

Riedel's thyroiditis in a patient with multiple sclerosis

Pirkko Kotilainen¹, Laura Airas², Tiina Kojo¹, Timo Kurki³, Kaisa Kataja¹,
Heikki Minn⁴ & Pirjo Nuutila¹

Departments of ¹ Medicine, ² Neurology and ³ Neuroradiology,
Turku University Central Hospital, Turku, FINLAND, and
⁴ Turku PET Centre, University of Turku, Turku, FINLAND

Correspondence to: Prof. Pirkko Kotilainen MD, PhD
Department of Medicine, Turku University Central Hospital,
Kiinamylynkatu 4-8, 20520 Turku, FINLAND
PHONE: +358 2 3130000
FAX: +358 2 3132030
EMAIL: pirkko.kotilainen@utu.fi

Submitted: September 2, 2004

Accepted: October 7, 2004

Key words: **multiple sclerosis; Riedel's thyroiditis**

Neuroendocrinol Lett 2005; **26**(1):67-68 PMID: 15726023 NEL260105C01 © Neuroendocrinology Letters www.nel.edu

Abstract

Our patient developed Riedel's thyroiditis soon after having an exacerbation of multiple sclerosis (MS). MS has been associated with other autoimmune diseases, including thyroiditis, and both Hashimoto's thyroiditis and subacute thyroiditis have been described in connection with MS. Yet, we are not aware of any other patient reported to have concomitant MS and Riedel's thyroiditis. The association between MS and Riedel's thyroiditis remains obscure but may reflect an autoimmune disorder common to both diseases.

Abbreviations:

MS = Multiple sclerosis

Introduction

Multiple sclerosis (MS) has been associated with other autoimmune diseases, including thyroiditis [3]. Both Hashimoto's thyroiditis and subacute thyroiditis have been described in connection with MS [7,4], but there are no reports on concomitant MS and Riedel's thyroiditis. Interferon- β is used as long-term treatment for MS, and some risk of thyroid dysfunction seems also to be involved with interferon- β [6]. Moreover, treatment of MS patients with anti-CD52 monoclonal antibody (Campath-1H) caused antibody-mediated thyroid autoimmunity in one third of patients [1].

Riedel's thyroiditis, or invasive fibrous thyroiditis, is a rare disease with an unknown etiology commonly considered to be autoimmune [2]. It may occur as an independent disease entity or as one

component of multifocal fibrosclerosis, the other manifestations including e.g., retroperitoneal fibrosis, sclerosing cholangitis, and fibrous changes of the mediastinum and lungs [2]. Our patient developed Riedel's thyroiditis soon after having an exacerbation of MS.

Case

A 41-year-old woman presented with paresthesia in her right side and lower extremity, and disturbances in the functions of the urinary bladder and bowel. The patient had been healthy until the delivery of her first child 16 months earlier, after which she had suffered from fatigue and weakness of her left leg and both hands for two months. On

admission, MRI revealed white matter lesions highly suggestive of demyelination periventricularly in the brain, and in the spinal cord at the CII–III level and ThVI–VII level. The major lesion in the brain was one centimeter in diameter; and the major lesion in the thoracic cord, two centimeters in length. Cerebrospinal fluid examination was normal except for a slightly increased IgG concentration. On a tentative diagnosis of MS, she was administered a high-dose intravenous methylprednisolone treatment. The bladder and bowel functions returned to normal, but paresthesias remained.

Three months later, the patient was readmitted with a large, hard thyroid mass with several nodules on the right side of her neck. Histological examination of the thyroid gland after palliative thyroid operation revealed dense fibrous tissue associated with scattered foci of lymphocytic cells, which verified the diagnosis of Riedel's thyroiditis. High-dose intravenous methylprednisolone therapy resulted in a rapid clinical response of her thyroid disease.

The clinical neurological picture with two neurological attacks typical for MS-disease, and MRI findings of brain and spinal cord were highly suggestive of active, relapsing-remitting MS-disease, and glatiramer acetate treatment was commenced. The patient experienced a new clinical relapse with weakness of her left leg three months later, and a control MRI of the brain showed new, active gadolinium-enhancing lesions typical of MS-disease. Moreover, the clinical examination revealed problems with coordination and balance. In neuropsychological testing there were slight defects in cognitive function. Subsequently, azathioprine was commenced to prevent exacerbations of MS-disease, and to treat the thyroid disorder.

Discussion

With the clinical presentation and the MRI findings of our patient, the diagnostic criteria for MS-disease are fully met [5]. In addition to MS-disease, our patient suffers from Riedel's thyroiditis, which started in a fulminant way shortly after an exacerbation of MS-disease. The temporal sequence of events suggests that the exacerbation of MS-disease could have triggered the onset of Riedel's thyroiditis.

An association between MS and other thyroid diseases has been established previously. According to one recent study [3], thyroid disorders were at least three times more common in women with MS than in female controls; hypothyroidism mainly accounted for the high rate of thyroid diseases among the female MS patients. The authors postulated that because the majority of hypothyroidism is due to Hashimoto's thyroiditis, the association of MS with hypothyroidism might indicate a shared susceptibility to autoimmunity. [3] Also the common development of autoimmune thyroid disease in patients with MS during treatment with Campath-1H supports the view of unique susceptibility to this complication, since Graves' disease has not been reported in patients treated with Campath-1H for various other disorders [1]. Moreover, attention has been focused on

the common occurrence of thyroid disorders in patients with MS, who are treated with interferon- β . In one study, a 33% frequency of thyroid dysfunction was observed in a series of 33 patients with MS during one-year treatment with interferon- β [6]. Hence, thyroid assessment has been recommended during the first year of the therapy. The pathogenetic mechanisms of interferon- β -induced thyroid disorders are not understood, but the risk appears to be related to preexisting thyroiditis. Because of the thyroid condition, glatiramer acetate rather than interferon- β treatment was commenced as immunomodulatory treatment for our patient.

The association between MS and Riedel's thyroiditis in our patient remains obscure but may reflect an autoimmune disorder common to both diseases.

REFERENCES

- 1 Coles AJ, Wing M, Smith S, Coraddu F, Greer S, Taylor C, Weetman A, Hale G, Chatterjee VK, Waldmann H, Compston A. Pulsed monoclonal antibody treatment and autoimmune thyroid disease in multiple sclerosis. *Lancet* 1999; **354**:1691–5.
- 2 Hay ID. Thyroiditis: a clinical update. *Mayo Clin Proc* 1985; **60**:836–43.
- 3 Karni A, Abramsky O. Association of MS with thyroid disorders. *Neurology*. 1999; **53**:883–5.
- 4 Kreiss Y, Cohen O, Pras E, Achiron A. Subacute thyroiditis in a patient with MS treated with interferon beta-1a. *Neurology* 1999; **53**:1606.
- 5 McDonald WI, Compston A, Edan G, et al. Recommended diagnostic criteria for multiple sclerosis: guidelines from the International Panel on the diagnosis of multiple sclerosis. *Ann Neurol* 2001; **50**: 121–7.
- 6 Monzani F, Caraccio N, Meucci G, Lombardo F, Moscato G, Casolaro A, Ferdeghini M, Murri L, Ferrannini E. Effect of 1-year treatment with interferon- β 1b on thyroid function and autoimmunity in patients with multiple sclerosis. *Eur J Endocrinol* 1999; **141**:325–31.
- 7 Roquer J, Escudero D, Herraiz J, Maso E, Cano F. Multiple sclerosis and Hashimoto's thyroiditis. *J Neurol* 1987; **234**:23–4.