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Autoimmune Thyroiditis Revealed by a Polyalgic Syndrome

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Abstract

Autoimmune thyroid disease (AITD), also called chronic lymphocytic thyroiditis or Hashimoto thyroiditis, is an inflammatory thyroiditis that is characterized by thyroid lymphocytic infiltration. It's one of the commonest causes of hypothyroidism, that has been associated with several autoimmune diseases including rheumatologic syndromes and connectives tissue diseases and has long been known to have several manifestations particularly in association with hypothyroidism. However, in some cases, especially when thyroid function is normal, several arguments are in favor of autoimmunity mechanisms, or the presence of an overlap syndrome, rather than thyroid dysfunction. There is also considerable evidence that AITD is highly associated with polyalgic syndrome or fibromyalgia. We report a 53 years old a case, followed for bilateral symmetric chronic polyarthritis, seronegativ, non-distorting and non-destructive since 6 years, coupled with a seacca syndrome, and arthromyalgic revealing autoimmune thyroiditis.

Keywords: Autoimmune Thyroid Disease (AITD), Hashimoto Thyroiditis, Polyalgic Syndrome

1. Introduction

Hashimoto's thyroiditis is an autoimmune disease characterized by diffuse lymphocyte infiltration of the thyroid gland and antithyroid antibodies. It is responsible for either goitrous or thyroid atrophy, and frequently variable intensity dysthyroidism [1, 2]. In the past, most rheumatological manifestations were attributed to the existence of dysthyroidism. However, there are several arguments in favor of the responsibility of the mechanisms involved in autoimmunity, or the presence of a ovelap syndrome [3,4], or the systemic inflammatory reaction associated with thyroiditis. Therefore, the rheumatic manifestations associated with chronic autoimmune thyroiditis (TCAI) can be classified into three categories: Those due to dysthyroidism; those secondary to a systemic autoimmune disease or those related to anti-thyroid autoimmunity. Some evidence suggests that TCAI is associated with polyalgic syndrome. Although the diagnosis is strengthened by the composite evidence from clinical, serologic, ultrasound, histologic, and physical findings, the presence of the autoantibodies alone suggests some degree of ATD. We report a case.

2. Case Report

Mrs. H. Z is 53 years old. Diabetic since 10 years, under oral insulin therapy and antidiabetic. Hypertension under treatment. Followed in rheumatology since 6 years, for chronic polyarthritis bilateral and symmetrical, affecting the small, medium and large joints, seronegative, non-deforming and non-destructive, associated with a subjective eye dry syndrome and objective without proven Gougerot-Sjögren syndrome. The physical examination during his follow-up found again; clinical signs of bilateral carpal tunnel syndrome, retractile capsulitis of the right shoulder, and twelve points of fibromyalgia, as well as spontaneous and induced myalgia. The number of painful points was 12 and swollen to 3. The thyroid was enlarged in volume without a palpable nodule. Regarding the biological results: the CRP was at 12 mg / l, a high sedimentation rate at 100 mm / 1st hour, electrophoresis of plasma proteins was normal. The blood glucose was at 1.01g / l. The

phosphocalcic blood and urine exam was without abnormality. The immunoassay had not found anti-nuclear antibodies, either Ac Anti DNAn or citrullinated cyclic protein antibodies (ACPA). In contrast, antithyroid antibodies were positive (anti-thyroperoxidase antibodies greater than 100 IU / ml). The values of T3, T4 and THus were normal. Thyroid ultrasound showed an atrophic and heterogeneous thyroid. Nevertheless, the X-rays and the ultrasound of the hands did not objectify synovitis nor osteo-articular destruction.

The polyalgic syndrome in the context of an autoimmune thyroiditis was retained, at the base of the clinical signs, the absence of an inflammatory rheumatism or connectivite, and the absence of a dysthyroidism, the presence of the anti-Ac thyroid, and the thyroid ultrasound aspect was in favor. The patient was undergoing symptomatic treatment and monitoring for thyroiditis. As for the capsulitis retractile, the symptomatic treatment is associated with functional rehabilitation. The clinical evolution was good. Decline: 2 years.

3. Discussion

The systemic manifestations associated with chronic autoimmune thyroiditis (TCAI) may be of interest to many organs, even though the most significant lesions are articular, muscular and cutaneous [2, 5] . Joint and periarticular signs have also been observed in euthyroid patients. Some are especially; retractile capsulitis of both shoulders [2,6], erosive osteoarthritis and chondrocalcinosis [7] and their association remains a hypothesis. The most common clinical signs, reported in the L. Punzi et al series, about 139 patients and join the case of our patient [2].

The already-common phenomenon of thyroid autoimmunity appears to be magnified by the presence of other forms of autoimmunity. Possible reasons for the co-occurrence may relate to shared environmental etiologic agents as well as shared genetics between several of these autoimmune diseases [7] . However, it could also reflect the peculiar susceptibility of the thyroid gland to autoimmunity irrespective of any shared features with other autoimmune diseases. Thus, there is an increased incidence of ATD in patients with oste- oarthritis and inflammatory forms of arthritis. The incidence of well-defined connective tissue diseases is well known to be increased in patients with ATD.

The relationship between arthritis and thyroid disease has been known for over a hundred years chiefly as an association with hypothyroidism [13]. A link between the forms of arthritis described in connection with thyroid disease and TSH levels was among the first to be investigated and described. Hypothyroidism was clearly found to be associated with various forms of osteoarthritis, and the possible role of elevated levels of TSH in causing the arthritis was suggested.

The articular clinical forms are varied: Polyarthralgia isolated or associated with myalgia, acute, remittent or chronic forms [9, 10]. Punzi reports in his study [8], 20 cases of polyarthritis with polyarthritis, no rheumatic disease associated, eight with euthyroidism, two with hyperthyroidism and ten with hypothyroidism, improved after 12 months on thyroxine therapy [8]. After the first publication of Riche and Bell in 1984, several authors report a mild form of seronegative polyarthritis, resembling rheumatoid arthritis (RA), associated with TCAI in euthyroidism [2, 11, 12] Of the 33 patients included in one study [2] , eight developed a severe form of RA, characterized by bone erosions and elevated IL1 beta in synovial fluid and a high prevalence of HLA DR4. The other cases continued to evolve in a mild form of polyarthritis, non-erosive responding to symptomatic treatments. Most findings described two types of polyarthritis associated with TCAI, one suggesting RA and TCAI, the other being a subgroup of seronegative polyarthritis. These were characterized by a good sensitivity to nonsteroidal anti-inflammatory drugs.

Hyperthyroidism was noted to be associated with rheumatic manifestations much less frequently. The classically described findings included myopathy with proximal muscle weakness, atrophy, and muscle cramps. Osteoporosis of course was the best recognized endocrine complication of the disease [12].

The possibility of spinal degenerative disease has been lumped together with reports of neck pain in the literature [13]. In contrast to the distribution normally described in RA, spinal disease in these patients spared the C1-C2 articulation but affected the C3-C4, C4-C5, and C5-C6 articulations (unpublished data). These findings sug- gest a possible correlation between autoimmune thyroiditis and spinal degenerative disc disease, which warrants more detailed, controlled studies and systematic exploration. It also confirms previous reports of a very strong association with FM.

The pathophysiological mechanism of these forms of polyarthritis and TCAI is explained by some hypotheses, including the production by synovial membrane lymphocytes, such as those of the thyroid gland, antithyroid antibodies more strongly and rapidly than peripheral lymphocytes [13]. Or the existence of an immunogenetic predisposition, proven by the intervention of HLA A2, HLA DR3, and HLA DR4 [14]. Other symptoms present in thyroid diseases, particularly hypothyroidism, include muscle signs [2, 15], which may be related to a preclinical state of hypothyroidism. This translates into electron microscopy of muscle biopsies by capillary alterations and mononuclear cell infiltrates, including macrophages and rare lymphocytes [16].

Our patient illustrates a case of polyalgic syndrome during chronic autoimmune thyroiditis, grouping together frequent clinical signs, including arthralgia, seronegative arthritis, myalgia, and fibromyalgia, although they exist are less noted by all studies, represented by the carpal tunnel syndrome and the capsulitis retractile witch can be attributed in our patient to diabetes, despite the regular monitoring.

Because the autoimmune thyroid diseases are so common and because the complaints related to them are such a common finding in clinical practice we argue that the anti- thyroid autoantibodies should be part of the regular rheuma- tology workup since they inform the clinical presentation and prognosis of disease and need to be factored into any effective management plan.

It can be argued that the thyroid gland is a rather accessible organ and should be the focus of more concerted efforts to study the genetics and etiopathogenesis of organ-specific and systemic autoimmunity at the translational level and at the level of fundamental basic research. Perhaps it is time for AITD to emerge out of the shadows and no longer be seen as the “other autoimmune disease.”

4. Conclusion

There is a direct relationship between certain rheumatic manifestations and chronic autoimmune thyroiditis, divided into three categories: those due to dysthyroidism, mainly hypothyroidism; those secondary to a systemic autoimmune disease associated, or that related to antithyroid autoimmunity, as the case of our observation. Patients with chronic autoimmune

thyroiditis may progress over time, from a minor form to a complete disease, through an evolution that begins with a subclinical stage.

Interest Conflict

All the authors do not have any possible conflicts of interest.

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