Case Report

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Neurology Section

Doctor I Am Swaying: An Interesting Case of Ataxia

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ABSTRACT

Auto-immune Hypothyroidism is one of the common causes of hypothyroidism. The usual clinical features of auto-immune hypothyroidism are constipation, fatigue, cold intolerance and weight gain. Rarely it can present with neurologic problems like reversible cerebellar ataxia, dementia, peripheral neuropathy,psychosis andcoma. A 45-year-old male presented with the history of gait-ataxia.Investigations revealed the hypothyroidism. Anti-TPO antibody was positive. Thyroid replacement therapy was started and our patient improved completely within 3 weeks. The main aim of this case report is to highlight the fact that hypothyroidism has to be considered in all patients who present with acute onset of cerebellar ataxia.

Key Words: Hypothyroidism, Cerebellar ataxia, Auto-immune thyroiditis

INTRODUCTION

Hypothyroidism is a common problem in clinical practice. In almost all the cases, the neurologic manifestations occur along with the systemic features of the disease. However, symptoms of neurologic dysfunction may be the presenting feature in some patients. Most of these neurological complications improve completely after thyroid hormone replacement.

In 1960 Jellinekand Kelly published their paper on an ataxic syndrome affecting six patients who were also found to have hypothyroidism [1]. In five of them cerebellar signs improved after hormone replacement. Jellinek and Kelly also mentioned the fact that the classical signs and symptoms of hypothyroidism may not be clinically obvious in patients with ataxia. Our patient did not have other classical signs and symptoms of hypothyroidism.

Hypothyroidism should be considered in all cases of cerebellar ataxia as it is a reversible cause of ataxia. There is hardly any recent published data on hypothyroidism causing ataxia. We present this case to highlight the fact that hypothyroidism can present as acute cerebellar ataxia.

CASE REPORT

A 45-year-old male presented to our hospital with history of unsteadiness of gait since 2 weeks. It was acute in onset and progressive in nature. There was no history of weakness of limbs, headache, vomiting, convulsions or loss of consciousness. There was no history of trauma to the head, fever or drug intake. History of occasional consumption of alcohol was present.

On examination his vitals were normal. Cognitive functions were normal. Neurological examination showed gait ataxia, dysarthria and dysmetria on finger-nose and heel-to-knee tests. There was clumsiness of rapidly alternating pronation and supination in the upper limbs mainly on the left side. There was horizontal nystagmus. The gait was wide-based and there was a tendency to fall to left side. Tandem walking was impaired.

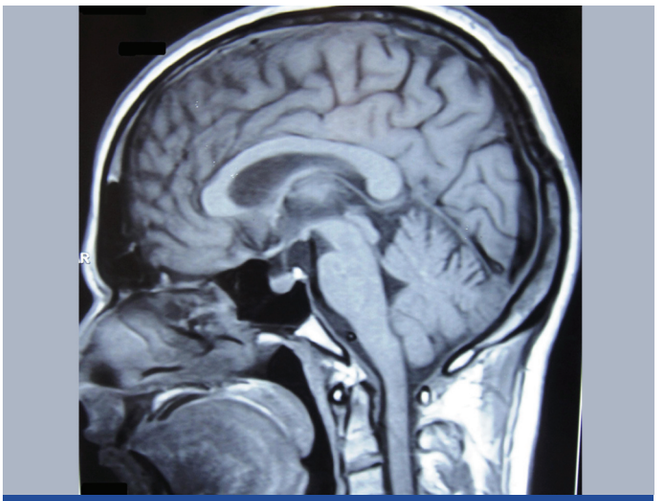
His fundus was normal. His power and reflexes were normal. Sensory system was normal. There was no evidence of autonomic dysfunction.

His investigations revealed:Hb: 13.3g% ,TLC:6,000 /mm3, DLC: N54, L36,E6,M4, ESR: 13mm Platelet count: 2, 28, 000/ mm. Serum electrolytes,blood sugar, renal and liver function tests were normal. HIV spot: negative. Serum CPK: 1315U/L. Total cholesterol: 280mg/dl, Triglycerides: 204mg/dl, HDL cholesterol: 24 mg/dl, LDL cholesterol: 206.20mg/dl, VLDL cholesterol: 40.8mg/dl.

T3:0.410ng/ml (0.6-2.02), Free T4: 0.137ng/ml(0.93-1.71), TSH: 198.7uIU/ml(0.27-5.5). The Serum anti-TPO antibody was elevated(143 IU/l; reference: <34).

His chest X-ray was normal. Ultrasonography of thyroid showed: hypoplastic thyroid with reduced echo texture. Patient refused FNAC of thyroid. MRI brain [Table/Fig 1] was normal.

His lab investigations suggested hypothyroidism (auto-immune).He



[Table/Fig-1]: Normal MRI Brain

was started on 100µg of thyroxine. He improved completely within 3 weeks. TSH after 3 months of therapy was 3.49 uIU/ml.

DISCUSSION

Hypothyroidism is one of the causes of acute onset (usually reversible) ataxia [2].Stroke,viral encephalitis and drugs can also cause acute cerebellar ataxia.Mass lesions in the posterior fossa, infections such as HIV, deficiency syndromes such B1 and B12, alcohol and para-neoplastic syndromes are causes of subacute onset cerebellar ataxia in an adult.

Hypothyroidism has been recognized as a cause of gait ataxia [3]. Restoring a euthyroid state after hormone supplementation has reversed the cerebellar symptoms in most patients [4], suggesting that symptoms were due to endocrine mediated dysfunction of the cerebellum. In some patients with hypothyroidism, the cerebellar deficits have persisted despite adequate treatment [5]. Our patient was started on 100µg of thyroxine and he improved completely within 3 weeks.

Selim and Drachman have reported six cases of cerebellar ataxia associated with auto-immune thyroiditis in whom the cerebellar symptoms progressed despite remaining euthyroid [6].They postulated that there were two separate mechanisms resulting in cerebellar nysfunction in patients with thyroid disorders. In hypothyroidism is 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. Our patient showed an excellent response to treatment. Dinkar et al have reported a case of Hashimoto's encephalopathy presenting purely with subacute cerebellar syndrome [7]. A similar case has been reported by Nakagawa et al [8].

Necropsies of patients with hypothyroidism and cerebellar ataxia have been reported.Barnard et al described degenerative changes in the cerebellum, particularly in the anterosuperior portion of the vermis, together with atrophy of ventral portion of the pons, transverse pontine fibres, and middle and superior peduncles in a patient with hypothyroidism and cerebellar ataxia. When I was a postgraduate student my neurology professor would always mention that hyponatremia and hypothyroidism should be considered in patients presenting with ataxia as they were reversible causes of ataxia. As a student I had read in Harrison(text book of medicine) that hypothyroidism can present with ataxia but I had never imagined that I would encounter a patient with ataxia as the predominant manifestation of hypothyroidism in my clinical practice.

CONCLUSION

Uncommon presentations of common conditions is sometimes seen in medicine.We recommend testing for thyroid hormones and Antithyroid antibodies(anti-TPO) in patients who present with ataxia. Classical signs of hypothyroidism may not be clinically obvious in patients presenting with ataxia. Such a case report will sensitize the clinicians about the fact that hypothyroidism can present as acute cerebellar ataxia so that they can start appropriate treatment without wasting time.

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