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Case Report
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Conversion of Autoimmune Thyroiditis into Grave's Disease during Pregnancy and Levothyroxine Intake

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Hypothyroidism; Grave's disease; Pregnancy; Levothyroxine; TSH receptor antibodies; Type 1 diabetes mellitus

Introduction

Pregnancy causes many changes in a woman's body. Some of them are transient in nature, while others remain for the rest of the patient' s life. Dramatic changes do not bypass the Thyroid Gland (TG). Against the background of pregnancy, both hypo- and hyperthyroidism can develop, which should be diagnosed and treated in time. Sometimes changes in thyroid function are so unexpected that they become an unpleasant surprise for a young mother and her doctor.

Clinical Case

A 26-year-old patient with Type 1 Diabetes Mellitus (T1DM) was admitted to the endocrinology department. T1DM was diagnosed in 2005 at the age of 10 years. At the onset of the disease, basal-bolus insulin therapy was started.

In October 2018, she became pregnant (at the time of pregnancy, high glycemia levels were noted with high variability from 2 mmol/l to 21 mmol/l), however, due to an embryony diagnosed at 9 to 10 weeks of pregnancy, vacuum aspiration of the uterine cavity was performed. Due to poor control of diabetes, an insulin pump was installed.

For the second pregnancy careful assessment was performed, including regular TSH control. A repeated increase in TSH up to 3.1 μIU/ml to 3.3 μIU/ml (0.4-4.0) was noticed.

There was a decrease in thyroid tissue echogenicity and diffuse heterogeneity according to the ultrasound data. FT4 (18.46 pmol/l (8.2-24.7)) and fT3 (5.18 pmol/l (2.76-6.45)) were within the normal range with elevated Antibodies to Thyroid Peroxidase (TPOAb) 104.6 IU/ml (<60), Antibodies to TSH receptors (TSHrAb) were not detected. Taking into account the presence of T1DM and increased TPOAb in combination with slightly elevated TSH during pregnancy planning, levothyroxine 50 mcg/day was prescribed.

Just before pregnancy TSH decreased to 1.3 μIU/ml. During pregnancy, there was a decrease in TSH to a minimum of <0.004 μIU/ml, levothyroxine treatment was cancelled and at the time of childbirth in July 2021 TSH level was 2.139 μIU/ml.

Two months after delivery there was a decrease in TSH to 0.028 μIU/ml. After another 3 months, the patient noticed a significant glycemic variability during the day and was admitted to hospital with high glycaemia.

A decrease in TSH to 0.008 μIU/ml was determined with increase in fT4 up to 31.7 pmol/l (8.2-24.7) and fT3 up to 12.8 pmol/l (2.76-6.45), TSHrAb were determined (5.12 IU/l (<1)). In a hospital tachycardia with heart rate up to 100 beats/min to 120 beats/min was revealed. According to ultrasound of the thyroid gland, the total volume was 6.7 cm^3, with diffuse changes in the tissue. So, she was diagnosed with Graves' disease.

Thus, the diagnosis "Autoimmune polyglandular syndrome type 3" as a combination of T1DM and Graves' disease was established. As the lactation at the time of treatment was stopped, antithyroid therapy with thiamazole 10 mg 2 times a day was initiated. Bisoprolol 7.5 mg was prescribed as pulse-lowering therapy.

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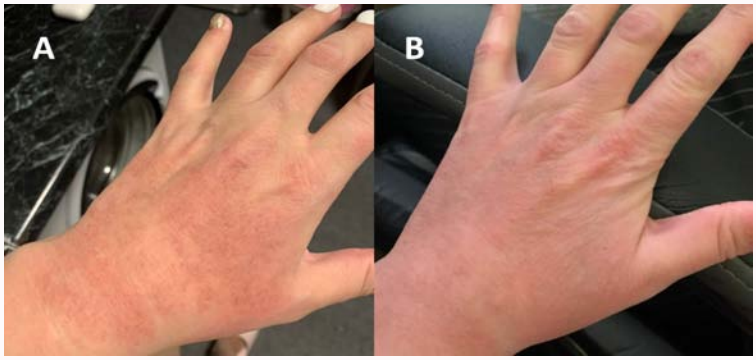

Figure 1: A) Erythema on the back of the hands. B) Condition after therapy correction.

Approximately 1 month after starting the drug, the patient noted an allergic reaction in the form of erythema on the back of the hands (Figure 1A). Thiamazole was replaced by propylthiouracil at a dose of 50 mg 3 times a day. Against this background, there was a rapid trend towards normalization of the skin condition (Figure 1B).

Despite the irregular intake of the drug, a dynamic study after 1 and 3 months after the start of therapy showed normal fT4 and fT3 and elevation of TSH, and therefore the dosage of propylthiouracil was reduced to 50 mg 2 times a day. Further, the patient also took the drug irregularly. There were some fluctuations in TSH level, however, over the next 7 months, both TSH and thyroid hormones were within the reference values, TSHrAb returned to normal range. As she stopped treatment and had target TSH after 4 months of discontinuation, we didn't resume anti-thyroid treatment and continue monitoring TSH.

Discussion

Despite significant differences in the pathogenesis and clinical presentation between Autoimmune Thyroiditis (AIT) and Grave's Disease (GD) a conversion of one autoimmune disease to another is described in the literature. Observations of the transition of GD to AIT are more common, and much less frequently we can see the development of GD in a patient with AIT. According to one theory, this is due to the absence of a critical mass of thyroid tissue capable to respond to TSHrAb in people with a long history of AIT [1]. In any case, hypothyroidism and thyrotoxicosis can occur consecutively in the same patient [2].

According to literature, it becomes obvious that the problem is quite rare in relation to the general prevalence of autoimmune thyroid disorders, and therefore it is valuable to describe each clinical case.

The reasons for the switch in thyroid function are not completely clear at the moment, however, it is clear that there are several factors that, both individually or in combination, can influence the transition from hypo- to hyperthyroidism and *vice versa*. First of all, this may be due to switching between stimulating (TSAb) and blocking (TBAb) TSHrAb. It was traditionally believed that patients with thyrotoxicosis have only TSAb, and patients with hypothyroidism have only TBAb. However, both types of antibodies can circulate in the same patient. Thyroid function may fluctuate depending on predominating antibodies [2].

Exact factors leading to the switch from the production of one type of TSHrAb to others aren't known for certain. There are observations that changes in the type of antibodies sometimes occur under the influence of levothyroxine or antithyroid drugs. During treatment period the concentration and mode of action of antibodies can change [3].

Another possible explanation for the transition from hypo- to hyperthyroidism is that the thyroid tissue, which has undergone very serious autoimmune damage that has led to hypofunction of the gland, eventually recovers sufficiently to respond to subsequent exposure to stimulating antibodies [4]. In addition, there are theories that the thyroid function can switch under the influence of various external factors, such as viral infection, pregnancy can also trigger the changes [5,6].

In case of our patient, there are several options for the causes of GD manifestation after childbirth. According to one theory, pregnancy was the stimulus for the conversion of hypothyroidism into thyrotoxicosis. During pregnancy, thyroid autoantibody levels usually decrease due to immunosuppression and/or hemodilution and are the lowest shortly before delivery, especially stimulatory TSHrAb levels. However, in the postpartum period, the levels of anti-thyroid antibodies are restored, and then can even increase [5].

According to the second theory, the conversion occurred under the influence of levothyroxine treatment. It is believed that can happen either due to the restoration of thyroid tissue and the better response to circulating TSAb, or due to a decrease in the number of TBAb during levothyroxine treatment and a comparative increase in TSAb [3,4]. In our patient, most likely, the trigger for the development of Graves' disease could be both the pregnancy itself and the use of levothyroxine. Unfortunately, there is no data on TSHrAb level before pregnancy, so it will not be possible to establish a precise cause retrospectively.

As such clinical observations occur sometimes in patients, they are really important for doctors, since in clinical practice a decrease in TSH in a patient treated with levothyroxine, in most cases will be regarded as an overdose of it, which may lead to dose reduction and loss of time, while detection of GD can significantly change the treatment and favorably affect the prognosis of the disease, preventing the development of complications.

Whatever the genesis of the development of GD in our patient, the value of this clinical case is that a patient with T1DM got a second autoimmune disease after pregnancy - GD. Thus, we can diagnose the autoimmune polyglandular syndrome type 3. This clinical case makes us remember the increased risk of developing a second autoimmune disease in patients with T1DM, which may manifest many years after the onset of diabetes, while pregnancy may trigger its manifestation.

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