Hashimoto's encephalopathy as a treatable cause of corticobasal disease

Sir,

A 66-year-old retired school teacher presented with difficulty in finding words, amnesia for recent events, inattention, and difficulty in walking for the last 2 years. She had jerky movements of right upper and lower limbs for 1.5 years. Three months prior to admission she showed psychomotor withdrawal and in 1 month she became bradykinetic and mute. She appeared withdrawn and apathetic with markedly reduced word output. Her speech was effortful and agrammatical with impaired sentence comprehension and relatively preserved single word comprehension. She scored 11/30 on the mini–mental state examination (MMSE) and had frontal and parietal lobe dysfunction. She had marked rigidity of all four limbs, more on the right side. Focal myoclonic jerks was seen over the right upper lower limbs during rest, aggravated by action and postures. She had severe postural instability. She had prominent and asymmetric parkinsonian features with predominant rigidity. She satisfied criteria for probable corticobasal degeneration (consensus criteria 2013[1]). A progressive nonfluent aphasia phenotype of corticobasal degeneration was considered. However, evaluation for a rapidly progressive dementia was done. Magnetic resonance imaging (MRI) scan of brain was normal except for a small right thalamic hematoma of size 0.5 cm × 0.5 cm. Electroencephalogram (EEG), serum vasculitis profile, human immunodeficiency virus (HIV)test, vitamin B12 deficiency test, and Venereal Disease Research Laboratory (VDRL) test were negative. Cerebrospinal fluid (CSF) study showed normal protein (51 mg%) and sugar (161 mg%) levels and absent cells. CSF N-methyl-D-aspartate (NMDA) receptor antibody and paraneoplastic panel, including antineuronal antibodies (ANA-1, 2, 3), Purkinje cell cytoplasmic antibodies, anti-glial nuclear antibody (AGNA-1), amphiphysin, collapsing response mediator protein (CRMP-5), Ma, Ta, were negative. Thyroid function tests revealed normal T3 (0.98 IU), T4 (14.8 IU), and thyroid-stimulating hormone (TSH) (3.09 IU). Anti-thyroid peroxidase (TPO) antibody titers were raised to 660 units (normal value being <20 units). Antithyroglobulin antibody was negative. In presence of anti-TPO antibodies, Hashimoto's encephalopathy (HE) was diagnosed.

She received parenteral methylprednisolone (1 g/day for 5 days, through intravenous route) followed by tapering doses of oral prednisolone. Within the initial 5 days of treatment, the patient showed significant improvement. She became more cooperative for examination and was found to have ideomotor apraxia and cortical sensory loss in the right upper arm. Her speech improved dramatically and comprehension became better. She was last seen 3 months after the discharge, and her higher mental functions were almost normal and extrapyramidal signs were absent. She did not have any myoclonic jerks and had a cautious gait. Repeat anti-TPO antibody titers were reduced (198 units, normal value <20 units).

Our patient satisfied clinical research criteria for probable sporadic corticobasal degeneration.[1] The right thalamic hematoma was small and did not have any clinical correlate. Thalamic bleeds have been reported in case of HE and are thought to be the result of a vasculitic process.[2] The high antithyroid antibody titer and good response to corticosteroid treatment confirmed the diagnosis of HE. Extrapyramidal symptoms are uncommon in HE and consist mostly of hyperkinetic movement disorders such as tremor, myoclonus, and choreoathtotic movements. However, hypokinetic movement disorders, such as micrographia[3] and rigidity, are rarely described.[4] To the best of our knowledge, this is the first report on cannabidiol (CBD) like presentation of HE.

Patients with phenotype of corticobasal disease can have varying pathologies.[5] In the series of patients with rapidly progressive dementia (RPD) described by Geswind *et al*. autoimmune encephalopathy was the third commonest cause.[6] Hence, investigation for HE should be done in corticobasal ganglionic degeneration (CBGD), if progression is rapid.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

Sasi Kumar Sheetal, Robert Mathew, Byju Peethambaran

Department of Neurology, Pushpagiri Institute for Medical Sciences, Thiruvalla, Kerala, India

For correspondence:
Dr. Robert Mathew, KMLRA 136, Mosque Lane, Kumarapuram, PO - Medical College, Trivandrum - 695 011, Kerala, India.
E-mail: robert_mathew90@yahoo.co.in

References

1. Armstrong MJ, Litvan I, Lang AE, Bak TH, Bhatia KP, Borroni B, *et al*. Criteria for the diagnosis of corticobasal degeneration. Neurology 2013;80:496-503.
2. Yamaguchi Y, Wada M, Tanji H, Kurokawa K, Kawanami T, Tanji K, *et al*. Steroid-responsive Thalamic Lesions Accompanying Microbleeds in a Case of Hashimoto's Encephalopathy with Autoantibodies Against α-enolase. Intern Med 2013;52:1249-53.
3. Inoue K, Kitamura J, Yoneda M, Imamura E, Tokinobu H. Hashimoto's encephalopathy presenting with micrographia as a typical feature of parkinsonism. Neurol Sci 2012;33:395-7.
4. Rugole B. Rigidity and hyperCKemia as presenting signs

of Hashimoto's encephalopathy. Can J Neurol Sci 2013;40: 753-4.
5. Mathew R, Bak TH, Hodges JR. Diagnostic criteria for corticobasal syndrome: A comparative study. J Neurol Neurosurg Psychiatry 2012;83:405-10.
6. Geschwind MD, Shu H, Haman A, Sejvar JJ, Miller BL. Rapidly Progressive Dementia. Ann Neurol 2008;64:97-108.

Access this article online

Quick Response Code:

Website: www.annalsofian.org

DOI: 10.4103/0972-2327.176859

How to cite this article: Sheetal SK, Mathew R, Peethambaran B. Hashimoto's encephalopathy as a treatable cause of corticobasal disease. Ann Indian Acad Neurol 2016;19:285-6.
Received: 21-08-15, **Revised:** 22-09-15, **Accepted:** 15-10-15