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Case Report

HEMICHOREA: AN UNUSUAL MANIFESTATION OF THYROTOXICOSIS

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ABSTRACT

Chorea is usually associated with neurological diseases. However, it can also be seen in patients with systemic lupus erythematosus (SLE), rheumatic fever (Sydenham's chorea), polycythemia vera, and thyrotoxicosis. Here, we report an interesting case of hemichorea in a 32-year-old lady, who presented with acute onset of jerky, non-repetitive involuntary movements of the left upper and lower limbs. After detailed evaluation, she was found to have thyrotoxicosis. Thyroid Tc-scan showed diffuse uptake suggesting Grave's disease. A magnetic resonance imaging of the brain was normal. Other causes of chorea like SLE and rheumatic fever were excluded. She was started on carbimazole. On follow-up after 6 weeks of therapy, there was the complete disappearance of chorea, and her thyroid hormone levels were normal.

Keywords: Extrapyramidal syndrome, Hyperthyroidism, Corpus striatum, Dancing movements.

INTRODUCTION

Chorea is usually a feature that is closely associated with the nervous system. People affected by this condition show dancing movements, commonly of the limbs. Even though it is distressing to the patient, it is not a dangerous condition [1]. Chorea is seen in many conditions such as APLA syndrome, Huntington's disease, rheumatic fever (Sydenham's chorea), systemic lupus erythematosus [2]. Chorea due to thyrotoxicosis was the first described in 1893 by Sir William Gowers [3]. There have been a few reports published in the past where patients presented with chorea due to thyrotoxicosis. This is one such report of a patient that we encountered in our hospital.

CASE REPORT

A 32-year-old lady presented with a history of abnormal movements of left upper and lower limbs. There was no history suggestive of motor weakness or sensory involvement. Her speech was normal. There was no history of a headache, seizures, rashes, or joint pain. On examination, she was completely conscious, had a tachycardia of 120 beats/min with a blood pressure of 120/80 mm of Hg. Ophthalmic examination was normal. She had violent choreiform movements of the left upper and lower limbs. Otherwise, her neurological examination was normal. The examination of other systems was within normal limits.

Hematological investigations showed a pattern of iron deficiency anemia. Renal function tests, liver function tests, and copper studies were normal. MRI of the brain was also normal. ASO titer was within normal limits, thus excluding streptococcal origin. ANA was negative. Thyroid function tests done showed T3 of 3.68 IU/ml, T4 of 16.35 IU/ml and thyroid stimulating hormone of <0.005 mIU/ml (indicating hyperthyroidism). Anti-TPO antibody levels were high 357.6 IU/ml. Thyroid Tc-scan done showed diffuse uptake by the thyroid gland, confirming thyrotoxicosis. She was treated with carbimazole for the same. 6 weeks later, when the patient turned up for a review, she was completely asymptomatic, and her thyroid hormone levels were normal.

DISCUSSION

Chorea is associated with a variety of disease states, indicating that this extrapyramidal syndrome has several factors associated to its pathophysiology. In Huntington's chorea, the neuronal damage is found in the striatum, whereas chorea in thyrotoxicosis is thought to be more of biochemical in origin, rather than being structural [4]. In all forms of chorea, it has been observed that dopamine agonists will produce exacerbation while dopamine antagonists ameliorate the condition. The mechanism of thyrotoxicosis causing chorea is hypothesized to be due to increased sensitivity of dopaminergic receptor sites in the corpus striatum. Klawans and Shenker have also shown that turnover of dopamine is lowered in hyperthyroid patients as a reflection of hypersensitivity of dopaminergic receptors [5]. Case reports of thyrotoxic chorea have noted rapid response to beta-blockers and dopamine antagonists given along with anti-thyroid drugs [6,7]. In our case, we had excluded most of the other causes of chorea and the fact that she improved after achieving euthyroid state gave us a strong clue that the chorea was secondary to thyrotoxicosis.

CONCLUSION

To conclude, chorea or hemichorea may be one of the primary features of thyrotoxicosis although this is a rare scenario. Once a patient with chorea walks into the clinic, the clinician almost always jumps to the conclusion that the patient has a sole neurological disorder. It should be kept in mind that there are other causes for chorea, and thyrotoxicosis (a medical emergency) is one of them.

REFERENCES

1. Huntington G. Classic articles on chorea. J Neuropsychiatry Neurosci 2003;15(1):109-12.
2. Sudo K, Tashiro K. Hyperthyroidism-associated chorea. Lancet 1998;352(9123):239.
3. Javaid A, Hilton DD. Persistent chorea as a manifestation of thyrotoxicosis. Postgrad Med J 1988;64(756):789-90.
4. McMenemy W. Huntington's chorea. In: Greenfield JC, McMenemy W, Norman MG, editors. Neuropathology. London: Edward Arnold; 1958. p. 475-521.
5. Klawans HL Jr, Shenker DM. Observations on the dopaminergic nature of hyperthyroid chorea. J Neural Transm 1972;33(1):73-81.
6. Sutherland GA. Chorea and Grave's disease. Brain 1903;26:210.
7. Fidler SM, O'Rourke RA, Buchsbaum HW. Choreoathetosis as a manifestation of thyrotoxicosis. Neurology 1971;21(1):55-7.