Case Report

Reversible cerebellar ataxia with thyrotoxicosis: An autoimmune brain disease in remission due to Graves' disease

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ABSTRACT

We hereby report a patient with seizure disorder who was on long term carbamazepine, admitted with features of thyrotoxicosis and cerebellar dysfunction. Anticonvulsant medications are cerebellar toxins; but in this case, reversal of cerebellar dysfunction was noted upon treatment of thyrotoxicosis with antithyroid drugs.

Key words: Ataxia, autoimmune, encephalopathy, thyrotoxicosis

Introduction

Autoimmune brain disease is a well known entity, but very few cases of ABD in association with thyrotoxicosis are reported. They are well described in patients with hypothyroidism. We report a patient with thyrotoxicosis and cerebellar dysfunction who was on long term carbamazepine for seizure disorder. The cerebellar dysfunction in this case due to Graves' disease and the patient showed progressive improvement with reversal of all symptoms and signs of cerebellar dysfunction upon treatment of thyrotoxicosis with antithyroid drugs.

Case Report

A 41-year-old male patient was admitted for evaluation of progressive imbalance of gait and difficulty in walking for two months. He was unable to sit in the bed without steadying himself. He has noticed a weight loss of 10 kg in the previous six months. Past medical history was significant for anti-epileptic medications for ten years for seizure disorder, which included carbamazepine 1000 mg / day in divided doses and clobazam 10 mg once daily. His seizures were under good control and the last episode was five years back. He was nonethanolic.

General physical examination showed diffuse goiter and tachycardia. His body weight was 54 kg with a BMI of 20.6 kg/m^2. His blood pressure was 150/60 mm of Hg. Central nervous system (CNS) examination showed dysarthria and there were no cranial nerve deficits. Proximal muscle weakness was noted in the lower limbs bilaterally with a power of 4/5. Examination of sensory system was unremarkable. Cerebellar functions showed bilateral nystagmus on eccentric gaze and in-coordination of both upper and lower limbs (LL > UL). Tandem walking was not possible and the gait was ataxic. Outstretched hands showed fine tremors bilaterally. Clinical impression was thyrotoxicosis with cerebellar syndrome.

On investigations, complete blood count, ESR, blood sugar, serum electrolytes, calcium, magnesium, renal and liver function tests were normal. Thyroid function tests initially on 2/10/2008 showed T3: 8 ng/ml; T4: 27.4 μg/dl; TSH: 0.1 μIU/ml suggestive of primary hyperthyroidism. Ultrasound neck showed diffuse hypoechogenicity in both the thyroid lobes with increased vascularity. Radionuclide

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thyroid scan showed diffuse increase in uptake suggestive of Graves' disease. ECG and echocardiography was normal. MRI brain was normal. Serum carbamazepine levels were also normal. Patient was started on tablet Carbimazole 10 mg thrice daily and tablet Propranalol 40 mg twice daily. On subsequent follow-up after 2 months, patient was feeling better symptomatically. After 7 months of starting antithyroid drugs, he showed remarkable improvement with no gait difficulty and in-coordination. During the subsequent follow-ups he showed sustained maintenance of near normal thyroid hormonal levels without any recurrence of initial symptoms. He was maintained on a dose of 20 mg of Carbimazole per day [Table 1].

Discussion

ABD consists of a syndrome of CNS, which is caused by antibodies or immune cells attacking the brain. Diagnosis is based upon history, clinical examination, and laboratory evaluation. The main features of cerebellar disorders include in-coordination, imbalance, and troubles with stabilizing eye movements. Cerebellar ataxia in association with autoimmune diseases is subacute, in which unsteadiness is associated with lateral veering. Imaging is done to rule out stroke, multiple sclerosis, or tumors. Shrinkage of the cerebellum is a feature of hereditary degeneration or toxin-mediated damage. Severe cerebellar symptoms with normal MRI scan usually suggest a paraneoplastic cerebellar problem; wherein the source of cerebellar injury is autoantibody, like in cases of autoimmune disorders.

Known CNS manifestations of thyrotoxicosis include seizures, pyramidal signs, chorea, depression, confusional state, dementia, and psychosis.[1] In thyrotoxic Hashimoto's encephalopathy, reduction in anti-thyroid-antibody concentration was reported with improvement in the manifestations of the disease.[2] Persistent hemichorea associated with thyrotoxicosis[3] and recurrent and reversible episodes of cerebellar ataxia with concomitant episodes of hyperthyroidism are also reported.[3] Chorea associated with thyroxine replacement therapy, which is not due to an autoimmune phenomena is also reported.[4]

Table 1: Serial thyroid function test reports and medications

DATE	Thyroid function tests	Medications
02/10/08	T3: 80 ng/dl; T4: 27.4 μg/dl; TSH: 0.1 μIU/ml	Carbimazole 10 mg thrice daily and Propranalol 40 mg twice daily.
15/12/08	T3: 33 ng/dl, T4: 9.7 μg/dl; TSH: 0.1 μIU/ml; Free T4: 1.4 ng/dl.	Carbimazole 10 mg thrice daily and Propranalol 20 mg twice daily.
06/05/09	T3: 0.7 ng/ml; T4: 7.9 μg/dl; TSH: 0.1 μIU/ml; Free T4: 1 ng/dl.	Carbimazole 10 mg twice daily
29/07/10	FT3: 4.74 pmol/L; FT4: 1.73 ng/dl.; TSH: 0.1 μIU/ml	Carbimazole 10 mg twice daily

*Normal range: FT4 = 0.93-1.71 ng/dL; FT3 = 3.1-6.8 pmol/L; TSH = 0.27-4.2 mu/L; T3 = 80-180 ng/dl; T4 = 4.6-12 ug/dl.

The patient in question had thyrotoxicosis due to Graves' disease with cerebellar syndrome. He had subacute ataxia with incoordination, a scanning speech and nystagmus. Graves' disease is associated with antibodies against TSH receptor, thyroglobulin, and thyroid peroxidase. Interestingly patient was also on carbamazepine, which is also known to cause cerebellar ataxia. Cerebellar ataxia in upper limbs triggered by addition of carbamazepine to lithium treatment is also reported.[5] The therapeutic serum concentration of this drug is 6-12 micrograms/ml and with levels of more than 9 micrograms/ml, CNS side effects such as vertigo and ataxia can develop. But the serum drug level was normal and the patient was continued with the same dose of carbamazepine as before and followed up closely for recovery. Patients with thyrotoxicosis due to Graves' disease are known to attain remission with antithyroid drugs for up to two years or more. Antithyroid drugs also have immunosuppressive action;[6] thereby reducing the serum TSH- receptor antibody levels and thus induces remission. The natural history of thyrotoxicosis due to Graves' disease is also variable with prolonged remissions or relapses and remissions. The conclusion that remission is associated with restoration of the euthyroid state, and that it is not a special drug effect, highlights the importance of making and keeping patients with Graves' disease euthyroid.[7]

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