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DECREASE THYROID HORMONES NEEDS AFTER INTRODUCTION OF OMALIZUMAB IN PATIENTS WITH CHRONIC SPONTANEOUS URTICARIA AND NON AUTO-IMMUNE HYPOTHYROIDISM

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INTRODUCTION

Chronic spontaneous urticaria (CSU) can be associated with thyroid diseases, mostly auto immune in 4.3-57.4% of cases. We report here two patients with CSU and non-autoimmune thyroid diseases, for which introduction of omalizumab lead to a decrease need of thyroid hormones.

CASE REPORT 1

- 53 years old woman

- history of hypertension and subtotal-thyroidectomy for thyroid nodule, no anti thyroperoxydase (TPO) antibody

- CSU resistant to H1 second-generation antihistamine (up to 3 /d), montelukast and methotrexate

- Treated with levothyroxine 175µg alterning with 150µg every other day, for a long time with stable level of TSH

- Omalizumab (300 mg/4 weeks) enabled a good control of CSU (Urticaria Control Test (UCT): 14/16).

- A few months after its introduction, despite a stable weight and the absence of introduction of any treatment able to interact with levothyroxine, the dosage of TSH was very low, and the posology of levothyroxine decreased to 125µg/d.

CASE REPORT 2

- 49 years old woman

- history of Biermer disease and hypothyroism post subacute thyroiditis (no anti TPO antibody)

- CSU resistant to H1 second-generation antihistamine (up to 4/d) and montelukast.

- Treated with levothyroxine 100µg a day and liothyronin 0.025 mg a day, for a long time with stable level of TSH.

- Omalizumab (300 mg/ 4 weeks 2 months, then 300 mg/2 weeks) enabled a good control of CSU (UCT: 15/16).

- A few months after its introduction, despite a stable weight and the absence of introduction of any treatment able to interact with levothyroxine, the dosage of TSH was very low, and the posology of levothyroxine decreased to 75µg/d.

OMALIZUMAB

- IgE monoclonal antibody licensed for CSU unresponsive to H1 second-generation antihistamine.
- Not known to interact with production, transport, metabolism or activity of iodothyronines.

In the absence of auto-immune thyroid disease for our two patients, we cannot hypothesize an action of omalizumab on anti-thyroid antibodies to explain the decrease need of thyroid hormones.
So, we can imagine two hypotheses; the first is that omalizumab interferes with a step of the thyreotropic axis, the second is that control of CSU promotes thyroid hormone production

CONCLUSION

We recommend to closely managing TSH after introduction of omalizumab in patients with any thyroid disease.