoma

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ABSTRACT

Pituitary enlargement secondary to primary hypothyroidism (PH) is a known but uncommon occurrence, and is also difficult to distinguish on CT and MRI from primary pituitary tumors. Following adequate hormone replacement with L-thyroxine, both symptoms and pituitary hyperplasia are reported to regress within a few months. It is important to recognize this condition so as to avoid unnecessary surgery.

Keywords: Congenital hypothyroidism, Pituitary adenoma

INTRODUCTION

The endocrine system is a complex group of organs and glands that relates to multiple other organs and systems in the body with the ultimate goal of maintaining homeostasis. This complex network functions through hormones excreted by several glands and released in the blood, targeting different body tissues and modulating their function. Any primary disorders affecting the endocrine glands and altering the amounts of hormones synthesized and released will lead to disruption in the functions of multiple organs. The central nervous system of a developing child is particularly sensitive to endocrine disorders. A variety of neurologic manifestations have been described as features of several endocrine diseases in childhood.[1]

For example, newborn infants with congenital hypothyroidism frequently have hyperbilirubinemia, and delayed skeletal maturation, reflecting immaturity of liver and bone, respectively, and they are at risk of permanent mental retardation if thyroid hormone therapy is delayed or inadequate; their size at birth, however, is normal. In contrast, hypothyroidism that develops after the age of three years (when most thyroid hormone-dependent brain development is complete) is characterized predominantly by a deceleration in linear growth and skeletal maturation but there is no permanent effect on cognitive development.

In the last several decades, there have been exciting advances in our understanding of fetal and neonatal thyroid physiology, and screening for congenital hypothyroidism has enabled the virtual eradication of the devastating effects of mental retardation due to sporadic congenital hypothyroidism in most developed countries of the world.[2]

CASE REPORT

A 6month-old girl was referred for developmental delay and failure to thrive. She was also suffering from poor weight and height gain since birth. She was born at full term, her birth weight was 2.5 kg, and she had delayed development milestone performance, constipation. There was no history of intake of any medication during

pregnancy except iron and folic acid, no ho abortion no ho hypothyroidism in mother.

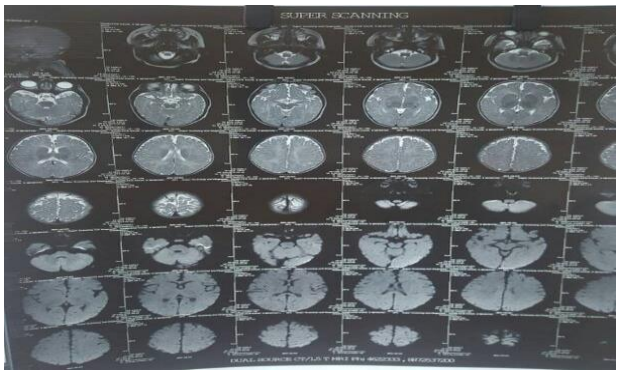

Figure 1: MRI brain scan 2.

Her length was 58cm (5th centile target height), her weight was 6.1 kg, and she had normal body proportions. Her pulse rate was 120/min and blood pressure 84/52 mmHg. She had pallor, dry scaly skin and cold extremities. There was a grade1 goiter. External genitalia were unambiguously female.

She had normocytic normochromic anemia, hemoglobin 8.8g/dl (normal 12-14 g/dl). TSH was 1259 µIU/ml (normal 0.35-6.4), T 3 0.50 pg/ml (normal 2.8-4.8), T 4 0.12 ng/ml (normal 0.8-2). The anti-TPO antibody level was IU/ml (normal IU/ml) and antithyroglobulin level (normal <4.17 IU/ml). Growth hormone was 1.41mg/mL (normal). GF binding protein-3 (IGFBP-3) was 1.22 g/Ml (normal). ACTH was 23.40 pg/mL(normal). Cortisol levels in the serum was 7.34 g/dl (normal). Prolactin µg/l (normal).

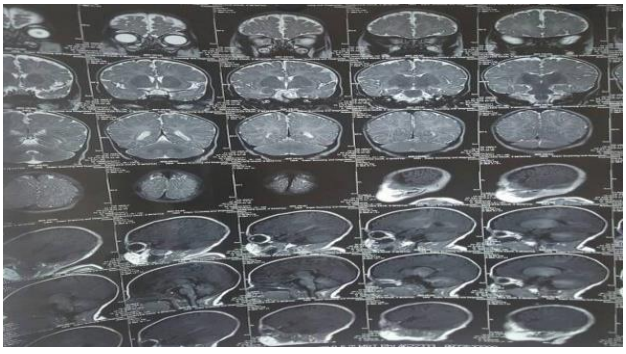

Figure 2: MRI brain showing pituitary adenoma scan 1.

Radiological investigations revealed delayed bone age (Greulich and Pyle's Atlas More Details). Ultrasonography of the thyroid was normal.

MRI brain measuring approximately 12.2(H) x12.5(Tr)x7.1(AP) mm, it is abutting the optic chiasm with no significant compression. No invasion into cavernous sinus seen Magnetic. Resource Imaging scan of sella revealed a sellar mass of 12.2 × 12.5 × 7.1 mm size.

Technetium 99 m thyroid scan showed poor and patchy uptake of radiotracer suggestive of thyroiditis.

A diagnosis of hypothyroidism due to autoimmune thyroiditis was made and she was started on a levothyroxine in a dose of 100 µg daily. Dose was titrated and euthyroid status was established. At 6-month followed up showed improvement in milestone, weight gain. MRI could not be repeated due to financial constraints.

Enlarged pituitary in our case was probably because of thyrotroph hyperplasia due to an end organ deficiency. Enlargement of the pituitary gland or pituitary adenoma have been described in hypothyroidism and as seen in our case some of these patients also had ovarian enlargement and multiple ovarian cysts.

DISCUSSION

With long-standing hypothyroidism, thyrotroph hyperplasia can result in the expansion of the sella turcica and the enlargement of the pituitary gland.[3] Khawaja, et al report that pituitary enlargement on MRI is found in 70% patients with primary hypothyrodism.[4] The pituitary mass may extend outside the sella turcica and produce clinical symptoms.[5] Radiana et al suggest that the greatly increased number of TSH-cells in methimazole-induced-hypothyroidism is due, at least partially, to the transdifferentiation of somatotroph into thyrotroph cells and a role for TRH stimulation in the transdifferentiation process.[6] Key transcription factors, such as Pit-1 and Gata 2 are known to be involved in pituitary endocrine cell differentiation.[7] Depending on demand, somatotrophs can reversibly transform into thyrotrophs.

In adults, pituitary hyperplasia with hypothyroidism generally exhibit in various forms like features of hypothyroidism, amenorrhea; galactorrhea; visual abnormalities and headaches.[8] While short stature or decreasing growth is a frequent reason for pediatric consultations, clinical features of hypothyroidism are subtle and missed. Hashimoto's thyroiditis was the likely main cause in our patients; because antithyroid antibodies were positive and radionuclide thyroid scans showed an asymmetrical thyroid gland with irregular distribution of 99mTc.[9] The other reason was congenital hypothyroidism, which was caused by thyroid hypoplasia or dyshormonogenesis. The recommended and appropriate replacement therapy for hypothyroidism is levothyroxine sodium. Since thyroxine is a stimulating factor for GH synthesis, GH production may be reduced in hypothyroic children. This is a very important phase of their height increase in prepuberty; so, after the complete disappearance of enlargement in the pituitary, rhGH was given to these 2 patients. The combination levothyroxine

with GH can be highly effective in increasing final height.[10]

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REFERENCES

1. Moltz KC, Postellon DC. Congenital hypothyroidism and mental development. Compr Ther. 1994;20(6):342-6.
2. Van Vliet G, Deladoëy J. Disorder of the thyroid in the newborn and infant. In: Sperling MA, ed. Pediatric Endocrinology. 4th ed. Philadelphia, PA: Elsevier Saunders; 2014:7.
3. Ashley WW, Ojemann JG, Park TS, Wippold FJ. Primary hypothyroidism in a 12-year-old girl with a suprasellar pituitary mass: rapid regression after thyroid replacement therapy: case report. J Neurosurg. 2005;102:413-6.
4. Khawaja NM, Taher BM, Barham ME, Naser AA, Hadidy AM, Ahmad AT. Pituitary enlargement in patients with primary hypothyroidism. Endocr Pract 2006;12:29-34.
5. Radiana S, Coculescua M, Morriss JF. Somatotroph to thyrotroph cell transdifferentiation during experimental hypothyroidism- a light and electron-microscopy study. J Cell Mol Med. 2003;7:297-306.
6. Joshi AS, Woolf PD. Pituitary hyperplasia secondary to primary hypothyroidism: a case report and review of the literature. Pituitary. 2005;8:99-103.
7. Alkhani AM, Cusimano M, Kovacs K, Bilbao JM, Horvath E, Singer W. Cytology of pituitary thyrotroph hyperplasia in protracted primary hypothyroidism. Pituitary. 1999;1:291-5.
8. Nicholas WC, Russell WF. Primary hypothyroidism presenting as a pituitary mass. J Miss State Med Assoc. 2000;41:511-4.
9. Hopper NW, Albanese A. Primary hypothyroidism in a child mimicking a pituitary macroadenoma. Horm Res. 2005;63:61-4.
10. Papakonstantinou O, Bitsori M, Mamoulakis D, Bakantaki A, Papadaki E, Gourtsoyiannis N. MR imaging of pituitary hyperplasia in a child with growth arrest and primary hypothyroidism. Eur Radiol. 2000;10:516-8.

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