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Multiple Sclerosis and Hashimoto's Thyroiditis: A Case Report

Multipl Skleroz ve Haşimoto Tiroiditi: Bir Olgu Sunumu

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ABSTRACT

It has been suggested that multiple sclerosis (MS) is an immune-mediated disease directed against central nervous system (CNS) myelin, although the exact pathogenesis is still unclear. There are a lot of reports regarding the coincidence of MS with other immune-mediated diseases. In recent years, there have been only few case reports of MS associated with Hashimoto's thyroiditis. This coincidence is especially important from both clinical and therapeutic standpoints. In this case report, we present a patient with MS and Hashimoto's thyroiditis and summarize the clinical features. *(Archives of Neuropsychiatry 2011; 48: 224-6)*

Key words: Multiple sclerosis, autoimmune disease, Hashimoto's thyroiditis

ÖZET

Multipl skleroz'un (MS) patogenezi tam aydınlatılmamış olmakla birlikte santral sinir sisteminin (SSS) miyelinine yönelik immun aracılı bir hastalık olduğuna inanılmaktadır. MS'le birlikte diğer immun aracılı hastalıkların birlikte görüldüğüne dair çok sayıda rapor bulunmaktadır. Son yıllarda, MS'in, Haşimoto tiroiditi ile birlikte görüldüğüne dair de birkaç adet vaka raporu bulunmaktadır. Klinik ve tedavi açısından bu birliktelik önem kazanmaktadır. Bu olgu sunumunda, MS ve Haşimoto tiroiditi olan bir olgu aktarılmış ve bu olgunun klinik özellikleri özetlenmeye çalışılmıştır. *(Nöropsikiyatri Arşivi 2011; 48: 224-6)*

Anahtar kelimeler: Multipl skleroz, otoimmun hastalık, Haşimoto tiroiditi

Introduction

Autoimmune diseases share a number of characteristics that suggest common etiologic pathways or mechanisms. Multiple sclerosis (MS) is associated with complex abnormalities of immunoregulation and a role of autoimmunity in its pathogenesis has been accepted. MS is thought to be due to T helper type 1 cell (Th1)-mediated autoimmunity, along with diseases like rheumatoid arthritis, type 1 diabetes mellitus, autoimmune (Hashimoto's) thyroiditis, inflammatory bowel disease and psoriasis, and all of these have been reported in MS patients at increased rates compared to controls (1-9).

In the view of the rare reports in recent years, the coexistence of MS and Hashimoto's thyroiditis is of special interest. Previous clinical and experimental observations suggest that thyroid hormones and the thyroid gland may be of clinical relevance in MS. Thyroid hormones are critical for CNS myelination and may be important for remyelination (10,11). In addition, fatigue, muscle weakness and depression are common and disabling symptoms in both MS and hypothyroidism (12,13). Also, there are some reports indicating that MS patients on interferon-beta (IFN-beta) and Campath-1H treatment may have higher occurrence of anti-thyroid autoantibodies and autoimmune thyroiditis (14-17). In contrast, recently Durelli et al. reported only random and insignificant changes in the frequency of thyroid dysfunction and autoimmunity over time, probably not related to IFN-beta (18).

In this case report, we summarize the clinical and laboratory findings of a patient with MS and Hashimoto's thyroiditis to emphasize again the importance of the thyroid function tests, which should be performed in MS patients with or without any complaints in order to investigate the possibility of coexistence and to assess the need for immunomodulatory therapy.

Case Report

A 37-year-old obese and hypertensive woman, who had headaches once in a month for almost 6 years, had been

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admitted to a neurosurgery clinic in June 2005. At that time, cranial MRI showed hyperintense signal abnormality in the corpus callosum and in the pericallosal areas on T2-weighted images.

In January 2006, she presented with vertigo, diplopia, ataxia and blurred vision in the left eye, which improved with pulse corticosteroid therapy for 10 days (1g/day methylprednisolone). Cranial MRI was performed, showing hyperintense areas in the periventricular white matter and a hyperintense, contrast enhancing lesion in the left frontal lobe on T2-weighted sequences.

In February 2006, she was admitted to our clinic for her headaches without any other complaints. She denied having any skin, oral or genital ulcerations. At that time, EDSS score was 0.0 (19). Visual and somatosensorial evoked potentials (VEPs and SEPs) were normal. The laboratory tests performed for the differential diagnosis were all negative (vitamin B12, anti-HIV, VDRL-RPR/TPHA, protein C, protein S, antithrombin III, lupus anticoagulants, factor V Leiden mutation, anti-histone ab, anti-nucleosome ab, anti-Sm ab, anti-U1 snRNP ab, anti-SS-A ab, anti-SS-B ab, anti-Scl-70, anti-centromere ab, anti-Jo1 ab, ANA , anti-ds DNA , anti-cardiolipin ab IgM and IgG).

In June 2006, diplopia and blurred vision developed and she was admitted to the hospital; the symptoms recovered spontaneously within 10 days. Cranial MRI showed 3 more contrast enhancing lesions. The IgG index was 0.28 and oligoclonal bands were negative.

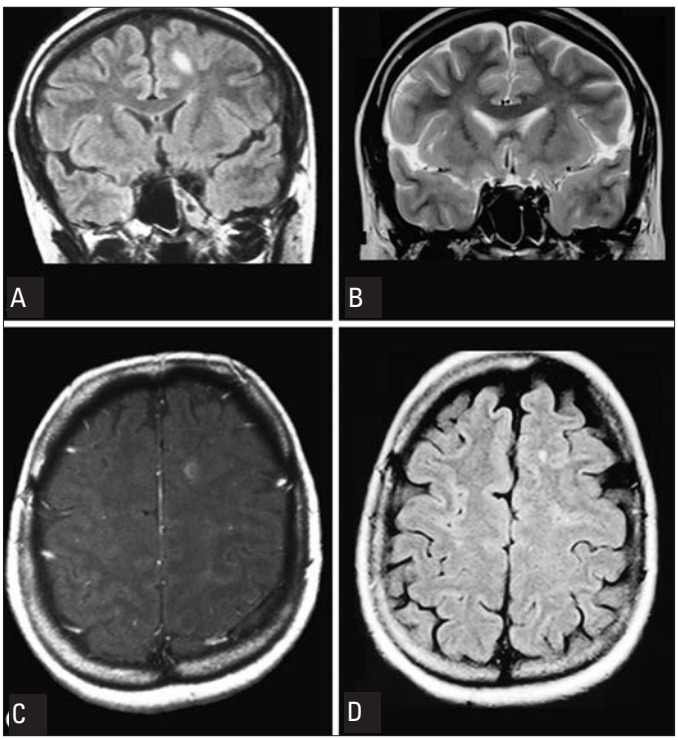

Figure 1. In June 2006, cranial MRI showed left frontal hyperintensity in coronal FLAIR images (A) and open ring contrast enhancing lesion in left frontal subcortical area in postcontrast T1-weighted images (C). In October 2007, the lesions in the previous MRI were regressed in the similar sections in coronal T2-weighted images (B) and in axial T2-weighted images (D)

In July 2006, the patient presented with blurred vision in the left eye and ataxia. EDSS score was 3.0. MRI showed 3 more contrast enhanced lesions compared to the recent MRI (Figure 1). VEPs and SEPs were abnormally prolonged in the left side. Thyroid function tests were as follows: TSH: 9.75 µIU/mL (0.35-0.9µIU/mL); T4: 0.67 µg/dL (4.87-11.72 µg/dL) with positive antithyroid antibodies (antithyroglobulin antibody, 4.84 IU/mL, antiperoxidase antibody, 1000 IU/mL). The symptoms improved with pulse therapy (1g/day methylprednisolone), and afterwards thyroid hormone administration for Hashimoto's thyroiditis was given.

In October 2007, she had no complaints and EDSS score was 0.0. Cranial MRI showed 2 more new lesions, but none of the lesions were contrast enhancing and the lesions on the previous MRI had regressed (Figure 1).

Discussion

Our case was diagnosed with clinical definite MS according to the McDonald's criteria (20) with clinically observed two attacks, lesions fulfilling the criteria of dissemination in time and space on MRI, and the abnormal VEPs and SEPs despite the absence of oligoclonal bands in the CSF. Also, the history and the laboratory tests for the differential diagnosis, which were all negative, supported our diagnosis.

Hashimoto's thyroiditis is one of the most frequent causes of primary hypothyroidism (17, 21-23). It is more common in women (female-to-male ratio, 8:1) and is most frequent at 30 to 50 years of age (23). Hashimoto's thyroiditis and MS are most frequently associated in women, as seen in our case (23). In prospective studies, autoimmune thyroid disorders are 3-fold more common in MS female patients compared with healthy women (23). The association of MS with Hashimoto's thyroiditis is also important from clinical and therapeutic standpoints. Symptoms such as proximal muscle weakness, myalgia, and fatigue are common both in MS and in hypothyroidism (12,13). In our case, the patient did not have any complaints related to thyroid disease and the thyroid abnormality was found in routine laboratory investigation. Therefore, it is important to perform thyroid function tests with or without such symptoms for the possibly coexisting Hashimoto's thyroiditis in MS patients. An important issue that should be addressed is the use of immunomodulatory therapy. The thyroid hormone levels of MS patients being treated with INF-beta and Campath-1H also should be monitored (24).

Previous clinical and experimental observations have suggested that thyroid hormones may be of clinical relevance in MS, because they are critical for CNS myelination and may play an important role in remyelination; whether thyroid hormones influence the remyelinating ability of oligodendrocyte precursors in especially early (fresh) demyelinating MS lesions is an important yet unresolved aspect of the role of the endocrine system on MS (25). In our case, in a two-year period, she had clinically observed two attacks with the increase of the contrast enhancing lesion load on subsequent MRIs. We could not use an immunomodulatory therapy, although she had an immunologically active disease, because of the patient's socio-economic status. For this reason, she received only

medication for hypothyroidism other than the attack therapy with steroids. During this period of time, we did not observe any attack or progression in the clinical course and a relative stationary state in active radiological lesions, which according to the shared etiopathogenesis of both diseases, could supposedly be related to the remyelinating effect of thyroid hormones. Further studies are required to show the effect of thyroid hormones on clinical course of MS and the influence on MS lesions.

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