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# A CASE OF HASHIMOTO'S ENCEPHALOPATHY IN A PATIENT WITH LITHIUM TOXICITY

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## A CASE OF HASHIMOTO'S ENCEPHALOPATHY IN A PATIENT WITH SUSPECTED LITHIUM TOXICITY

### ABSTRACT

Hashimoto's encephalopathy is a rare neurological disorder of unknown etiology. We presented, a case of middle aged male with bipolar disorder on Lithium carbonate for 30 years, admitted with altered behavior for 2 weeks and high Lithium levels. He was admitted with the suspicion of Lithium toxicity. EEG showed generalized epileptiform discharges. MRI brain revealed frontal cortical atrophy. CSF detailed report was normal. Further workup showed a high TSH level and positive anti-TPO (anti thyroid peroxidase) antibodies. Based on clinical picture, raised antibodies and no discernible cause, diagnosis of probable Hashimoto's encephalopathy was made. He received pulse of methylprednisolone for five days and his symptoms improved dramatically. Our case report highlights the importance of diagnosing a rare neurological syndrome in a complex clinical scenario.

### Key Words:

Hashimoto's encephalopathy, Lithium toxicity, Rare.

### INTRODUCTION:

Hashimoto's encephalopathy (HE) is an uncommon neurological syndrome associated with Hashimoto's thyroiditis, first reported by Brain et al in 1966.<sup>(1)</sup> The disease includes a wide spectrum of neurologic symptoms ranging from stroke like presentation to diffuse progressive pattern of slow cognitive decline with dementia, confusion and hallucination. Treatment with corticosteroids is almost always successful. Other forms of immunomodulation, such as intravenous immune-globulin and plasma exchange, may also be effective.

### CASE REPORT:

A 55 years old businessman, known case of Hypertension and Bipolar disorder for 30 years was admitted through outpatient department with irrelevant talking for 2 weeks. His family was concerned for the recent changes in the patient's behaviour as he was not recognizing, passing stool in the bedroom, extremely irritable and agitated. He also left home for 2

days and was found unresponsive outside shopping mall. During these two weeks patient has been unpredictable, shouting obscenities, responding to internal stimuli and smiling. He developed tremors in both hands 6 months back and there was a history of stimulant use in the past. He was being treated for bipolar disorder with Lithium 300mg thrice daily and Carbamazepine 200 mg twice daily for the past 30 years and was on Lisinopril 5 mg daily for hypertension. Before illness he usually have mood swings but able to perform his work well. On examination, he was a middle-aged male, lying on bed, conscious, aggressive, not following commands and with significant palilalia. Neurological examination showed grossly intact cranial nerves, increased tone in all four limbs, no focal deficit, normal reflexes and downgoing plantars. No signs of meningeal irritation, nystagmus or myoclonus. Baseline workup revealed Hemoglobin of 9.6 gm/dl (Normocytic normochromic) deranged renal parameters (urea:150 mg/dl, Creatinine:3.44 mg/dl), TSH of 6 Miu/L(0.4-4) Lithium levels of 3.9 mmol/L (0.4-2 mmol/L) His lithium was stopped and intravenous hydration was started. EEG showed generalized epileptiform

discharges. MRI brain showed frontal cortical atrophy, without any contrast enhancement. CSF detailed report was unremarkable. HIV and VDRL serologies were negative. Patient was started on Acyclovir and Sodium valproate due to the presence of epileptiform discharges. Carbamazepine was continued. He received Haloperidol and later, kept on Quetiapine (25 mg twice a day) for agitation. During the seven days of hospital stay despite of correction of metabolic parameters patient did not show any improvement. HSV PCR came out as negative. His workup (anti NMDA antibodies, anti GABA antibodies) for autoimmune encephalitis was negative. Anti TPO (anti-thyroid peroxidase) came out to be positive and patient was given pulse of methylprednisolone for 5 days. His symptoms improved dramatically and he was discharged after 7 days, on tapering doses of steroids.

## DISCUSSION:

Hashimoto's encephalopathy (HE) is an uncommon neurologic syndrome associated with Hashimoto's thyroiditis, first reported by Brain et al in 1966. He reported an individual with autoimmune thyroid disease who presented with recurrent stroke-like episodes occurring independently of the thyroid status.<sup>1,2</sup>

The etiology of HE remains unclear. The mechanism of HE does not appear to be related to the thyroid status and varies from patient to patient. In two recent reviews, 23% to 35% of patients had subclinical hypothyroidism, 17% to 20% had hypothyroidism, 7% had hyperthyroidism and 18% to 45% were euthyroid.<sup>4</sup> Our reported case was hypothyroid. The development of neurological symptoms may occur

A hospital based epidemiologic study on symptomatology consistent with HE showed prevalence to be about 2.1 per 100,000.<sup>3</sup> The disorder occurs more frequently between age 44 to 46 years, with a female-to-male ratio of four to one, consistent with our patient.

Literature reviews suggests two major patterns of presentation about 25% of patients followed a stroke-like pattern of multiple recurrent episodes of focal neurologic deficits with a variable degree of cognitive dysfunction and consciousness impairment, remaining 75% present with a diffuse progressive pattern of slow cognitive decline with dementia, confusion and hallucinations.<sup>3</sup>

Shaw et al proposed the diagnostic criteria for HE based on the presence of cognitive impairment with or

without neuropsychiatric symptoms; seizures; stroke-like events; focal neurological deficit or movement disorders; elevated antithyroid antibodies; and corticosteroid responsiveness.<sup>4</sup>

EEG abnormalities are seen in 90% to 98% of patients with HE, usually a nonspecific slow background activity. Focal spikes or sharp waves and transient epileptic activity are less common<sup>5</sup>

EEG performed on two occasions in our patient showed generalized epileptiform discharges.

In a review of 82 patients with HE, brain computed tomography or MRI showed abnormalities in 49% such as cerebral atrophy, focal cortical abnormality, diffuse subcortical abnormality and nonspecific subcortical focal white matter abnormality<sup>6</sup>. Our patient showed frontal cortical atrophy with no parenchymal abnormalities, which is either due to bipolar disorder or a part of Hashimoto's encephalopathy.

Elevated titers of antithyroid antibodies (especially TPO-Ab) are the most relevant paraclinical finding, which could be considered to be a hallmark of HE. Presented case showed, that all patients with progressive cognitive impairment or with a dementia of unclear origin should be tested for HE.

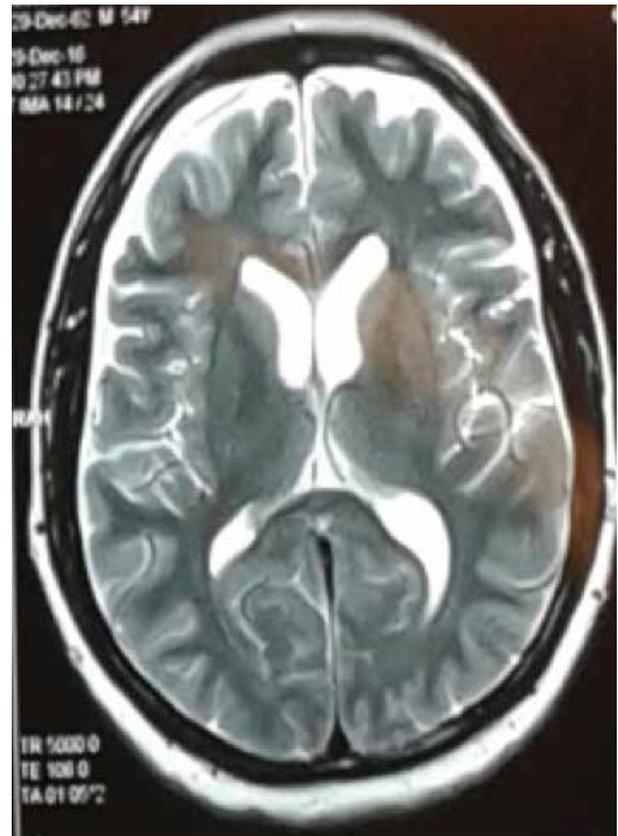
The diagnosis of HE should be considered even if Hashimoto's thyroiditis is not known in patients with euthyroid states but with high serum and/or CSF titers of antithyroid antibodies. About 50% of cases are non-responders to corticosteroid therapy, thus non-responsiveness should not be exclusion criteria for HE, other therapeutic strategies should be tried: immunosuppressive therapy (azathioprine or cyclophosphamide) and recently proposed in some cases plasmapheresis or intravenous immunoglobulins. Our patient responded well to corticosteroid pulse therapy.<sup>7,8</sup>

Our patient was admitted with the suspicion of Lithium toxicity due to high serum lithium levels. Laboratory workup revealed high urea. His mental status did not improve despite the correction of deranged metabolic parameters. We exclude all other infective causes of encephalopathy by MRI brain and CSF studies. This led us to think of atypical causes of encephalopathy like NMDA encephalitis and Hashimoto's encephalopathy. Positive anti thyroid peroxidase antibodies and dramatic response to steroids prove it to be a case of Hashimoto's encephalopathy.

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## FIGURE LEGEND:



T2 Weighted image showed significant frontal cortical atrophy .

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**Author's contribution:**

**Saba Zaidi:** Study concept and design, data collection, data analysis, manuscript writing, manuscript review

**Bashir A. Soomro:** data analysis, manuscript writing, manuscript review