Cerebellar Ataxia as the Presenting Feature of Hypothyroidism

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ABSTRACT

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Symptoms and signs of the hypothyroidism vary in relation to the magnitude and acuteness of the thyroid hormone deficiency. The usual clinical features are constipation, fatigue, cold intolerance and weight gain. Rarely it can present with neurologic problems like reversible cerebellar ataxia, dementia, peripheral neuropathy, psychosis and coma. We present the case of a 56 year old male who came to us with complaints of gait ataxia of 1 month duration with no sensory and labyrinthine pathologies. Investigations revealed frank hypothyroidism and patient improved dramatically with thyroxine supplementation.

Keywords: Hypothyroidism, Gait ataxia, Reversible ataxia, Progressive non familial adult onset cerebellar degeneration

*See End Note for complete author details

INTRODUCTION

Some conditions are well recognised to produce progressive non-familial adult onset cerebellar degeneration (PNACD), including nutritional deficiencies, intoxication, hypoxia, hyperthermia, paraneoplastic syndromes, and olivopontocerebellar degeneration of unknown cause. Hypothyroidism has been described as a cause of gait ataxia, presumably due to cerebellar dysfunction, although the exact mechanism(s) by which it might produce this syndrome is unclear. In most reported cases, the ataxia has been reversed by thyroid replacement therapy, suggesting that it was caused by the metabolic and physiological effects of the hormonal deficiency.

CASE REPORT

56 year old male patient was referred to us with complaints of gradual onset of unsteadiness of gait of 1 month duration. It was gradually progressive in nature. There was no history of vertigo, dysarthria, weakness of limbs or any bulbar symptoms. He was not an alcoholic and had an uneventful past medical history

On examination he had stable vitals. Neurological examination revealed gait ataxia alone with no other cerebellar signs. Tandem walking was impaired and the gait was wide based. Motor system and sensory system was within normal limits.ENT evaluation ruled out any labyrinthine pathology.

Routine blood investigation was within normal limits. Serum vitamin B 12 levels were within normal limits. There were no electrolyte abnormalities. CT Brain ruled out structural and cerebrovascular accident. Screening for HIV was negative. In further evaluation of cerebellar ataxia, thyroid function tests were ordered **(Table 1).**

Table 1. Thyroid Function Tests

Test	Result
T 4	0.67 ng/ml[4.5-11]
T3	0.410ng/ml[0.7-2.0]
TSH	>100mcIU/ml
Anti tpo	655

He was started on 100 ug thyroxine and he made a complete recovery in 2 weeks

DISCUSSION

Hypothyroidism is one of the causes of acute onset ataxia. Stroke, viral encephalitis and drugs can also cause acute cerebellar ataxia. Mass lesions in the posterior fossa, infections such as HIV, and vitamin deficiencies like B1 and B12, alcohol and paraneoplastic syndromes

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are causes of sub acute onset cerebellar ataxia in an adult.[1]

There were two separate mechanisms resulting in cerebellar dysfunction in patients with thyroid disorders. In hypothyroidism not associated with auto-immunity, the endocrine disorder produces cerebellar dysfunction that could be reversed by thyroid replacement. In patients with autoimmune thyroiditis not reversed by thyroid replacement therapy, auto-immune mediated cerebellar degeneration was a likely mechanism.[2]

Physiological reduction of cardiac output, cerebral blood flow, and reduced oxygen and glucose consumption by cerebellar neurons has been suggested.[3] Adamset al[4] favoured slowed muscle relaxation (pseudomyotonia) over cerebellar dysfunction as the cause of ataxia in hypothyroidism.

The other neurological manifestations of hypothyroidism include Holfman syndrome, pseudomyotonia, Myxedema, Wottmans sign, Entrapment Neuropathies and Hashimotos encephalopathy and defective hearing.[5]

The present report highlights the association of progressive non-familial adult onset cerebellar degeneration with Hashimoto's autoimmune thyroiditis, signalled by increased TPO-Ab and evidence of more general autoimmunity. We recommend testing for antithyroid antibodies and other evidence of autoimmunity in patients who present with PNACD. Some authors suggest that immune modulation therapies, such as plasmapheresis, IVIg, or immuno-suppressive agents, deserve a trial in these patients.[2]

END NOTE

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Editor's Remarks: The case report discusses a rare presentation in Hypothyroidism. The causative mechanisms are also discussed.

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