Thyrotoxicosis Resulting in Unilateral Upper Limb Chorea and Ballismus

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A 67-year-old male, with a medical history significant for remote right frontoparietal stroke and thyroidectomy with ablation for thyroid papillary carcinoma 20 years ago with subsequent hypothyroidism, developed acute-onset, unilateral, left-arm chorea/ballism upon awakening. There were no exposures to new medications or toxins. His thyroid hormone replacement medications had not changed in dosage or formulation. He had no family history significant for movement disorders. On neurologic examination, his mental status and cranial nerves were within normal limits. He had spasticity, mildly reduced strength, and 3+ hyperreflexia involving the left hemibody. There were ongoing, non-distractible choreiform movements of the left upper extremity, with predominant distal involvement (Video Part 1). The remainder of his examination was normal.

MRI brain performed the next day showed only chronic right frontoparietal encephalomalacia with no acute infarcts (Figure 1). Paraneoplastic panel, antiphospholipid antibodies, renal and liver function, and copper studies were unremarkable. Thyroid stimulating hormone was low (0.06 mIU/L) and free triiodothyonine/free thyroxine were mildly elevated, at 16.9 pmol/L (ref 7.0–17.0) and 5.6 pmol/L (ref 3.3–6.0), respectively. His thyroglobulin was normal, at <0.2 pmol/L (ref ≤ 53.0). It was concluded that the underlying etiology to his hemiballismus was his thyrotoxicosis, which manifested as an asymmetric movement disorder due to his prior unilateral brain insult. Endocrinology was consulted, his L-thyroxine dosage was decreased, and propranolol was titrated to 20 mg twice a day. After two months, chorea/ballismus diminished considerably (Video Part 2).

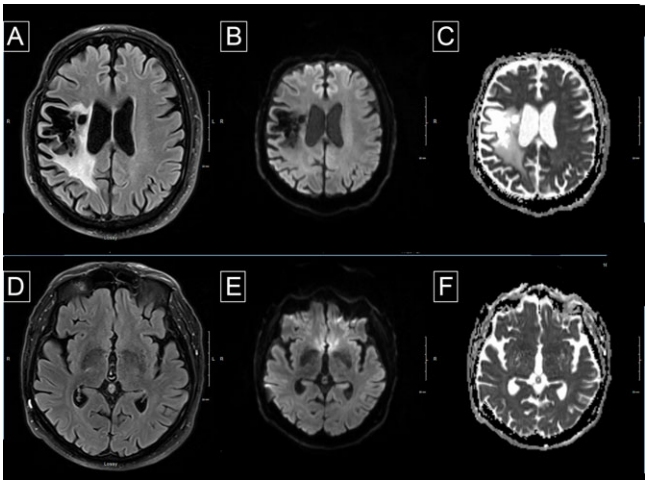

Figure 1: *TOP: MRI brain demonstrating right encephalomalacia from remote stroke. Panels (A–C) represent FLAIR, DWI, and ADC sequences, respectively. BOTTOM: MRI brain demonstrating normal basal ganglia structures without evidence of acute infarct. Panels (D–F) represent FLAIR, DWI, and ADC sequences, respectively.*

Few cases have been reported in the literature of unilateral chorea associated with thyrotoxicosis, and such distinct focality is usually suggestive of an underlying cerebral lesion.[1,2] This case illustrates that hyperthyroidism can present as a focal movement disorder in patients with prior cerebral insult. Hemibody chorea classically localizes to the contralateral subthalamic nucleus, but may be associated with lesions in the contralateral basal ganglia or corona radiata.[3] Systemic disorders such as nonketotic hyperglycemia and polycythemia vera can also present with asymmetric involvement. While Sydenham chorea may present asymmetrically, the current subject's age makes this diagnosis much less likely. The acuity of his symptoms should provoke readers to think about sporadic, acquired causes of hemichorea, including structural, metabolic, autoimmune, infectious, or drug etiologies.[3] Given the age of the current subject, the first investigation to consider would be MRI imaging to look for a new vascular lesion (i.e., stroke). In light of his otherwise negative investigations looking for alternative etiologies, thyrotoxicosis-associated hemichorea was ultimately suspected to be the most likely diagnosis.

Treatment of hyperthyroidism-associated chorea consists of correcting the underlying biochemical abnormalities, and the addition of symptomatic agents, if necessary.[4] While some patients experience relief of their movement disorder with endocrinologic correction alone, there are reported cases of persistent chorea despite euthyroidism.[5] Multiple theories exist with regard to the underlying pathophysiology of hyperthyroidism-associated chorea, including functional striatal dysfunction, hypersensitivity to dopaminergic receptors, and induction of a hyperadrenergic state due to dysregulation of the sympathetic nervous system.[4]

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The significant improvements in our subject's choreiform movements after the introduction and titration of a beta blocker seem to support the theory that hyperthyroidism-induced chorea may be at least partly associated with a hyperadrenergic state.

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Statement of Authorship

RL and MM were responsible for conception, organization, and execution of the research project. RL wrote the first draft of this article. MM, AB, and TM reviewed and edited this article for intellectual content.

Ethical Standard Statement

The authors confirm that approval of an institutional review board was not required for this work. Informed consent was obtained from the patient. We confirm that we have read the journal's position on issues involved in ethical publication and affirm that this work is consistent with those guidelines.

Supplementary Material

To view supplementary material for this article, please visit https://doi.org/10.1017/cjn.2021.136

References

1. Baba M, Terada A, Hishida R, Matsunaga M, Kawabe Y, Takebe K. Persistent hemichorea associated with thyrotoxicosis. Intern Med Tokyo Jpn. 1992;31:1144–46. doi: 10.2169/internalmedicine.31.1144.
2. Nagaoka T, Matsushita S, Nagai Y, Kobayashi K. A woman who trembled, then had chorea. Lancet. 1998;351:1326. doi: 10.1016/S0140-6736(97)11126-6.
3. Termsarasab P. Chorea. Contin Lifelong Learn Neurol. 2019;25:1001. doi: 10.1212/CON.0000000000000763.
4. Docherty MJ, Burn DJ. Chapter 20: Hyperthyroid chorea. In: Weiner WJ, Tolosa E, editors. Handbook of clinical neurology. Vol. 100. Hyperkinetic movement disorders. Elsevier; 2011, pp. 279–86. doi: 10.1016/B978-0-444-52014-2.00020-3.
5. Javaid A, Hilton DD. Persistent chorea as a manifestation of thyrotoxicosis. Postgrad Med J. 1988;64:789–90. doi: 10.1136/pgmj.64.756.789.