Hypothyroidism masquerading as cerebellar ataxia

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Abstract
Cerebellar ataxia can be a rare possible presentation of autoimmune hypothyroidism. We report a case of a young 20 years old female, who presented with bilateral cerebellar ataxia without any other significant neurological features. In detail investigations, she was discovered to be a case of Hashimoto Thyroiditis with positive anti-thyroid peroxidase (TPOAb) antibody and low freeT4 and freeT3 levels. She improved dramatically with levothyroxine therapy and iv pulse methylprednisolone. Thus, hypothyroidism should be looked for as a reversible and treatable cause in all cases of bilateral cerebellar ataxia.

Keywords: Hypothyroidism, Autoimmune, Cerebellar ataxia, TPOAb.

Introduction
Cerebellar ataxia is well known to be caused by a long list of infections, demyelination, metabolic factors, vascular insults, nutritional deficiency, paraneoplastic syndromes and some toxins. Hypothyroidism has been described as one of the reversible causes of cerebellar ataxia, although the exact pathophysiology is not well known.1,5 Hashimoto’s thyroiditis has been described as a chronic inflammation of the thyroid gland and the most common cause of hypothyroidism.6 The ataxia has been observed to improve by thyroid replacement therapy in many reported cases, so possibly it could be due to the physiological effects of thyroid hormone deficient state.7,9 However, in many cases, the prognosis of the ataxia has been bad even after the hormonal therapy.1,3,7 We report a case of a young lady presenting with subacute onset bilateral cerebellar ataxia, which on detailed work up, found to be caused by autoimmune hypothyroidism. She was treated with levothyroxine and corticosteroids, showed remarkable improvement.

Case Report
A 20 years old, normotensive, nondiabetic right handed lady presented gait ataxia of about two months duration. First of all, she noticed irregularity in her writing two months back then over a span of one month, she discovered that she was facing difficulty in buttoning up her clothes and she has developed smearing of face while eating. Rapidly in a month, she became grossly ataxic with a tendency to fall while walking. There was no history of any loss of consciousness, fever, convulsion, headache, visual complaints or any other neurological symptoms. She gave no history of sensory disturbances and her bowel & bladder function was normal. Besides this, she also had three months of secondary amenorrhea. Her other medical history was unremarkable. She is the second child of her parents with no history of any consanguinity. Her parents, one elder brother all are healthy without any history of neurological or endocrine disorders.

General physical examination was normal. Neurologically, higher mental functions were intact. A horizontal nystagmus was present on lateral gaze bilaterally, she showed marked dysmetria in both upper and lower limbs. She also had dysdiadochokinesia. She had normal muscle bulk with hypotonia and slight distal weakness (4+/5) in all limbs. Deep tendon reflexes were normal except diminished at the ankles. Plantar reflexes were flexor bilaterally. Sensory examination revealed no abnormality. Her gait was broad based and reeling to either side. Rest of the examination was normal.

She was investigated thoroughly for major causes of sub acute bilateral cerebellar ataxia. MRI brain with screening of spine with contrast was done which was normal (Fig. 1). TSH was raised(31Uiu) and FT4(2ug/dl), FT3 (40ng/dl) were low, further she was tested for anti TPO Ab which was strongly positive. Her thyroid ultrasound revealed tiny colloid cysts. Her ESR was 85 mm/hour. Her hemoglobin was 10.5 gm% with HCT 31.2% and MCV 109, though her Vit B12 and folate was normal. Other routine liver function test and renal parameters including serum electrolytes were normal. Possible viral infections including HSV, CMV, HIV were ruled out by serology. VDRL, TPHA for syphilis was negative and vasculitic panel including ANA, cANCA, pANCA was also negative. Analysis of CSF for ADA, VDRL, Oligoclonal bands (OCB) & IgG Index was also normal. NCS of crossed limbs revealed no abnormality. She investigated for paraneoplastic antibodies which were negative. She even tested negative for anti-gliadin antibody.
Discussion

Hypothyroidism is not now in literature as a reversible cause of cerebellar ataxia. However, the pathogenesis of cerebellar ataxia in hypothyroid patients has been investigated in detail. A case of cerebellar ataxia has been reported in literature in association with Hashimoto's thyroiditis. It has been suggested that in hypothyroid state, there is a reduction of cellular oxygen and glucose consumption by cerebellar neurons which paramount to pathological changes in the cerebellum. It has been observed that in hypothyroid patients, there has been a possible association of cerebellar ataxia and autoimmune thyroid disease. Studies demonstrate up to 14 percent of children and adolescents with congenital hypothyroidism show one or the other findings of cerebellar ataxia. They may be positive for thyroglobulin and thyroid peroxidase antibodies in all cases presenting with acute onset cerebellar ataxia. There is an association of thyroxine and cerebellar degeneration in one hypothesis. In another hypothesis, it has been suggested that in hypothyroid state, there is a reduction of cellular oxygen and glucose consumption by cerebellar neurons which cause pathological changes in the cerebellum. It has been suggested that in hypothyroid state, there is a reduction of cellular oxygen and glucose consumption by cerebellar neurons which cause pathological changes in the cerebellum.

Conclusion

We report a case of bilateral cerebellar ataxia in a young lady who had hypothyroidism presenting as acute onset cerebellar ataxia. She was started on thyroxine replacement therapy. Her symptoms improved dramatically for three days to which she responded to methylprednisolone for three days. Her thyroid profile also stabilized. On long-term follow-up of one year, there was no recurrence of symptoms.

References