



CASE REPORT

Complex partial status epilepticus as a manifestation of Hashimoto's encephalopathy

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KEYWORDS

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Complex partial seizure;
Status epilepticus;
Corticosteroid

Summary Epileptic seizures are a frequent manifestation of Hashimoto's encephalopathy. However, status epilepticus associated with Hashimoto's encephalopathy are not well characterized in medical literature. We described here a 16-year-old girl who presented with complex partial status epilepticus associated with elevated anti-thyroid antibodies. Ictal EEG showed lateralized high amplitude rhythmic delta waves over the right hemisphere and ictal single-photon emission computed tomography revealed regional hyperperfusion of the right parietal and temporal lobes. The patient was unresponsive to antiepileptic drug therapy but responded to intravenous steroid treatment. Screening of serum anti-thyroid antibodies for unexplained encephalopathy with epileptic seizures is suggested, as early recognition and prompt steroid treatment may lead to a favorable prognosis.

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Introduction

Hashimoto's encephalopathy is a rare, chronic relapsing and remitting encephalopathy associated with auto-antibodies against thyroid components.¹ Clinical presentations of Hashimoto's encephalo-

pathy are usually non-specific, and common neurologic manifestations include cognitive impairment, behavior changes, stroke-like episodes, seizures, psychosis, tremor, myoclonus, stupor, and even myelopathy.^{1,2} Epileptic seizures, both generalized and partial, are common manifestations that have been reported in 66% of patients.^{1,3} However, status epilepticus occurring with Hashimoto's encephalopathy is infrequently reported in the literature. We report here a case of Hashimoto's encephalopathy presenting as complex partial status epilepticus and review other reported cases in medical literatures.

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Case report

A 16-year-old girl was admitted to the Neurology ward due to mental disturbances and prolonged confusional states. One month before admission, she developed subacute onset of impaired attention, bizarre behavior, self-talking, insomnia, and mood disturbances. After admission, she had episodic disorientation about 7–8 times/day, with confusion and acute psychotic states. The duration of attacks was from 30 to 40 min. Meaningless vocalization, laughing, chewing, and lips smacking were also noted during the attacks. Between episodes, the deteriorated consciousness occasionally did not return to the baseline. After two generalized motor convulsions, she received treatment immediately with intravenous lorazepam and subsequent intravenous phenytoin therapy (began with a loading dose of 800 mg and maintained at 300 mg/day). However, consciousness did not improve and episodic mental changes persisted, despite serum phenytoin levels were within therapeutic range. She gradually developed intermittent, semi-rhythmic clonic movement of the left arm, accompanied by alternations of consciousness.

The patient's gestational and neonatal history was unremarkable, with normal developmental milestones. There was no history of major systemic or infectious diseases. Family history of epilepsy or febrile convulsion was also negative. Physical examination was unremarkable. Laboratory studies, including complete blood count, alanine transaminase/aspartate transaminase, blood urea nitrogen/creatinine, and serum electrolytes were all within normal limits. The cerebrospinal fluid was likewise unremarkable, as well as tests for drug screens, blood

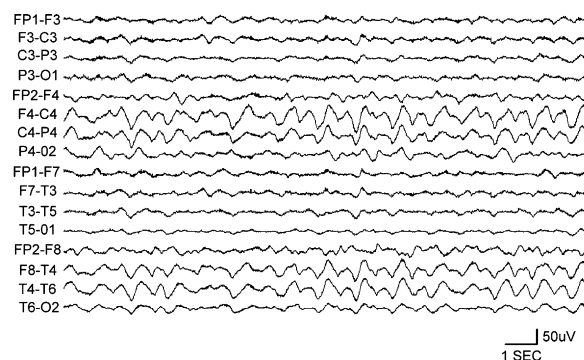


Figure 1 Ictal EEG showed continuous, rhythmic, lateralized delta waves over the right hemisphere.

lead and copper level, porphyria screens, syphilis, anti-nuclear antibody, viral infections (herpes simplex virus, human immunodeficiency virus, hepatitis B, hepatitis C, and cytomegalovirus and Epstein-Barr virus), tumor makers, and serum electrophoresis.

Ictal electroencephalogram (EEG) revealed continuous high amplitude rhythmic lateralized delta waves over the right hemisphere (Fig. 1). Intermittent photic stimulation during EEG studies did not evoke abnormal photoparoxysmal response. Ictal brain ^{99m}Tc single-photon emission computed tomography (SPECT) revealed a hyperperfusion state over the right parietal and temporal lobes (Fig. 2A). Brain magnetic resonance imaging showed hyper-intense lesions on fluid-attenuated inversion recovery-sequences (Fig. 2B) and T2-weighted images over the right medial temporal area.

Thyroid function tests showed that serum free T4 was 2.05 ng/dL (normal, 0.79–2.01 ng/dL), T3 was 97.6 ng/dL (normal, 52–175 ng/dL), and thyroid-stimulating hormone was below 0.2 $\mu\text{IU/mL}$

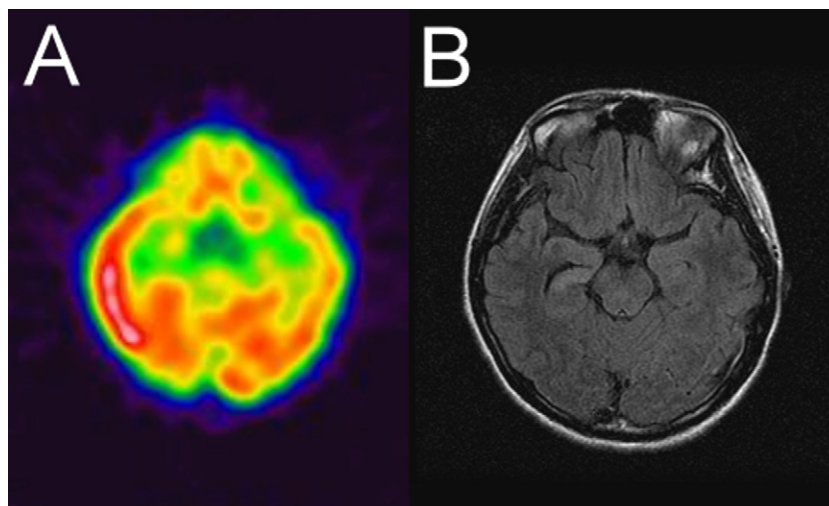


Figure 2 (A) Ictal SPECT revealed regional hyper-perfusion over the right parietal and temporal lobes. (B) Interictal MRI showed hyperintensity on fluid-attenuated inversion recovery-sequences image over the right medial temporal area.

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