Turkish Journal of Endocrinology and Metabolism, (2004) 4 : 139-141 **CASE REPORT**

Dysarthria Associated with Hashimoto's Disease in A Type 1 Diabetic Patient

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We report a diabetic patient seen at our department, presenting with worsening dysarthria. The patient was a 25- year- old woman. On examination the patient had a palpable thyroid gland (grade 1b), and the Romberg test was mildly impaired. Her thyroid profile showed an increased level of TSH, and a decrease in the levels of free T3 and free T4-findings consistent with hypothyrodism. Serum levels of antithyroid antibodies (TG-Ab and TPO-Ab) were increased. Ultrasonographic appearance of the thyroid gland was diffuse and hypoechoic. A brain CT showed cerebellar atrophy. Inasmuch as the findings of the patient were consistent with Hashimoto's disease and progressive non-familial adult onset cerebellar degeneration, L-thyroxine treatment was begun. Dysarthria was observed to resolve on follow-up visits following the maintenance of the L-thyroxine treatment.

Key words: **Dysarthria, Hashimoto's disease, Diabetes mellitus**

Case

In this paper, we report a female patient presenting with dysarthria. She was 25 years old. She had a ten year history of type 1 diabetes mellitus and she had been taking insulin. She also noticed a slight impairment in her coordination while buttoning up clothes and writing. She did not consume alcohol. She had three sisters who were suffering from diabetes mellitus. There was no family history of similar neurologic symptoms.

The patient had a palpable thyroid gland. She was hypothyroid at the presentation. The neurological examination disclosed a normal mental status. The Romberg test was mildly impaired. The patient's language was intact, but her articulation was dysarthric. Her muscle tone and power were normal. The patient's deep tendon reflexes and primary sensory modalities were intact throughout. Table 1 shows the thyroid profile of the patient.

Her thyroid profile showed increased TSH, and decreased both free T3 and free T4. Either thyroglobulin antibody (TG-Ab) and thyroid peroxydase antibody (TPO-Ab) were increased. The serum biochemistry tests and complete blood count were unremarkable. The ESR was 30 mm/hour. Antinuclear antibody was negative. Serum vitamin-B12 and folic acid were normal (312 pmol/L and 8.4 nmol/L respectively). Serum protein electrophoresis was normal (Albumin, alfa-1 globulin, alfa-2 globulin, beta-1 globulin, beta-2 globulin and gamma globulin 54.0, 3.2, 12.1, 6.3, 5.3 and 18.3 percent respectively).

A thyroid scan demonstrated a decreased ^{99m}Tec uptake. The ultrasonographic appearance of the thyroid gland was diffuse and hypoechoic. The cranial CT showed cerebellar atrophy (Figure 1).

The findings of the patient were consistent with Hashimoto's disease and progressive non-familial adult onset cerebellar degeneration (PNACD) (2), thus L-thyroxine treatment was begun.

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Table 1. Thyroid function tests on admission.

	TSH (0.27-4.2) µIU/mL	Free T4 0.93-1.7 (ng/mL)	Free T3 1.8-4.6 (pg/mL)	TG-Ab 0-35 (IU/mL)	TPO-Ab 0-60 (IU/mL)
Results	11.8	0.22	1.35	520	1080

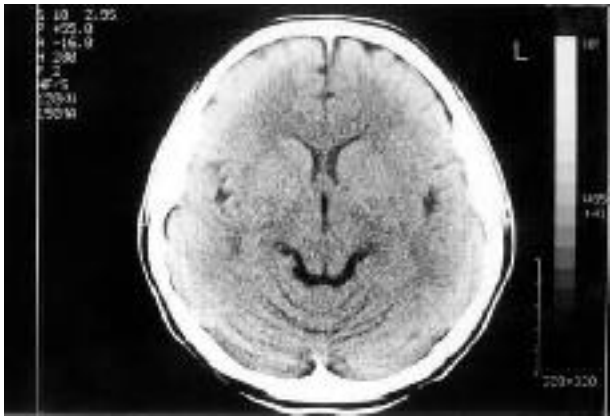

Figure 1. Brain CT of the patient.

Discussion

Some conditions are well recognised to produce PNACD, including nutritional deficiencies, intoxication, hypoxia, hyperthermia, paraneoplastic syndromes, systemic lupus erythromatosus, late onset diabetes mellitus and olivopontocerebellar degeneration of unknown cause. Nevertheless, the exact mechanisms by which this syndrome might be produced are unclear. In most reported cases, cerebellar syndrome has been reversed by thyroid replacement therapy, suggesting that it was caused by metabolic and physiological effects of the hormonal deficiency. In some patients, however, despite thyroid replacement therapy the cerebellar syndrome may persist (3-5). On the other hand, thyroid autoimmunity is the most prevalent immunological process affecting patients with type 1 diabetes (6).

In this paper we report a patient who had type 1 diabetes mellitus and Hashimoto's thyroiditis presenting with dysarthria. On admission the patient was mild hypothyroid. The cranial CT of the patient revealed cerebellar atrophy (Figure 1).

Hashimoto's thyro iditis is characterised by lymphocytic infiltration of the thyroid gland and circulating antithyroid antibodies to thyroglobulin or thyroid peroxidase, which are present in 70%-95% of patients (7). Increased levels of anti-TPO were described in several autoimmune disorders. A range of autoimmune diseases has been associated with Hashimoto's thyroiditis including rheumatoid arthritis, systemic lupus erythromatosus, ulcerative colitis, pernicious anemia, myastenia gravis, multiple sclerosis, and thyroid ophtalmopathy (8). An encephalopathy has also been reported in association with subclinical Hashimoto's thyroiditis (9).

Autoimmune mediated cerebellar degeneration is a more likely mechanism in Hashimoto's autoimmune thyroiditis, and thyroid replacement therapy may not reverse the symptoms related with cerebellar involvement (2). According to the maintenance of adequate thyroid hormone replacement therapy, our patient became euthyroid and dysarthria reversed.

There are several mechanisms by which autoimmunity associated with Hashimoto's thyroiditis might induce cerebellar degeneration. As widespread autoimmune reactivity can be seen in patients with Hashimoto's disease, cerebellar degeneration may be mediated by another unidentified circulating antiPurkinje cell antibody. In this case, the autoimmune thyroiditis would be serving as a marker, identifying the presence of a more generalised autoimmune disorder involving the cerebellum. Alternatively, if an immunological cross reactivity exists between shared thyroid and cerebellar antigens, antithyroid antibodies could specifically affect the cerebellum. Abnormalities of glutamic acid decarboxylase (GAD) enzyme have been demonstrated in tissues from patients with olivopontocerebellar atrophy and GAD antibodies have been detected in some patients with cerebellar cortical atrophy and other cerebellar degenerative diseases. A relation between GAD antibodies and type I diabetic patients is also known (10). On the other hand, thyroid autoimmunity may be diagnosed either at the onset of diabetes or during the follow up (11). These immunological and clinical profiles are

similar to the clinical presentation of our patient. However, any relation with dysarthria and diabetes mellitus in this patient is not clear.

The present report may indicate the association between dysartria, which is probably due to cerebellar disorder, and Hashimoto's thyroiditis, but it is obscure which mechanisms play role in the development of dysarthria.

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