



## Autoimmune Thyroid Disease Presenting as Ataxia

Authors

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### Abstract

*Besides typical clinical features, hypothyroidism can present with neurological features like reversible cerebellar ataxia, dementia, peripheral neuropathy, coma, etc. We present a case of 40 year old female patient presented with complaints of symmetrical and progressive unsteadiness while walking, dysarthria for a period of two weeks. On examination scanning speech present, dysmetria on finger-nose test and heel-knee test, dysdiadokinesia present, tandem gait impaired. On evaluation CSF analysis was normal, MRI brain plain and contrast was normal. TSH was 150  $\mu$ IU/L, anti TPO antibodies were 684 IU/ml. She was diagnosed as autoimmune thyroid disease with hypothyroidism and was given levothyroxine supplementation. she improved symptomatically. Hypothyroidism should be considered in all cases of cerebellar ataxia as it is a potentially treatable and reversible cause of ataxia.*

**Keywords:** Autoimmune thyroid disease, hypothyroidism, cerebellar ataxia.

### Introduction

Hypothyroidism is a common problem in clinical practice. Common systemic manifestations include fatigue, cold intolerance, constipation, weight gain, decreased appetite, hair loss, dry skin and hoarseness. A variety of central and peripheral nervous system manifestations are common in patients with hypothyroidism like reversible cerebellar ataxia, dementia, peripheral neuropathy, coma, etc. Most of these complications are partially or fully responsive to thyroxine replacement<sup>(1)</sup>. There is scarce recent published data on hypothyroidism causing ataxia. Here we present a case of hypothyroidism presenting with cerebellar ataxia.

### Case Report

We present a case of 40 year old female patient non hypertensive, non diabetic, non alcoholic and non smoker presented with complaints of symmetrical and progressive unsteadiness while walking, dysarthria for a period of two weeks. History of constipation, cold intolerance from few months. No history of fever, rash, headache, vomitings, loss of consciousness, weakness in any part of body, trauma or recent vaccination. No history of any drug usage or toxic exposure. No significant past or family history. On examination vitals were stable; no thyroid swelling or dry skin. On neurological examination, cognitive functions were normal. Cranial nerves were intact, motor system showed delayed relaxation of reflexes, sensory system intact, cerebellar system: no nystagmus, no tremor, scanning speech present,

dysmetria on finger-nose test and heel-knee test, dysdiadokinesia present with wide based gait.

On evaluation her complete blood picture with peripheral smear, renal function test, liver function test, serum electrolytes, serum cortisol and CSF analysis were normal. Ultrasound abdomen and chest x ray were normal. MRI brain plain and contrast was normal. Viral markers were negative, serum vitamin B12 was 290 pg/ml. TSH was 150  $\mu$ IU/L, anti TPO antibodies were 684 IU/ml. She was diagnosed as Autoimmune thyroid disease with hypothyroidism causing ataxia and started on levothyroxine 75  $\mu$ g. Patient improved symptomatically and on 3 months follow up her TSH was 3.2  $\mu$ IU/L.

### Discussion

Hypothyroidism is one of the causes of acute onset ataxia. Stroke, viral encephalitis and drugs can also cause acute cerebellar ataxia<sup>(2)</sup>. Mass lesions in the posterior fossa, infections such as HIV and vitamin deficiencies like B1 and B12, alcohol and paraneoplastic syndromes are causes of sub acute onset cerebellar ataxia in an adult. Hypothyroidism has been recognized as a cause of gait ataxia.

In early case series of patients with adult-onset hypothyroidism, a wide-based gait ataxia was a prominent feature in a considerable number, around 10 to 30% of cases. In many cases, this was the presenting feature, and variably included clumsiness, intention tremor, dysmetria and dysarthria<sup>(3)</sup>. Treatment of the hypothyroid state led to improvement or resolution of the cerebellar features. The pathogenesis of cerebellar dysfunction in patients with decreased thyroid function is uncertain. Restoring a euthyroid state with L-thyroxine has reversed the cerebellar symptoms in most patients, suggesting that their symptoms were due to endocrine mediated dysfunction of the cerebellum<sup>(4)</sup>. Physiological reduction of cardiac output, cerebral blood flow, and reduced oxygen and glucose consumption by cerebellar neurons has been suggested. Cerebellar degeneration in patients with raised antithyroid

antibodies may be immune mediated. Some cases of hypothyroidism associated cerebellar ataxia do not reverse with normalization of their thyroid state with thyroid hormone replacement and may be harboring undiagnosed Hashimoto's thyroiditis<sup>(5)</sup>. Our case presented with progressive cerebellar gait ataxia with response to thyroxine supplementation.

### Conclusion

Hypothyroidism presenting as cerebellar ataxia is rare. Profound signs of hypothyroidism may or may not be present. Hypothyroidism should be considered in all cases of cerebellar ataxia as it is a potentially treatable and reversible cause of ataxia.

### References

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