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Letter to the Editor

Severe hypothyroidism due to autoimmune thyroiditis in a child: a one-year follow-up

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Dear Editor,

Autoimmune thyroiditis is considered to be the most common autoimmune condition, with a female preponderance of 2:1.[1,2] While its overall prevalence peaks in adulthood, autoimmune thyroiditis may affect children and adolescents, particularly during early to mid-puberty.[3] Hypothyroidism secondary to autoimmune thyroiditis may develop insidiously with slow and progressive physical changes that may remain unrecognized even to parents.[4] Nevertheless, early recognition of symptoms and signs of hypothyroidism is important to prevent its negative effects on growth, metabolic function, and intellectual performance.

An eleven-year-old Caucasian boy was evaluated for poor linear growth, with normal weight gain starting at the age of six and never investigated previously. Physical examination showed short stature (height: -2.2 SDS; target height: 0.1 SDS) with overweight (body mass index: +1.7 SDS), bradycardia, sparse-brittle hair, dry and thickened skin with myxedema involving the extremities (Figure 1). The child appeared severely hypokinetic with apathy and mildly reduced intellectual performance (low-normal IQ score of 81, assessed by Wechsler Intelligence Scale for Children, third edition-WISC III). He also reported fatigue, constipation, sleepiness, impaired school performance, and cold intolerance.

Bone age was severely delayed (6 years as assessed by the Greulich and Pyle standard). Serum thyroid-stimulating hormone (TSH) levels were remarkably increased (1,648 mIU/L) with undetectable concentrations of thyroid hormones and elevated anti-microsomal and anti-thyroid peroxidase antibodies titers (>2,040 IU/mL and >1,020 IU/mL, respectively). Thyroid ultrasound disclosed hypoechoic, inhomogeneous, and atrophic thyroid gland, confirming the diagnosis of severe hypothyroidism secondary to autoimmune thyroiditis.

Biochemical evaluation also revealed hypertransaminasemia (aspartate aminotransferase 151 U/L, alanine aminotransferase 117 U/L), dyslipidemia (total cholesterol 360 mg/dL, high-density lipoprotein cholesterol 29 mg/dL, low-density lipoprotein cholesterol 294 mg/dL, triglycerides 221 mg/dL), and rhabdomyolysis (creatine kinase 6,883 U/L). Cerebral MRI showed a pituitary mass of 18×12×10 mm extending into the suprasellar region, compatible with pituitary hyperplasia.

Low-dose levothyroxine administration was promptly started and progressively increased [0.5 μg/Kg/die (considering ideal weight according to height) for 2 weeks followed by 0.75 μg/Kg/die for 1 month, 1 μg/Kg/die for 1 month and 1.5 μg/Kg/die for

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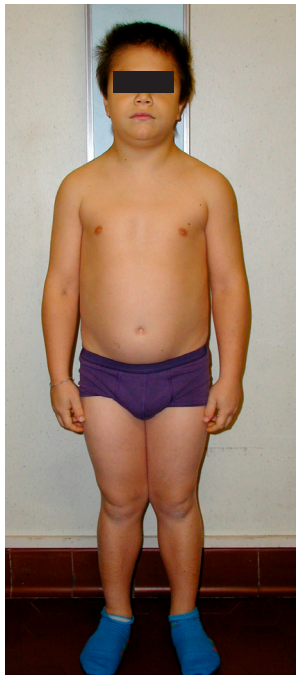

Figure 1. The patient at diagnosis with apathy, myxedema of the extremities, and sparse-brittle hair.

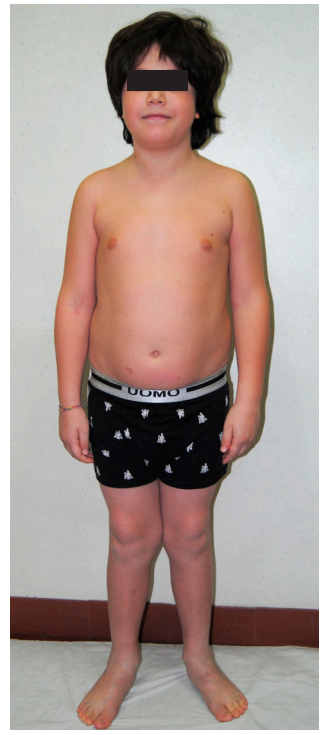

Figure 2. The patient after twelve months of therapy with levothyroxine. The resolution of myxedema and the normalization of hair growth were noticeable in association with the disappearance of apathy.

1 month, continuing with 2 μg/Kg/die afterwards]. Free thyroxine (fT4) and TSH levels normalized after 3.5 and 6 months of therapy, respectively. After twelve months of follow-up height improved to -1.7 SDS. The child displayed normal hair, complete resolution of myxedema, normalization of biochemical alterations and intellectual performance (IQ score 92) (Figure 2). Finally, MRI showed the resolution of pituitary hyperplasia.

The case we describe herein illustrates that forms of severe juvenile hypothyroidism are still relevant today. It seems unbelievable that nobody had recognized the progressive onset of severe hypothyroidism before the child developed severe multi-organ complications. Moreover, this case demonstrates that levothyroxine administration significantly improves the changes due to long-lasting hypothyroidism acquired during childhood.

A careful monitoring of growth is essential to avoid late diagnosis of diseases with possible long-term sequelae, such as acquired hypothyroidism. A child with poor linear growth should always be adequately investigated. Finally, the detection of a child with impaired linear growth associated with normal weight gain should always arouse the suspicion of hypothyroidism.[4]

DISCLOSURE STATEMENT:

The authors have nothing to disclose.

PATIENT CONSENT

Obtained from the parents.

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