

Catatonia Secondary to Hashimoto's Encephalitis: A Case Report

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Abstract

Introduction: Catatonia is a neuropsychiatric syndrome with motor and behavioral symptoms. In addition to psychiatric illnesses, this syndrome can also be associated with general medical conditions. Few cases of catatonia associated with autoimmune disorders have been reported.

Case report: We report the case of a 56-year-old female patient diagnosed with Hashimoto's encephalitis, who presented firstly with mystical delusions and tachypsychia. Despite the proper treatment for psychosis, the symptoms worsened and she developed catatonia. Complementary investigations showed high titers of anti-thyroperoxidase antibodies. Hashimoto encephalitis was suspected and intravenous prednisolone was started. As there was an only partial improvement in psychiatric symptoms, plasmapheresis was chosen. The patient evolved with a drastic improvement in symptoms after the third session of plasmapheresis.

Conclusion: An organic etiology should always be considered in cases of catatonia, especially in those with no previous psychiatric history. Doctors should always think about measuring thyroid function when faced with a case of catatonia.

Keywords: Hashimoto's encephalopathy • Psychosis • Catatonia

Introduction

Catatonia is a neuropsychiatric syndrome characterized by behavioral changes and motor disorders, which occurs in approximately 8% of patients hospitalized for psychiatric disorders. About 20% of catatonias can be attributed to a medical condition other than purely psychiatric [1-4]. Some cases of catatonia associated with autoimmune diseases and paraneoplastic syndromes have been described [5-8]. The recommended first-line treatment is benzodiazepines and electroconvulsive therapy [9]. However, in the case of association with autoimmune diseases, plasmapheresis and the use of corticosteroids are effective treatment options [10,11]. Here we present a case highlighting that the presence of catatonia associated with psychosis can be a misleading presentation for an underlying secondary etiology.

Case Report

A 56-year-old female patient admitted to our early intervention Psychiatric Service, after a brief hospitalization in a psychiatric ward, presenting soliloquies, grandeur and mystics' delusions, euphoria, tachypsychism, hyporexia, decreased need for sleep and psychomotor agitation that started one month before admission. She denied previous psychotic symptoms and use of psychoactive substances. As comorbidities she referred hyperthyroidism, which had been diagnosed five months ago, and using tapazole 20 mg daily. In a complementary laboratory investigation for differential diagnosis of the first psychotic episode, according to the service protocol, it was found

Thyroid Stimulating Hormone (TSH) of 0.004 mU/L, tetraiodothyronine (free T4) of 1.1 ng/dl and anti-thyroperoxidase (anti-TPO) of 1322 U/ml, without other significant changes. An antipsychotic was started in an adequate dose and an outpatient referral was made to an endocrinologist. After two weeks, the patient returned with stupor, mutism, staring gaze, catalepsy, automatic obedience and minimal oral intake. She had no parkinsonian signs. She had been consulted by a general practitioner who increased tapazole to 30 mg daily. On physical examination, she had a thyroid enlarged diffusely about three times, mainly at the expense of the right lobe, without palpable nodules or cervical lymph nodes. The ultrasound showed a thyroid with irregular contours and a diffuse heterogeneous echotexture, without nodules. Bush-Francis scale [12] applied with a total score of 16 points. She was given 7.5 mg of intramuscular midazolam, single dose, with a positive benzodiazepine test. Given this, the antipsychotic was discontinued and lorazepam 6 mg was started, with a progressive increase up to the dose of 9 mg daily. However, the patient maintained fluctuations in psychotic and catatonic symptoms, and the hypothesis of Hashimoto's encephalitis was raised due to the high titers of anti-TPO. Pulse therapy with methylprednisolone 1000 mg daily intravenously, has been prescribed during five days. After seven days of finishing pulse therapy, the patient had partial improvement of the condition, but as there were residual symptoms, it was decided to perform five sessions of plasmapheresis, with an interval of one week between sessions. After the third session of plasmapheresis, patient presented a significant improvement in her condition and was discharged asymptomatic, with an outpatient return scheduled with an endocrinologist. Six months after hospital discharge, patient remained without psychiatric symptoms.

Discussion

Hashimoto's thyroiditis is the most common form of thyroiditis in childhood and adulthood [13], with a prevalence of 1.2% [14] and 5% [15,16], respectively. Neurological and psychiatric complications of thyroiditis include neuropathy, cerebellar dysfunction, encephalopathy, myxedema, coma, dementia, depression and psychosis [17]. Hashimoto's encephalitis is a rare syndrome, first described by Brain et al. in 1966 [18]. It should be noted that for the diagnosis of Hashimoto's encephalitis, thyroid function can vary from normal to pathological among patients. Clinical manifestations include

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confusion, coma, seizures, psychosis, dementia, catatonia, myoclonus and myelopathy. Its diagnosis is suspected whenever symptoms of acute or subacute encephalopathy are associated with high serum levels of anti-TPO antibodies in the serum and sometimes in the Cerebrospinal Fluid (CSF). Also, an increase in the concentration of proteins in the CSF can be seen. Nonspecific electroencephalogram and magnetic resonance findings are frequent. Finally, there may be responsiveness to corticosteroids and plasmapheresis. Course tends to be progressive or recurrent [17,19-21]. Pathophysiology of Hashimoto's encephalitis remains poorly understood, but among the proposed mechanisms are cerebral vasculitis and the neuronal reaction mediated by antibodies [22]. Recent publications suggest that insufficient blood flow in the left prefrontal cortex and anterior cingulate areas cause neuropsychiatric symptoms, such as psychosis, disturbance of consciousness and mood disorders [23,24]. Hashimoto's encephalitis is a very likely diagnosis in our patient due to the high titers of anti-TPO antibodies and the improvement of her clinical symptoms once treatment with prednisolone and plasmapheresis has been started.

Conclusion

Hashimoto's encephalitis is a complex diagnosis. An organic etiology should always be considered in cases of catatonia and first episode psychosis, especially in those with no previous psychiatric history or presenting atypical symptoms. Doctors should always think about measuring thyroid function when faced with a case of catatonia or first-episode psychosis.

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