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THYROID AUTOIMMUNITY AND DIABETES MELLITUS

Autoimmune endocrine diseases are disorders in which immune dysregulation results in immune attack on the endocrine glands. The most common autoimmune endocrine disorders are type 1 diabetes mellitus and the autoimmune thyroid diseases. Autoimmune thyroid disorders are the most prevalent immunological diseases in patients with type 1 diabetes. Thyroid autoimmunity is more prevalent in diabetic females than males and is associated with persistently higher levels of thyroid and b-cell autoimmune markers.

Cross-sectional studies have reported a prevalence of hypothyroidism in 12–24% of female and in about 6% of male patients with type 1 diabetes, as well as in 3–6% of type 2 diabetic patients. Hyperthyroidism occurs in 1–2% of patients with diabetes. Thyroperoxidase autoantibodies are a marker of autoimmune thyroiditis and are found in 20–30% of patients with type 1 diabetes. It may take years for patients with positive autoimmune markers to develop thyroid disease. As a consequence, some authors recommend routine screening of thyroid function in type 1 diabetes patients. The data indicate that this evaluation must be done at diagnosis, at least in adolescent and adult patients.

Only limited data are available on the possible influence of the coexistence of thyroid autoimmunity and autoimmune diabetes on the nature and course of diabetes. The most frequently cited finding refers to poorer diabetes compensation during pregnancy, particularly in the second and third trimesters and in the early post-partum period, in female diabetic patients with positive thyroid antibodies. To date, studies of diabetic patients with disease duration longer than l0 years have not demonstrated a different prevalence of chronic diabetic complications (retino-nephro- neuropathy) associated with the presence or absence of autoimmune thyroiditis.

The pathogenetic mechanism underlying the simultaneous occurrence of these clustered autoimmune diseases has not been clearly elucidated. There is evidence that common genetic determinants, in particular sharing of HLA risk alleles or other genes outside the HLA region (*CTLA4* gene) could be involved. Moreover, environmental factors are also assumed to be involved in the pathogenesis of these complex diseases. Whether the influence of exogenous agents leads to a faster onset of these autoimmune diseases, and particularly of type 1 diabetes, in patients with multiple diseases compared to those with only one disease needs to be assessed by epidemiological studies.

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