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BRIEF COMMUNICATION

Two cases describing the effects of hypothyroidism on puberty and growth

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Primary hypothyroidism is common in girls before and during adolescence, and those affected present with a short stature and delayed puberty; however, a long-standing hypothyroidism can cause precocious sexual development [1]. Central precocious puberty (CPP), a result of premature activation of the hypothalamus-pituitary-adrenal axis, is accompanied by rapid height gain but affects adult height because of early epiphyseal fusion. And when CCP is concomitant with thyroid hormone deficiency, growth spurts are especially impaired. Replacement therapy with levothyroxine reverses the signs of precocious puberty in girls with this condition, and permits good catch-up growth. We report on 2 cases of primary hypothyroidism with significant effect on pubertal progression and statural growth.

A girl of short stature aged 11 years and 3 months presented with irregular vaginal bleeding of a 2-month duration. The remainder of her developmental history, including her birth and successive milestones, was normal. She denied development of other sexual characteristics such as breast enlargement or pubarche. Her height was 117 cm (<3rd percentile) and weight 25 kg (10th percentile). A pubertal assessment revealed the following development: breast, stage B_2; axillary hair, A_1; and pubic hair, P_1 (Tanner staging). On physical examination she had dry skin, no goiter, and delayed relaxation of deep-tendon reflexes. A hand X-ray revealed a bone age of 7 years. A thyroid function test revealed serum concentrations of total triiodothyronine (T3) of 0.36 ng/mL (normal range, 0.86–1.8 ng/mL), total thyroxine (T4) of 1.04 μg/dL (normal range, 4.5–12.0 μg/dL), and thyroid stimulating hormone (TSH) greater than 150 mIU/L (normal range, 0.2–5.0 mIU/L). Her levels of luteinizing hormone, follicle stimulating hormone (FSH), and prolactin were within prepubertal range. On ultrasound the ovaries were normal and the uterus was of prepubertal-size. The patient was treated with levothyroxine. Vaginal bleeding stopped and at a follow-up visit 4 months later she had gained 4 cm in height.

A girl of short stature aged 9 years and 6 months presented with pubertal features that had progressed for 2 years. Her mother noticed breast development and then pubarche, and menarche had occurred the previous month. Her parents noticed no growth spurts in the past 2 to 3 years. The remainder of her developmental history, including her birth and successive milestones, was normal. Her height was 126 cm (10th–25th percentile); her weight was 34 kg (75th percentile); and her upper segment to lower segment ratio (ie, the distance from the top of the head to the pubic bone divided by the distance from the pubic bone to the soles of the feet) was 1.1. Pubertal assessment revealed the following development: breast, stage B_4; axillary hair, A_2; and pubic hair, P_2. On physical examination she had a goiter

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and hung-up reflexes. A hand X-ray revealed a bone age of 11 years. A thyroid function test revealed the following serum concentrations: T3, 1.1 ng/mL; T4, 5.6 μg/dL, and TSH, 19.7 mIU/L. Her serum levels of prolactin (7.2 μg/L), luteinizing hormone following stimulation with gonadotropin releasing hormone (46.2 IU/L), and FSH (10.4 IU/L) were postpubertal. On ultrasound the uterus was of adult sized (5.1×2.1×3.7 cm) and the ovaries were normal. Neuroimaging was advised but deferred by the patient. She was treated with levothyroxine and after 6 months she had gained 1.5 cm height and her menstrual cycle was regular.

Severe primary hypothyroidism can cause a distinct form of isosexual precocity, as initially described by Van Wyk-Grumbach [1]. Unlike in other forms of precocious puberty, thyroxine replacement results in prompt correction or amelioration of the features associated with precocious puberty. Boys usually present with macro-orchidism without excessive virilization. The proposed causes of the pseudo precocity found in children with hypothyroidism are increased secretion of gonadotropins due to hormonal overlap, hyperprolactinemia, reduced gonadotropin clearance, and stimulation of FSH receptor by TSH [2].

The growth plate is sensitive to estrogen and children with central precocious puberty have a tall stature initially but eventually a short stature because of early epiphyseal fusion. The lack of growth spurts in children with CPP could be due to an underlying growth hormone deficiency or hypothyroidism, the latter representing an additional risk of short stature. Reports that central CPP can be associated with growth hormone deficiency have been much more frequent than reports of its other association with hypothyroidism. Yet, treatment with gonadotropin releasing hormone analogues and growth hormone is controversial for children with this condition [3]. Hypothyroidism is widely prevalent and, as shown in the present report, may coexist with CPP.

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