

An Apparently Healthy Girl with Poor School Performance – A Case Report of Hypothyroidism

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Hypothyroidism is caused by a deficiency in the secretion of thyroid hormones produced in the thyroid gland. Because thyroid hormone is essential for normal growth and development, adequate metabolism, and proper brain development, the consequences of hypothyroidism in childhood can be devastating. A delay of treatment in this group can result in permanent brain damage and mental retardation. There are two major forms of hypothyroidism in the pediatric population: (1) congenital hypothyroidism, which represents a group of diseases developing at conception or during gestation and are present at birth; and (2) acquired hypothyroidism, which usually appears after 6 months of age and arises from autoimmune destruction of the thyroid. The management of hypothyroidism in an infant or young child poses challenges distinct from those of adult hypothyroidism because of the need to diagnose and treat affected children as quickly as possible. So it is critical that the physician should be aware of the possibility of hypothyroidism when seeing a child who fails to grow and/or shows an unexplained decline in neurodevelopment.

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Introduction

Hypothyroidism is the most common disturbance of thyroid function in children; acquired hypothyroidism is most often caused by autoimmune thyroiditis.¹ As in adults, acquired hypothyroidism can be caused by both thyroid disease (primary hypothyroidism) and hypothalamic-pituitary disease (central

hypothyroidism); whatever its cause, hypothyroidism in children can have deleterious effects on growth, pubertal development and school performance.² This case highlights this common finding, discusses clinical situations in which gradual dose escalation of levothyroxine and periodic follow up for better clinical outcome.

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Case Report

An 11-year-old girl, 1st issue of her non consanguineous parents born by normal vaginal delivery at term with normal birth weight, brought to Endocrinology OPD, BSMMU for evaluation of short stature, weight gain and gradual deterioration of school performance. Her developmental milestones were normal. No history suggestive of congenital hypothyroidism. History of facial puffiness, cold intolerance and constipation since nine year of age. No history of taking psychotropic medications (e.g. lithium carbonate, interferon gamma). No family history of short stature. On examination, Height 114 cm (<3rd percentile), Mid parental height 142 cm \pm 5cm, weight 38 kg, BMI 29.24 kg/m² (>75 percentile), upper segment (US) 64 cm, lower segment (LS) 50 cm, US:LS ratio 1.28 (disproportionate short stature), arm span 104 cm. puffy face, pale complexion, coarse, thick, dry skin, thyroid gland – palpable, delayed relaxation of reflexes present, no development of secondary sexual characteristic. Investigations reveal Hb-10.4g/dl, FT₄ - 0.3 ng/dl (0.8-1.8), TSH >75 μ IU/ml (0.4-4.0), Anti-TPO antibody >1000 IU/ml (up to 35 IU/ml), Radiological bone age : Corresponds to 6 to 9 years, Ultrasonography of thyroid: Both lobes of thyroid gland are slightly thinner than normal limit with hypo echoic parenchymal echo texture (RL= 8mm and LL= 8.8mm in anterior-posterior diameter), Thyroid scan: Thyroid gland is normal in size with uniform radio-tracer distribution. Considering history, physical examination and investigations this patient was diagnosed as a case of short stature and poor school performance due to childhood acquired primary hypothyroidism (Hashimoto's thyroiditis).

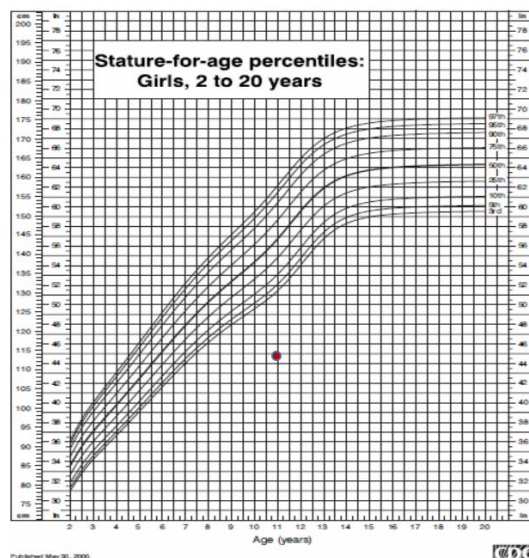


Fig -1. Growth chart: Before treatment

Treatment: Tab. thyroxine 50 μ gm daily for 1 week, then 100 μ gm daily continued, Follow up visit after one month with investigation TSH and FT₄. **1st Follow up after one month:** Improvement of general wellbeing, clinically euthyroid, body wt: 30 kg (38 kg previously), height: 114 cm, No secondary sexual characteristics, TSH: 0.42 μ IU/ml (0.4-4.0), FT₄:1.78 ngm/dl (0.8-1.8), Treatment: tab. thyroxine (75 μ gm) daily. **2nd Follow up after 3 months:** Improvement of general wellbeing, clinically euthyroid, Body wt: 25 kg (30 kg previously), height: 118 cm (114 cm previously) no secondary sexual characteristics, TSH:10.57 μ IU/ml, FT₄:1.17 ng/dl, tab. thyroxine (100 μ gm), advice for F/U after 6 months with TSH & FT₄ report. **3rd Follow up after 6 months:** Improvement of general wellbeing, clinically euthyroid, body wt: 27 kg (25 kg previously), height: 127 cm (118 cm previously), breast development: Tanner stage 2, no axillary or pubic hair, TSH: 1.7 μ IU/ml, FT₄ 1.5 ng/dl, tab. thyroxine 100 μ gm & 125 μ gm every alternate day, F/U after 6 months with TSH & FT₄ report.



Fig-2. Before treatment Fig-3: After treatment

Discussion

The most common manifestation of hypothyroidism in children is declining growth velocity, often resulting in short stature.³ The growth delay tends to be insidious in onset, and it may be present for several years before other symptoms occur, if they occur at all.⁴ Thus, any child with declining growth velocity should be evaluated for hypothyroidism.

Another common feature is altered school performance.⁵ Performance often declines, but it improves in some children, perhaps because they are less active and, therefore, less easily distracted and better able to concentrate. One reason for delay in diagnosis is that parents see the latter changes as positive.

Other common symptoms are sluggishness, lethargy, cold intolerance, constipation, dry skin, brittle hair, facial puffiness, delayed puberty and muscle aches and pains.⁶ If the cause is hypothalamic or pituitary disease, the child may have headaches, visual symptoms, or manifestations of other pituitary hormone deficiencies.⁷

The most common physical finding at presentation is a diffusely enlarged thyroid gland (goiter). In one study showed that, a noticeable goiter was present in 39.5 percent of children with autoimmune thyroiditis.⁸ Alternatively, the thyroid gland may be normal in size or not palpable at all. Abnormalities on physical examination include short stature, apparent overweight (more fluid retention than obesity), puffy facies with a dull, placid expression, bradycardia, and delayed deep tendon reflexes.

Treatment of acquired hypothyroidism in children involves oral L-T₄ taken in a single daily dose, determined by the child's size. The recommended daily dosage by age 4 $\mu\text{g}/\text{kg}$ from 6 to 10 years, and 2 to 3 $\mu\text{g}/\text{kg}$ from 11 to 20 years.⁷ The optimal maintenance dosage should normalize levels of serum TSH and maintain serum FT₄ or total T₄ levels in the middle or upper range of normal for age. Excessive dosages should be avoided to prevent accelerated skeletal maturation and consequently compromised final adult height and hyperthyroidism as well.

Conclusion

Any child presenting with poor school performance needs exclusion of hypothyroidism. Thyroxine supplementation to normalize thyroid function is essential to improve growth and intelligence.

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