

CASE REPORT

LATE DIAGNOSIS AND INADEQUATE TREATMENT
OF AUTOIMMUNE THYROIDITIS IN A CHILDNAVASARDYAN L.V.^{1,2,*}, MARKOSYAN R.L.^{1,2}¹ Department of Endocrinology, Yerevan State Medical University, Yerevan, Armenia² Clinic of Endocrinology, "Muratsan" University Hospital Complex, Yerevan State Medical University, Yerevan, Armenia

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ABSTRACT

Hypothyroidism is not just hormonal changes in thyroid status, but is a complex of laboratory findings, ultrasonographic changes and clinical features, including dry skin, mental retardation, edema, weakness, poor linear growth with increased weight for height, thyroid gland enlargement, bradycardia, blood pressure changes and heart rate decrease, etc. Clinical signs of hypothyroidism in pediatric population vary from asymptomatic cases to severe mixedematous ones.

The main cause of hypothyroidism is known to be autoimmune thyroiditis (Hashimoto's thyroiditis). The childhood prevalence of chronic autoimmune thyroiditis peaks in early to mid-puberty and a female preponderance of 2:1 have been reported. As it is known, autoimmune thyroiditis is T-cell induced mediated autoimmune destruction of follicular cells of thyroid gland, leading to thyroid hormones (T3, T4) insufficiency and to increase in thyroid stimulating hormone levels, and is characterized by the presence of autoantibodies against thyroglobulin and thyroperoxidase.

Present study describes a case of late diagnosis and inappropriate treatment of autoimmune thyroiditis in a 9-years old girl and its consequences. Ultrasonography revealed characteristic structural abnormalities of autoimmunity with thyroid size diffuse enlargement and significant increase of hormone levels in the girl. She was diagnosed with autoimmune thyroiditis on the basis of hormonal analyses and instrumental examination results. The proper lifelong therapy with levothyroxine was administered, which had positive effect, i.e., thereafter, the reduction of thyroid gland sizes and decreased levels of hormones in blood was noted. However, late diagnosis and inadequate treatment resulted in irreversible changes in thyroid structure and cosmetic defects of neck.

Thus, it can be concluded, that early diagnostics and adequate therapy are the main cornerstones in the prevention of further irreversible complications.

KEYWORDS: autoimmune thyroiditis, hypothyroidism, diffuse goiter, levothyroxine, complications.

Introduction

Autoimmune thyroiditis, also known as Hashimoto's thyroiditis or chronic lymphocytic thyroiditis, seems to be the most common cause of hypothyroidism in pediatric population [Cappa M et al., 2011]. The childhood prevalence of chronic autoimmune thyroiditis peaks in early to mid-puberty [Hunter I et al., 2000; Ergul B et al., 2013] and a

female preponderance of 2:1 have been reported [Kordonouri O et al., 2002]. Autoimmune thyroiditis develops on genetical and environmental base [Tanda M et al., 2009; Tomer Y, Huber A, 2009; Brent G, 2010]. As it is known, autoimmune thyroiditis is T-cell induced mediated autoimmune destruction of follicular cells of thyroid gland, leading to thyroid hormones (T3, T4) insufficiency and increase in thyroid stimulating hormone levels [Villanueva R et al., 2003; Dittmar M et al., 2011], and is characterized by the presence of autoantibodies against thyroglobulin and thyroperoxidase

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[Deirdre C et al., 2010]. They are found not only in the blood samples of patients with autoimmune thyroiditis, but also in some individuals without clinical and hormonal changes in thyroid status. Antithyroperoxidase antibody levels tend to correlate with autoimmune thyroiditis better than anti-thyroglobulin antibodies [Caturegli P et al., 2007; Klecha A et al., 2008].

Thyroid stimulating hormone is known to stimulate not only the secretion of thyroid hormones, but also thyroid enlargement, leading to diffuse goiter in case of thyroid stimulating hormone elevation. One of the main pathways of diffuse goiter in autoimmune thyroiditis is the primary hypothyroidism with highly elevated thyroid stimulating hormone levels [Gopalakrishnan S, Marwaha R, 2007].

There are two known forms of autoimmune thyroiditis – hypertrophic or goitrous, and atrophic [Gopalakrishnan S et al., 2008]. Histologically, hypertrophic form of autoimmune thyroiditis is characterized by diffuse lymphocytic infiltration of thyroid gland. Thyroid follicles may be reduced in size or enlarged. Individual thyroid cells are often enlarged with oxyphilic cytoplasm (usually defined Hurthle cells). In contrast, the gland of atrophic autoimmune thyroiditis is small with lymphocytic infiltration and fibrous replacement of the parenchyma.

Ultrasonography of the thyroid gland can reveal characteristic structural abnormalities such as generalized hypo-echogenicity due to inflammation and diffuse lymphocytic infiltration with occasional germinal centers (pseudonodules) [Enăchescu V et al., 2006; Vlachopapadopoulou E et al., 2009; Navasardyan L, Gevorgyan M, 2014].

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A 9 year old girl applied to the Clinic of Endocrinology of “Muratsan” Hospital Complex in 2014, approximately in a month after the detection of goiter by primary physician (Fig. 1). The enlarged thyroid gland was seen “ad oculus” as multinodular, however, the palpation of it revealed it diffusely enlarged and not tender. The skin was dry; the decline in mental activities was noted.



FIGURE 1. Hypertrophic form of autoimmune thyroiditis: Goitrous autoimmune thyroid: a neglected case in a 9 year old girl.



FIGURE 2. Thyroid gland after treatment: a - after 6-month treatment, b - after two-year treatment

Weakness, constipation, weight gain and headaches were not registered, but as a result of the study the decrease was revealed in voltage by ECG. Ultrasonography found characteristic structural abnormalities of autoimmunity with thyroid size diffuse enlargement, total volume of the gland was 36 cm^3 . Neither nodules nor cysts have been found by ultrasonography. Levels of hormones in blood were: thyroid stimulating hormone $>100 \text{ UI/ml}$ (norm – 0.5-4.0), free T4 – 3.2 pmol/l (norm – 12-22), free T3 – 1.1 pmol/l (norm – 3.1-6.8), anti-thyroperoxidase $>600 \text{ IU/ml}$ (norm <34), and anti-thyroglobulin $>1000 \text{ IU/ml}$ (norm <100).

She was diagnosed with autoimmune thyroiditis on the basis of hormonal analyses and instrumental examination results [Setian N, 2007]. The proper lifelong therapy with levothyroxine was administered. However, interestingly, the compliance of patient was too poor, so that she didn't take her medicine adequately. Figure 2 shows the thyroid gland enlargement after six-month treatment, and figure 3 – thyroid gland after two-year treatment with compensated thyroid status – thyroid stimulating hormone = 1.22 UI/ml , free T4 – 15.8 pmol/l . Gland size reduction is obvious even without ultra-

sonography (total volume is 20.2 cm^3 according to ultrasonography). However, that reduction was not enough satisfying for age median. Late diagnosis and inadequate treatment resulted in irreversible changes in thyroid structure and cosmetic defects of neck, which probably cannot disappear even after long period of adequate therapy.

Discussion

Mostly autoimmune thyroiditis is diagnosed not in early stages at the onset of disorder, but in stages of clinical manifestation. As the disease progresses, subclinical and then clinical hypothyroidism appears. Highly elevated thyroid stimulating hormone can lead to diffuse goiter – enlargement of thyroid sizes compared to age median, which is mostly defined by ultrasonography, and rarely by palpation. Early diagnosis of thyroid dysfunction is necessary to prevent the negative effects of hypothyroidism not only on growth and metabolic function, but also on goiter development. Autoimmune thyroiditis should be suspected in the presence of goiter, even in the absence of signs and symptoms of thyroid dysfunction, particularly in children.

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