Thyroglobulin antibodies are associated with symptom burden in patients with Hashimoto's thyroiditis

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Objectives: Hashimoto thyroiditis (HT) is the most common form of autoimmune thyroid disorders, caused by antibodies to thyroglobulin (TgAb) and thyroid peroxidase (TPOAb). Today there is rising interest in influence of thyroid antibodies on human health. In our study we have included large cohort of patients with HT to investigate possible influence of thyroid autoimmunity on symptom burden.

Methods: In the period from 2013 to 2017 we collected data from 290 HT patients at the Department for Nuclear Medicine at the University Hospital Split, including 270 females (93%) and 20 males (7%). We collected information on thyroid-specific phenotypes (TSH, T3, T4, fT4, TgAb, TPOAb, thyroid volume) and other clinical phenotypes (age, body surface area, number of hypothyroidism symptoms, blood pressure) from patients with HT without levothyroxine (LT4) therapy.

Results: We have analysed correlations between thyroid-specific and other clinical phenotypes (Table 1). The most frequently reported symptom was weakness (63%), and the rarest symptom was slow speech (Figure 1). We have found significant positive correlation between TgAb levels and the number of symptoms (r=0.25, P=0.0001) in HT patients that remained significant after adjustment for TPOAb, T3, TSH levels and thyroid volume (β=0.66, SE=0.3, P=0.0299). Increased TgAb levels are significantly associated with fragile hair (P=0.0043), face edema (P=0.0061), edema of the eyes (P=0.0293) and harsh voice (P=0.0349) (Figure 2.).

Conclusions: TgAb have important effect on general health and clinical manifestations of HT, and elevated TgAb level may cause the observed symptom burden in HT patients, leading to conclusion that not all HT patients may be clustered in one group. Based on these results, we recommend screening for TgAb antibodies in HT patients with symptom burden. The symptoms in patients with HT should be further differentiated to those that are truly caused by hypothyroidism and those that develop due to autoimmunity per se.