Case Report

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Case Report - IgLoN5 Antibody Encephalitis: Uncovering a Rare Cause of Neurological Decline in an Elderly Patient

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ABSTRACT

Background: Iglon5 disease is a rare neurodegenerative and autoimmune condition characterized by antibodies against the neuronal cell adhesion protein Iglon5. The disease presents with a complex combination of neurological symptoms, including parasomnia, gait disturbances, and myoclonus. First identified in 2014, IgLON5 antibody disease has been associated with significant morbidity and mortality. As this condition is relatively underreported, it poses a diagnostic challenge for clinicians, especially with its prevalence in the Middle East not studied. This case report discusses an 80-year-old female patient with a progressive neurological decline, later diagnosed with Iglon5 antibody encephalitis.

Case Report Summary: An 80-year-old woman presented with dizziness and unsteadiness, which progressively worsened over several months. As her condition deteriorated, she developed abdominal pain, muscle cramps, rapid jerking movements in her limbs, and motor impairment to the point where she became unable to walk or hold objects. The patient did not present with sleep disorders such as parasomnia, excessive sleepiness or insomnia, which is usually a characteristic sign of anti-Iglon5 disease. An extensive diagnostic workup was performed, including MRI, EEG, nerve conduction studies, and blood tests. The patient tested positive for serum Iglon5 antibodies, confirming the diagnosis. She was treated with clonazepam for myoclonus, gabapentin for pain, and received intravenous immunoglobulin (IVIG) therapy. She was subsequently transferred to a rehabilitation facility for further care.

Conclusion: Iglon5 disease is a rare but severe neurodegenerative condition that requires a high degree of clinical suspicion for diagnosis. As the field of autoimmune focused medicine advances and new research emerges, there are new antibodies found. It can become difficult for physicians to stay informed of all the autoimmune panels they must order to diagnose a patient correctly. Hence it is important to create resources to easily track the potential autoimmune panels that a patient with similar symptoms can present with. Early identification and intervention, including the use of immunotherapies like IVIG, are essential in managing the disease and improving patient outcomes.

Keywords: IgLoN5 Antibody; Autoimmune Encephalitis; Neurodegeneration; Myoclonus; Intravenous Immunoglobulin Therapy; IVIG

Introduction

Neurodegenerative disorders associated with autoantibodies, such as Iglon5 antibody disease, have become increasingly recognized in clinical practice. First described in 2014, Iglon5 disease is a rare autoimmune disorder that combines features of neurodegeneration and encephalitis. It is characterized by the presence of antibodies against Iglon5, a neuronal cell adhesion protein involved in synaptic function. This disease presents with a wide array of neurological symptoms, including abnormal sleep behaviors, bulbar dysfunction, cognitive impairment, gait disturbances, and movement disorders such as myoclonus. The disease mechanism is still not entirely understood but

is thought to involve both autoimmune and neurodegenerative pathways. Given its rarity and complex presentation, the condition is often misdiagnosed, leading to delays in appropriate treatment. Early recognition and intervention are crucial for improving patient outcomes, as untreated Iglon5 disease can lead to significant functional decline and, in many cases, death. This report presents a case of an 80-year-old woman with Iglon5 antibody disease, emphasizing the importance of clinical suspicion and the role of a multidisciplinary approach in managing this complex neurodegenerative disorder [1-3].

Case Report

Patient Presentation and History

An 80-year-old female presented to the clinic in April with dizziness and episodes of unsteadiness that progressively worsened over a period of four months. Initially, these symptoms were mild, and conservative management was recommended. However, as time passed, the patient developed additional neurological complaints, including abdominal pain, muscle cramps, and involuntary jerking movements in her limbs and body. Over the next few weeks, her condition further deteriorated, and she became unable to walk or hold objects due to significant motor impairment. Her medical history was largely unremarkable, with no prior neurological or systemic illnesses. She was a nonsmoker and had no history of alcohol use. There was no family history of neurodegenerative diseases or autoimmune disorders. Given the progressive and multifocal nature of her symptoms, a comprehensive neurological evaluation was initiated.

Diagnostic Workup

The initial clinical suspicion was for encephalitis or a neurodegenerative disorder. A battery of tests was performed to investigate the underlying cause of her symptoms:

MRI (without Contrast): The MRI of the brain showed no evidence of structural abnormalities, inflammation, or lesions suggestive of encephalitis or stroke. A spine MRI revealed degenerative disc disease but no acute abnormalities that could explain the patient's symptoms.

EEG: The electroencephalogram (EEG) revealed mild, nonspecific slowing, which was not diagnostic but consistent with an underlying encephalopathic process.

Ultrasound (Hepatobiliary): An ultrasound of the liver and biliary system was normal, ruling out metabolic causes for her symptoms.

CT Head: A CT scan of the head showed no evidence of intracranial hemorrhage, mass effect, or ischemic stroke.

Nerve Conduction Study: Peripheral nerve function was found to be normal, excluding peripheral neuropathy as a cause of her symptoms.

Serum Encephalitis Antibodies: The patient tested positive for Iglon5 antibodies, confirming the diagnosis of Iglon5 antibody encephalitis.

Paraneoplastic Panel Serum: The panel was positive for Iglon5 antibodies, and malignancy was ruled out.

These findings established the autoimmune etiology of her condition, with Iglon5 antibodies being the primary factor driving her neurological decline.

Treatment and Management

Following the diagnosis of Iglon5 antibody disease, a multidisciplinary approach was employed. Given the patient's significant motor impairment and involuntary movements, symptomatic management was initiated to improve her quality of life:

- Clonazepam was prescribed to control the myoclonic jerks, which significantly reduced the severity of her symptoms. Clonazepam, a benzodiazepine, has been commonly used in such cases to manage movement disorders and has shown efficacy in controlling myoclonus.
- Gabapentin was introduced for pain management, particularly for the patient's muscle cramps and back pain, both of which were limiting her mobility and causing significant discomfort.
- Lidocaine patch was applied for localized knee pain, which had worsened as a result of her inability to walk.

In addition to these symptomatic treatments, the patient was also started on intravenous immunoglobulin (IVIG) therapy. IVIG has been employed in the treatment of autoimmune encephalitis to reduce antibody-mediated neuroinflammation. A treatment regimen was planned, with the patient receiving her first dose in the hospital and additional doses scheduled post-discharge.

Rehabilitation

Given the severity of the patient's functional decline, she was transferred to a rehabilitation facility for further management. Intensive physiotherapy was initiated to improve her mobility and strength. Occupational therapy focused on helping her regain independence in daily activities, while speech therapy was introduced to address mild bulbar symptoms that had developed later in her course of the disease.

Discussion

Iglon5 antibody disease is a rare and recently discovered disorder that combines features of neurodegeneration and autoimmunity. The clinical spectrum of Iglon5 disease is broad, with many patients presenting with nonspecific symptoms such as dizziness, gait disturbances, and myoclonus. However, sleep disturbances, such as parasomnia and obstructive sleep apnea, are often hallmark features.

In our patient, the absence of these sleep-related symptoms and the initial mild presentation led to a delayed diagnosis. Current understanding of Iglon5 disease suggests that it may lie at the intersection of autoimmune encephalitis and a tauopathy-related neurodegenerative disorder. The presence of anti-Iglon5 antibodies is considered diagnostic, though some patients with the antibody may not display clinical symptoms for years. Management of the disease focuses on immunotherapy, including IVIG and corticosteroids, to reduce neuroinflammation. Clonazepam and gabapentin are often used to control the movement and pain symptoms associated with the disorder. The rarity of Iglon5 antibody disease means that there are no standardized treatment protocols, and management often relies on case reports and small case series. Our case highlights the importance of early recognition and the need for a comprehensive, multidisciplinary approach to care. Although immunotherapy may slow disease progression, the prognosis remains poor for many patients due to the progressive neurodegenerative nature of the disease.

Conclusion

This case report highlights the complexity and challenges of diagnosing and managing Iglon5 antibody disease. Early diagnosis and intervention with therapies such as IVIG, along with symptomatic treatment, are essential for improving patient outcomes. A multidisciplinary approach that includes neurological, physical, and occupational therapies can help manage the disease's wide array of symptoms and improve the patient's quality of life. As understanding of this rare disease expands, so too will the opportunities for better therapeutic options and improved prognoses for affected individuals.

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