

Hashimoto's encephalopathy presented with mutism: a case report

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INTRODUCTION

Hashimoto's encephalopathy presents with high serum antithyroid antibody concentrations in association with an excellent response to immunosuppressant therapies such as corticosteroids.¹ Females were four to five times more likely to be affected by this condition, with a mean age of onset between 41 and 48 years², though rare occasional paediatric presentations were also reported.^{3,4} Encephalopathy, episodes of seizures, stroke-like focal neurological deficits, myoclonus and cognitive impairment are the common manifestations of this condition. Neuropsychiatric manifestations such as hallucinations and non-specific behavioural disturbances were reported in approximately 30% of patients as per the literature.^{2,5} Because of this, it often gets misdiagnosed as a psychiatric condition.

Here, we report on a child who presented with mutism and gait difficulties, and upon evaluation was diagnosed as having Hashimoto's encephalopathy.

CASE HISTORY

A 10-year-old boy presented to us with a 2-month history of fatigue and progressively reduced speech output at school. A preceding history of fever and photosensitivity (erythema over sun-exposed parts) for 3 days was reported. At home, he would often become mute, more so in front of any stranger. This was corroborated by video clips provided by the family members. In the previous 2 weeks, he also had developed gait difficulties, that is, motor weakness of the bilateral lower limbs. Therefore, he would sit in place and watch television for a whole day without attending school. His food intake also came down; mainly he took fluids/liquid food for the past 1.5 months but denied any difficulties while swallowing.

During hospital admission for 1 month, he continued to show mutism; that is, in front of hospital staff, he would not speak, not even look straight, but he would talk

with his parents using very minimal words. The liaison psychiatry team made a provisional diagnosis of selective mutism based on Diagnostic and Statistical Manual of Mental Disorders (DSM-5) criteria, although this was an atypical age of presentation. Even though there was a significant reduction in his overall speech output, there was a level of selectivity in that. Neurological examination revealed exaggerated deep tendon jerks. Cognitive function examination was not possible to do as he was not speaking with the resident, but according to his parents, he was not showing any confused or disoriented behaviour. The retrospective assessment of neurodevelopmental and scholastic performance prior to the illness onset was within normal limits. Blood investigations including blood sugar, liver function test, renal function test and serum electrolytes were within normal limits. Serum tri-iodothyronine (T3) was low (12.2 ng/dL, ref value 100–200 ng/dL), but thyroxine and thyroid-stimulating hormone were normal. Parathyroid hormone was low (4.5 pg/mL, ref value 10–55 pg/mL), but serum calcium was normal. MRI brain with a screening of spine was normal. Electroencephalography (EEG) and ECG were within normal limits. HIV, venereal disease research laboratory test, cerebrospinal fluid analyses, gram stain and staining for acid-fast bacilli, peripheral smear and antinuclear antibody profile were within normal limits. The only positive test was antithyroid peroxidase (anti-TPO) with high titre (113.5 IU/mL). Suspecting autoimmune/Hashimoto's encephalitis, he was started on empirical therapy of 12 g immunoglobulin intravenous (IVIg) infusion by a neurologist every day for 5 days. After 1 week, the patient was more talkative, responded to the comment, started walking slowly, attended self-need and had good eye contact with the ward staff. He was referred to physical medicine and rehabilitation for physiotherapy. The patient resumed school after 6 months. He was followed up



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for the subsequent 2 years. He was capable of coming to consultation alone and was doing well with the help of physical and psychiatric home-based rehabilitation that included motivational exercise and positive self-talk, speech training, social skills training, anxiety management, and coping skills training, graded exposure, and relaxation techniques.

DISCUSSION

This is a unique case of Hashimoto's encephalitis presenting as selective mutism. Generally, Hashimoto's encephalopathy presents with psychosis, mood fluctuations, insomnia, irritability and cognitive dysfunction.^{4,6} Vasculitis or disseminated encephalomyelitis secondary to impairment in cerebral perfusion is the main cause of encephalopathy.⁷ Hashimoto's encephalopathy can also present with reversible dementia with differential diagnosis of Creutzfeldt-Jakob's disease or viral encephalitis.⁴ Seizures, confusion, headache, ataxia and hallucinations are common paediatric presentations.³ EEG often shows epileptiform waves, and MRI findings are generally nonspecific. Thyroid function may be normal; however, thyroid abnormalities do not correlate with encephalopathy. Even anti-TPO antibody titre does not correlate with the symptomatic presentation.

Selective mutism is a child psychiatric condition, which usually presents around the age of 5 years, characterised by a refusal to speak in particular settings. In this condition, children may speak at home but not in other situations or front of strangers. However, often they note by nodding or by writing if encouraged. This may lead to impaired scholastic performance and isolation from peers. This condition frequently associates with an anxiety disorder as a comorbid diagnosis. As per DSM-5 criteria, minimum period of 1 month is sufficient to make a diagnosis.

Mutism can present in functional psychiatric disorder; however, it is extremely important to perform a thorough physical and systemic examination to rule out organic causes for mutism. In the case of psychiatric disorder, mutism is often a gradual onset during preschool age. It is commonly linked with social phobia, autism or other neurodevelopmental disorders. In selective mutism, children often face the dilemma of whether they should speak or not; eventually, they get adapted to certain kinds of stimulus.^{8,9} Any form of encephalitis, either chronic or acute, can present with a complex neurobehavioural problems that have been previously reported in the literature. It is only possible to rule with sensible clinical examination and laboratory investigations.¹⁰⁻¹²

In our case, the only positive findings are positivity of anti-TPO and low T3, which can be non-specific as these can also be detectable in a healthy population. However, clinical correlation and improvement with IVIG are the pointers towards the neuropsychiatric presentation of an organic condition. The parathyroid level might be

increased for a transient period as in our case, which can be explained by an inflammatory process involving both the glands.¹³ This condition is generally steroid responsive, but in children, only 55% of patients were found to be responsive, which was also very slow. Steroid-resistant cases are treated with immunoglobulin or plasmapheresis.³ In our case, the patient was treated with IVIG as the neurologist wanted prompt improvement in the children.

We are presenting this case as this is the first-ever reported case of selective mutism in the background of autoimmune or Hashimoto's encephalitis; hence, this may be of great value in the field of neuropsychiatry.

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