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LETTERS TO THE EDITOR

Hashimoto Thyroiditis as a Cause or Consequence of Obstructive Sleep Apnea

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We are writing to highlight the potential interaction between the pathogenesis of obstructive sleep apnea (OSA) and Hashimoto thyroiditis (HT), the most common autoimmune disease of the thyroid. HT is characterized by lymphocytic infiltration of the gland and the presence of thyroid autoantibodies, and has been associated with several systemic comorbidities.[1]

Despite the consistent association between sleep disorders and hypothyroidism, the interaction between OSA and autoimmune thyroiditis has not been well established, which could be significant because of the relatively high prevalence of OSA.[2]

It has been suggested that OSA can increase the risk of the development of autoimmune diseases, such as HT. This mechanism could be explained by the cellular injury induced by hypoxia in OSA. This could result in an upregulation of the immune response due to exposure to antigen-presenting cells. A study investigating the prevalence of HT in euthyroid individuals suspected of having OSA concluded that patients with OSA presented higher HT prevalence.[2]

There is some evidence suggesting that the relationship between OSA and HT may be bidirectional. A study examining this coexistence in seven patients found that five patients with HT showed characteristics of OSA, whereas no sleep breathing disorder was found in the control group. This suggests that OSA may develop in patients with autoimmune thyroiditis.[1] Another study did find that patients with high serum thyroid-stimulating hormone (TSH) levels reported higher scores in the Epworth Sleepiness Scale than those with normal TSH levels. The study concluded that subclinical hypothyroidism and treatment did not influence the prevalence of OSA, but sleep propensity is increased by untreated subclinical hypothyroidism.[3]

Considering the immunologic aspects, interleukins (IL)-6 and -10 are emerging as prominent blood markers for OSA, although it is not yet fully elucidated. If this is confirmed, these biomarkers could make the diagnosis and follow-up of OSA considerably easier.[4]

With the increasing number of sleep-disordered breathing diseases,[5] clinicians must be prepared to implement appropriate management aiming at the prevention of health complications, such as thyroid diseases.[4] If future studies make the association between HT and OSA clearer, HT should also be considered as a possible risk factor for OSA.

Polysomnography is the gold standard for diagnosing sleep disorders and OSA severity. We suggest it is important that it be considered in the evaluation of patients with HT, especially for those with symptoms of OSA. Further research is needed to build a better understanding of this interaction, aiming to improve the sleep quality and treatment outcomes of this population as well as contributing to better quality of life.

CITATION

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REFERENCES

1. Erden S, Cagatay T, Buyukozturk S, Kiyan E, Cuhadaroglu C. Hashimoto thyroiditis and obstructive sleep apnea syndrome: is there any relation between them? *Eur J Med Res*. 2004;9(12):570–572.
2. Bozkurt NC, Karbek B, Cakal E, Firat H, Ozbek M, Delibasi T. The association between severity of obstructive sleep apnea and prevalence of Hashimoto's thyroiditis. *Endocr J*. 2012;59(11):981–988.
3. Resta O, Carratù P, Carpagnano GE, et al. Influence of subclinical hypothyroidism and T4 treatment on the prevalence and severity of obstructive sleep apnoea syndrome (OSAS). *J Endocrinol Invest*. 2005;28(1):893–898.
4. Tufik SB, Berro LF, Andersen ML, Tufik S. Prevalence and classification of sleep-disordered breathing. *Lancet Respir Med*. 2015;3(4):263–264.
5. Tufik S, Santos-Silva R, Taddei JA, Bittencourt LR. Obstructive sleep apnea syndrome in the Sao Paulo Epidemiologic Sleep Study. *Sleep Med*. 2010;11(5):441–446.

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