Hashimoto’s thyroiditis (hypertrophic chronic lymphocytic thyroiditis): The centennial of a discovery

Jean-Louis Wémeau

CHRU, hôpital Claude-Huriez, clinique endocrinologique Marc-Linquette, 59037 Lille cedex, France

Correspondence: Jean-Louis Wémeau, CHRU, hôpital Claude-Huriez, clinique endocrinologique Marc-Linquette, 59037 Lille cedex, France. jl-wemeau@chru-lille.fr

In 1912, Hakaru Hashimoto published the description of four cases of goiter, which were histologically characterized by a lymphoplasmocytic infiltrate with diffuse inflammatory alterations of the thyroid parenchyma and fibrosis [1]. The young student pointed out that the histological appearance of these goiters (“struma lymphomatosa”) was very different from the colloid goiters that he was used to studying, as well as those from Grave’s disease, infectious thyroiditis (especially related to tuberculosis or syphilis) and the fibrous thyroiditis described by Riedel in 1896. He pondered the cause of the disease, and emphasized similarity with the histological data observed with lacrimal, salivary, lymph node and splenic involvement of Mikulicz’s disease.

The four observed cases involved female patients over the age of 40 years who had formed very firm diffuse goiters, to the extent that the diagnoses were suggestive of fibrous thyroid or cancer. But these were isolated, with hardly any severe signs of compression and no adenopathy. The postoperative course of these goiters seemed more complicated than usual. One case was complicated by recurrence, soon followed by spontaneous reduction of the hypertrophy.

These observations were published in Germany in the journal Archiv für Klinische Chirurgie, Berlin, just before World War I. They remained forgotten for several decades. As a reminder, Hashimoto decided to continue his training in Göttingen, Germany and then in England. It was said that he was dismissed upon his return. He continued to practice as a general practitioner in his hometown of Igamachi in the highlands west of Kyoto. He died of typhoid fever at the age of 52 years.

The description of lymphocytic thyroiditis was rediscovered in the United States in 1936, and the disease was labeled Hashimoto’s thyroiditis (chronic lymphocytic thyroiditis) in medical textbooks.

An essential step was the characterization in 1956 by Rose and Witebsky of experimental thyroiditis created by the injection of thyroid extracts and Freund adjuvant in rabbits, which showed histological aspects similar to those described by Hashimoto [2]. In the same year, Roitt and Doniach reported in the Lancet the presence of autoantibodies directed against thyroglobulin [3]. Until then it had seemed inconceivable that an individual would develop antibodies directed against his own body (“horror autotoxicus”). This was the first time that the possibility of autoimmune diseases had been suggested, and Hashimoto’s disease established itself as the model of organ-specific autoimmune diseases.
With regard to history, there is agreement that the hypertrophic forms of lymphocytic thyroiditis, which can progress to atrophy although not inevitable, would be labeled Hashimoto’s thyroiditis. In terms of the pathogenic, histologic and biologic perspectives, these forms did not differ greatly from atrophic lymphocytic thyroiditis causing myxoedema, or asymptomatic autoimmune thyroiditis or thyroiditis occurring with nodules or cancer. These aspects are the subject of the article by Orgiazzi [4]. Even before the description of lymphocytic thyroiditis by Hashimoto, the simultaneous occurrence of hypothyroidism with other endocrinopathies had been demonstrated in Germany in 1904 by Erhlich, and in 1908 in France by Claude and Gougerot. In 1980 these conditions of polyglandular failure were divided into four types by Neufeld [5], and then grouped into two varieties. Childhood-onset type 1 autoimmune polyendocrine syndrome has autosomal recessive transmission linked to a mutation of the AIRE gene, which controls the production of antibodies at the thymic and peripheral levels [6]. This can be differentiated from type 2 (or 2/3), which is much more frequent, starts in adulthood, and is polygenic and multifactorial, the characteristics of which are presented here by Kahaly [7]. Whether isolated or occurring with polyendocrinopathies, autoimmune involvement of the adrenal glands alters the quality of life and is life-threatening; it is explained and its care are described in the article by Napier and Pearce [8]. The pathogenic access and assessment of autoimmune involvement in parathyroid and pituitary are still in the early stages, while the immunologic and pathogenic data and management perspectives are better understood in diabetes mellitus, as confirmed in the article by Boitard [9].

The description of the histological changes of Hashimoto’s thyroiditis, only just a century ago, gained credibility over several decades. It is a good example of a situation in which a brilliant clinician provided the clinical and anatomopathological description of a disease, leaving it to future generations to understand the mechanism and, in the current case, to establish the far-reaching concept of autoimmune diseases, sometimes organ-specific, polyglandular or general.

Disclosure of interest: the author declares that he has no conflicts of interest concerning this article.

References