Hashimoto’s Encephalopathy Presenting as Acute Psychosis

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Abstract

Background: Hashimoto’s encephalopathy is a relapsing encephalopathy occurring in association with Hashimoto’s thyroiditis, with high titers of anti-thyroid antibodies. Clinically the patients may present with acute or subacute encephalopathy, seizure, myoclonus, and tremulousness, stroke like episode, amnesia or dementia. Here we are reporting a case of hashimoto’s encephalopathy who presented with features of acute psychosis.

Case Report: A 56 years old Asian female presented with acute onset of altered behavioural abnormality and declining brain function. Serology revealed high anti thyroperoxidase antibodies. EEG and MRI were consistent with hashimoto’s encephalopathy.

Conclusion: Hashimoto’s encephalopathy is a rare complication of hashimoto’s thyroiditis and is a diagnosis of exclusion and should be suspected in a case of encephalopathy, high anti thyroid antibodies and response to glucocorticoid.

Keywords: Hashimoto’s encephalopathy; Acute psychosis; Magnetic resonance imaging

Introduction

Hashimoto’s encephalopathy has been described as an encephalopathy, with acute or subacute onset, accompanied by seizures, tremor, myoclonus, ataxia, psychosis, and stroke like episode with relapsing/remitting or progressive course. Hashimoto’s encephalopathy is supposed to be of autoimmune origin as supported by its association with other autoimmune diseases. Hashimoto’s encephalopathy is more common in women than in men. It has been reported in paediatric, adult and elderly populations throughout the world. Hashimoto’s encephalopathy is associated with CSF changes, EEG and radiological abnormalities. Hashimoto’s encephalopathy appears to be a rare disorder, but, as it is responsive to treatment with corticosteroids, it must be considered in cases of investigation negative encephalopathy. Here we report an interesting case of Hashimoto’s encephalopathy who presented with psychiatric manifestations.

Case Report

A 56 years old female diagnosed previously as hypertensive and hypothyroid on 25microgram of levothyroxine daily presented with features of acute psychosis in the form of speech incomprehensible sound. Over a period of three to four days she became irritable, irrelevant talking, aggressive behaviour and urinary incontinence, altered sleep cycle, and later only stuttering speech.

Although she did not have fever, headache, vomiting, sensory or motor abnormality or history of previous similar complains or any other psychiatric complains. On examination she was restless, irritable, apraxic, disoriented and not following commands with incomprehensible sound.

Her blood pressure was 160/90 in her right arm supine position, her oral temperature was 38.6 c, and respiratory rate was 18/minute. Pupils were bilateral small and reacting to light and consensual reflex was present. She had no sensory or motor deficit. There was no hepatosplenomegaly and examination of chest and heart were normal.

Routine lab examinations including complete blood count, liver function test and renal function test were within normal parameters. CSF examination showed no cells, sugar of 77 mg/dl and protein of 40 mg/dl. Thyroid function test revealed T3, T4 and TSH of 0.77, 4.77 and 10.87 respectively and anti thyroperoxidase antibody value of 2147.50 units. Chest x ray was within normal limits and two dimensional colour Doppler of heart showed normal heart functions with mild left ventricular hypertrophy. Additionally, antinuclear antibody titre, anti-double-stranded DNA, anti-hepatitis B core antigen, hepatitis B surface antigen, anti-hepatitis C virus, lupus anti-coagulant and Veneral Disease Research Laboratory test results were negative. The electroencephalogram (EEG) showed a slow background activity (Figure1).

T2 weighted MRI brain showed chronic lacunar infarct in bilateral periventricular region (Figure 2). During course of the management of the patient, Injection Methyl prednisolone was given intravenously.

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autoantibody level was, on day 13th of admission which was declined to
she started communication through spoken words. The repeat serum
by end of 2nd week. , but her symptoms relapsed as we started tapering
1225 units. Psychosis, which was the major presentation, disappeared
While rapid improvement can be observed within 1 to 3 days, as in
findings suggest acute disseminated encephalomyelitis as potential
models [6], while others favour cerebral angiitis as a paradigm [5]. This
manifesting with psychiatric symptoms and dementia [11]. Our patient
encephalopathy as it is a treatable condition and would avoid
mismanagement and unnecessary medications and is very rewarding.

To sum up keep high index of suspicion in any patient with
thyroid disorder with atypical presentation of acute psychosis and
encephalopathy [15] should always be suspected for hashimoto’s

Discussion

Hashimoto’s encephalopathy in patient with hypothyroidism, who
is on replacement therapy, may be precipitated by infection, trauma,
burn, or other metabolic stressors. More than a 30 case reports have
been published since Professor Brain described it first in 1966 [1].
Clinical manifestations can vary from cognitive impairment and
seizure to ataxia and movement disorder. The average age of onset in
reported cases [2-10] is 47 years (range: 14 to 78 years). Approximately
85% of the patients are women. HE can manifest in two major ways:
1) relapsing/ remitting, also referred as vasculitic type, manifesting as
encephalopathy and stroke-like episodes. 2) Diffuse progressive type
insidious onset and progressive course with fluctuations and
manifesting with psychiatric symptoms and dementia [11]. Our patient
presented with acute psychosis in the form of hallucinations followed
by subacute encephalitis. Differential diagnoses includes conditions
are Creutzfeldt-Jakob disease, rapidly progressive dementias, and
paraneoplastic and non-paraneoplastic limbic encephalitis. With the
background history of hypothyroidism and atypical presentation as
acute psychosis associated with high titres of antithyroid antibodies,
in particular antithyroid peroxidase antibodies followed by subacute
encephalitis, we suspected hashimoto’s encephalopathy. Thyroid auto
antibodies were positive and EEG abnormality was consistent with the
diagnosis of hashimoto’s encephalopathy which showed persistent fall
in levels with clinical improvement.

Pathogenesis of the disease is still not completely defined although
autoimmunity is supposed to be the triggering factor [12]. Some
findings suggest acute disseminated encephalomyelitis as potential
models [6], while others favour cerebral angiitis as a paradigm [5]. This
is further supported by the response to immunosuppressive therapy
[13] and presence of several neuronal and thyroid auto antibodies [14].
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