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THYROID AUTOIMMUNITY IN THE CONTEXT OF OBESITY, INSULIN RESISTANCE AND METABOLIC RISK: CURRENT EVIDENCE

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ABSTRACT

Background: Patients with obesity may show thyroid autoantibodies despite preserved thyroid hormone levels. The significance of this finding for insulin resistance and metabolic disturbances is not fully defined.
Aim: To summarise current evidence on the relationships between thyroid autoimmunity, obesity, insulin resistance, and metabolic risk, with particular attention to euthyroid individuals.
Methods: Population-based, observational, and interventional studies published in recent years were reviewed. The literature was analysed in relation to thyroid autoantibodies, metabolic characteristics, markers of insulin resistance, and the effects of metabolic interventions.
Results: Thyroid autoantibodies occur more often in people with obesity and metabolic syndrome, particularly in those with abdominal obesity and lipid disturbances. Associations with glucose levels and blood pressure are less consistent.
In euthyroid individuals, thyroid autoimmunity is commonly accompanied by insulin resistance and dyslipidaemia, along with altered adipokine profiles and markers of low-grade inflammation.
Weight reduction achieved through dietary intervention or bariatric surgery has been associated with lower thyroid autoantibody levels. At the same time, patients with thyroid autoimmunity tend to achieve smaller reductions in body weight compared with those without antibodies.
Conclusions: Thyroid autoimmunity is linked to specific metabolic phenotypes rather than acting as an independent determinant of metabolic risk. Improvement in metabolic status may be associated with reduced autoimmune activity, while the presence of thyroid autoimmunity appears to be related to a less favourable response to weight-loss interventions.

KEYWORDS

Thyroid Autoimmunity, Obesity, Insulin Resistance, Metabolic Syndrome, Thyroid Autoantibodies, Euthyroid State

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1.Introduction

Autoimmune thyroid diseases are usually discussed in the literature mainly in relation to changes in thyroid function. Other clinical aspects, such as their relationship with obesity and metabolic disturbances, have been analysed less frequently.

In autoimmune thyroid disease, antibodies against thyroid peroxidase and thyroglobulin are typically present. They may be detected long before hypothyroidism develops. Thyroid hormone levels usually remain within the reference range at this stage, and patients are classified as euthyroid [1,2].

In recent years, thyroid autoimmunity has also been reported in individuals with excess body weight, suggesting that metabolic factors may be involved in its clinical presentation [3,4].

Obesity is a chronic disease associated with insulin resistance, dyslipidaemia and increased cardiovascular risk [5]. In clinical practice, patients with obesity are often evaluated for thyroid disorders, including autoimmune thyroid disease. Thyroid autoantibodies can be detected in patients with obesity despite normal thyroid hormone levels [1].

Obesity is most often classified according to body mass index. BMI reflects body weight but does not describe fat distribution. Abdominal and visceral adipose tissue are metabolically active and are associated with chronic inflammation and altered adipokine secretion [6]. These processes are related to immune activation observed in autoimmune thyroid disease [7].

Insulin resistance is common in patients with obesity and may be present even when thyroid hormone levels are normal. Changes in thyroid-stimulating hormone within the reference range have been reported in this group [6]. Thyroid autoantibodies have also been described in patients with insulin resistance despite

normal thyroid hormone levels [8,9]. The duration of excess body weight may also be relevant for the development of thyroid autoimmunity [2].

The aim of this review is to examine clinical and population-based studies that assessed thyroid autoimmunity in the context of insulin resistance, metabolic risk, and obesity.

2. Thyroid autoimmunity and obesity

The coexistence of obesity and autoimmune thyroid disease has been frequently observed in clinical practice. Population-based studies have examined the prevalence and incidence of thyroid autoantibodies in individuals with excess body weight [10].

In a nationwide study, Kim et al. reported a higher prevalence of thyroid autoimmunity in participants meeting criteria for metabolic syndrome compared with those without this diagnosis [11]. Obesity was one of the main components associated with antibody positivity.

More detailed analyses have focused on fat distribution rather than body mass index alone. In a 9-year follow-up study, Amouzegar et al. did not observe a higher overall incidence of thyroid autoantibody development in individuals with increased baseline body weight. However, when abdominal obesity phenotypes were analysed separately, central fat accumulation was associated with a higher risk of thyroid autoimmunity [12]. Similar associations between central obesity and thyroid autoantibody positivity were reported in other populations [13]. Together, these findings indicate that fat distribution, particularly abdominal adiposity, may be more relevant than body mass index alone.

Clinical studies have also examined thyroid autoimmunity in individuals with obesity who had thyroid hormone levels within the reference range. In these patients, thyroid autoantibodies were present despite normal thyroid hormone concentrations and without clear biochemical thyroid dysfunction [1,8]. In patients with Hashimoto's thyroiditis, obesity was associated mainly with differences in metabolic profile rather than with more advanced thyroid impairment [14].

3. Insulin resistance and thyroid autoimmunity

Insulin resistance commonly accompanies obesity and is associated with increased cardiometabolic risk [5,15].

To minimise the influence of overt thyroid dysfunction, several studies evaluated the relationship between insulin resistance and thyroid autoimmunity in individuals with normal thyroid function [8,9]. Insulin resistance was assessed using HOMA-IR, calculated from fasting plasma glucose and fasting insulin concentrations.

In a study by Blaslov et al., euthyroid individuals with Hashimoto's thyroiditis had higher HOMA-IR values than antibody-negative subjects ($p = 0.003$). Each increase of 1 unit in HOMA-IR was associated with an increase of approximately 18.1 IU/mL in TPOAb ($p = 0.001$).

Similarly, in a large study of non-obese euthyroid individuals, those with autoimmune thyroiditis had higher mean HOMA-IR than controls (2.78 ± 1.60 vs 2.33 ± 1.49; $p < 0.05$) [8,9].

In both analyses, higher HOMA-IR values were observed despite normal TSH and free thyroid hormone concentrations. Insulin resistance was also associated with less favourable glycaemic and lipid profiles [9,15], and similar metabolic disturbances have been reported before the development of overt thyroid dysfunction in autoimmune thyroid disease [2,4,16].

4. Metabolic syndrome and thyroid autoimmunity

Metabolic syndrome is defined by abdominal obesity together with at least two additional components: elevated triglycerides (≥150 mg/dL), reduced HDL cholesterol (<40 mg/dL in men and <50 mg/dL in women), elevated blood pressure (≥130/85 mmHg or current antihypertensive treatment), or impaired fasting glucose (≥100 mg/dL) [5].

Kim et al. analysed 4,775 euthyroid adults; 1,206 (25%) met the criteria for metabolic syndrome. TPOAb positivity was higher in the MetS group than in those without MetS (7% vs 5%, $P = 0.002$), and median TPOAb levels were 6.8 vs 6.3 IU/mL ($P < 0.001$) [11].

In the same cohort, TPOAb positivity was associated with selected components of metabolic syndrome. Individuals with positive TPOAb had higher odds of abdominal obesity (OR 1.675; 95% CI 1.302–2.154; $p < 0.001$), low HDL cholesterol (OR 1.603; 95% CI 1.244–2.066; $p < 0.001$), and elevated blood pressure (OR 1.418; 95% CI 1.099–1.829; $p = 0.007$) compared with antibody-negative subjects [11].

Not all components of metabolic syndrome showed the same strength of association. Relationships were more consistent for abdominal adiposity and lipid abnormalities, whereas associations with glucose levels and blood pressure were weaker or less consistent in other studies [17,18].

Beyond classical metabolic syndrome components, altered adipokine profiles have also been reported in individuals with metabolic syndrome, including higher leptin and lower adiponectin levels [19,20]. Leptin concentrations were associated with thyroid autoantibody positivity independently of body mass index and thyroid hormone levels [3]. This specific metabolic phenotype is also characterised by low-grade chronic inflammation, reflected by mildly elevated hs-CRP, increased interleukin-6 concentrations, and evidence of inflammasome activation [21,22,23].

5. Sex- and age-related differences in thyroid autoimmunity and metabolic risk

Thyroid autoimmunity differs between women and men, both in prevalence and in its metabolic-related associations. Thyroid autoantibodies are more frequently detected in women than in men [1,11,23]. However, in men their presence appears to be more closely linked with metabolic disturbances.

In population-based studies, men with positive thyroid autoantibodies were more often classified as having metabolic syndrome and more frequently presented with lipid abnormalities compared with women with thyroid autoimmunity [11,13,23]. In women, thyroid autoantibodies were often detected in euthyroid individuals without marked metabolic disturbances [1,23]. These observations suggest that the clinical context of thyroid autoimmunity may differ by sex.

Age also modifies the relationship between thyroid autoimmunity and metabolic risk. In younger individuals, thyroid autoantibodies are often detected incidentally, without overt thyroid dysfunction or significant metabolic abnormalities [2]. In contrast, in older adults thyroid autoimmunity is more frequently accompanied by visceral obesity, dyslipidaemia and other cardiometabolic risk factors [1,11].

Long-term exposure to excess body weight appears to be relevant. In the 1946 British birth cohort, greater weight gain from childhood to adulthood was associated with a higher likelihood of thyroid autoantibody positivity at 60–64 years of age [2]. Similarly, in a 9-year follow-up study, persistent abdominal obesity phenotypes were associated with an increased incidence of thyroid autoimmunity [12]. These findings indicate that long-term excess adiposity may be relevant for the later development of thyroid autoimmunity.

6. Impact of metabolic interventions on thyroid autoimmunity

The effect of weight reduction on thyroid autoimmunity has also been examined.

In a cohort of patients treated with bariatric surgery, significant weight loss was observed during follow-up ($p < 0.001$). In individuals with positive thyroid autoantibodies, TPOAb levels decreased after surgery. However, patients with thyroid autoimmunity achieved a lower percentage reduction in body weight than antibody-negative subjects [24].

Similar findings were reported in a six-month dietary intervention study in women with obesity and Hashimoto's thyroiditis. Weight loss was followed by significant decreases in anti-TPO and anti-TG levels ($p < 0.05$). Larger reductions in fat mass were linked to greater declines in antibody concentrations [25].

In patients undergoing dietary or surgical weight reduction, antibody levels decreased during follow-up. Patients with thyroid autoimmunity achieved a smaller percentage reduction in body weight compared with antibody-negative subjects.

7. Conclusions

In euthyroid adults, TPOAb positivity is reported in about 5–7% of individuals and is more common in those with metabolic syndrome. Studies describe an association between thyroid autoimmunity and obesity, insulin resistance and lipid disturbances.

However, these associations differ according to sex and age and seem to be related to the duration of excess body weight. Thyroid autoantibody positivity is most often observed in individuals with visceral obesity, lipid abnormalities and features of low-grade inflammation, whereas links with glucose metabolism and blood pressure are less consistent.

Available data do not indicate a clear causal link between thyroid autoimmunity and metabolic risk. Results from Mendelian randomisation and longitudinal studies point to a complex relationship. In euthyroid individuals, the extent to which thyroid autoantibodies independently affect cardiometabolic risk remains unclear.

Weight reduction has been accompanied by decreases in thyroid antibody levels in interventional studies. At the same time, patients with thyroid autoimmunity tended to achieve smaller reductions in body weight. The durability of antibody reductions and their impact on future thyroid dysfunction have not been established.

Further longitudinal studies with prolonged follow-up are necessary to clarify the clinical significance of thyroid autoimmunity in patients with metabolic risk.

Author's contribution:
Wiktoria Polkowska- Conceptualization, review and editing, investigation, methodology
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Magdalena Korba- Conceptualization, visualization, resources
Adrianna Kowalik- Review, data curation, investigation
Agnieszka Krajewska- Resources, writing- rough preparation, data curation
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