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CASE REPORT

Morvan Syndrome Masquerading as Anxiety Disorder: A Case Report Highlighting the Importance of Recognizing Organic Signs in Psychiatric Settings

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Abstract: Morvan syndrome is a rare subtype of autoimmune encephalitis, primarily characterized by increased peripheral nerve excitability, autonomic dysfunction, and severe insomnia. This report presents a 28-year-old female patient who sought medical attention due to widespread pain, refractory insomnia, limb sensory abnormalities, and low mood, initially diagnosed as "anxiety disorder". Neurological examination revealed muscle twitching in the limbs. Electromyography indicated increased peripheral nerve excitability, with serum testing showing weak positivity for anti-Leucine-rich Glioma Inactivated 1 (LGI-1) IgG antibodies and strong positivity for anti-Contactin-associated Protein-like 2 (CASPR2) IgG antibodies. Cerebrospinal fluid Pandy's test was weakly positive. The final diagnosis was Morvan syndrome complicated by anxiety disorder. Following treatment, the patient's symptoms significantly improved. This case highlights the diagnostic challenges of Morvan syndrome, particularly when patients present with prominent psychiatric symptoms that mimic functional disorders. It underscores the critical importance of screening for subtle organic signs—specifically fasciculations and widespread pain—in patients with refractory anxiety to prevent misdiagnosis and facilitate timely immunotherapy.

Keywords: morvan syndrome, autoimmune encephalitis, pain, anxiety disorder, voltage-gated potassium channels

Introduction

Morvan syndrome is an extremely rare subtype of autoimmune encephalitis, characterized by increased peripheral nerve excitability, autonomic dysfunction, severe insomnia, and central nervous system symptom.[1] Reports of this disease are still relatively scarce both domestically and internationally.[2] Previous studies have reported cases of Morvan syndrome combined with physical diseases such as lung cancer,[3] but cases associated with mental disorders, such as anxiety disorder, remain rare. However, the clinical presentation of Morvan syndrome can be deceptive. Patients frequently present with dominant psychiatric symptoms—such as severe anxiety, insomnia, and agitation—that co-occur with widespread neuropathic pain. This combination often mimics functional somatic disorders, leading clinicians to interpret the pain as a psychosomatic manifestation rather than an organic sign. Consequently, subtle neurological features like fasciculations are easily overlooked, resulting in misdiagnosis of psychiatric conditions and significant delays in appropriate immunotherapy. This disease is often associated with voltage-gated potassium channel (VGKC) antibodies, particularly anti-CASPR2 and anti-LGI-1 antibodies, but some atypical cases may show negative results for these antibodies.[4] Morvan syndrome is also frequently associated with neoplastic diseases, particularly thymomas, with approximately 38–56% of patients with Morvan syndrome developing thymoma.[5] Therefore, dynamic follow-up monitoring of tumor markers is crucial for patients with Morvan syndrome. Another disease, Isaacs' syndrome, shares some clinical symptoms with Morvan syndrome, such as increased peripheral nerve excitability. Both conditions can present with positive anti-LGI-1 or CASPR2 antibodies, but Isaacs' syndrome does not feature insomnia or other central nervous system symptoms. It is worth noting that a recent report described a case in which

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Isaacs' syndrome worsened into Morvan syndrome after thymectomy.[6] This case report describes a patient with Morvan syndrome who presented with prominent anxiety and widespread pain, resulting in an initial misdiagnosis of a psychiatric disorder. It is clinically noteworthy as it illustrates the diagnostic pitfall of mistaking organic neuropathic symptoms for functional anxiety, highlighting the critical need to recognize fasciculations and dual antibody positivity in psychiatric settings to ensure timely management.

Case Report

The patient is a 28-year-old female who was admitted to the Psychiatry Department of a hospital on October 11, 2023, due to "widespread pain for 50 days, worsening in the past month". Fifty days prior to admission, without any apparent trigger, the patient developed persistent, squeezing-type pain throughout her body, affecting both the bones and muscles. The pain intensified with movement, and while walking, she experienced severe tearing-like pain in the anal and perineal areas. This was accompanied by intermittent hand tremors, feelings of restlessness, palpitations, chest tightness, shortness of breath, abdominal bloating, constipation, dizziness, and headaches. She also reported abnormal temperature sensations in the extremities, with the left lower limb feeling cold and the skin icy, while the right lower limb often felt a burning sensation. The patient experienced soreness in both upper limbs and numbness in both lower limbs. Sleep was poor, with almost no sleep on most nights. There was no fever or vomiting. Due to these physical symptoms, the patient gradually began to experience frequent feelings of worry, nervousness, low mood, reluctance to communicate, frequent crying, a disinterest in activities, and spent most of the day in bed. She intermittently had negative thoughts, feeling that life was meaningless. On September 15, 2023, she visited an outpatient clinic at another hospital, where a brain CT scan, chest X-ray, routine blood tests, and biochemical examinations showed no significant abnormalities. She was diagnosed with a "depressive state" and "Shugan Jieyu capsules (a traditional Chinese patent medicine indicated for depression and anxiety) 0.72g bid, Lorazepam 1mg qn, and Venlafaxine extended-release tablets 150mg qd". After one week of treatment, the patient felt no improvement and discontinued the medication on her own. One month prior to admission, the patient noticed no obvious trigger for the worsening of her symptoms and sought treatment at our hospital. Since the onset of her condition, the patient was clear-headed, but her mental state was poor, with poor appetite and sleep. Over the past month, she had lost 9 kg. The patient had no significant past medical history. Personal history: Unemployed, introverted before the illness, no smoking or alcohol habits, and no history of exposure to industrial toxins, dust, radioactive substances, or contaminated water. No significant family medical history.

Neurological examination: Muscle twitching is present in the limbs, and muscle contractions are noticeable. Muscle tone in the limbs is normal, tendon reflexes are symmetric and elicited, muscle strength is 5/5, and pathological reflexes are not elicited.

Mental status examination: The patient was cooperative but exhibited a depressed mood with reduced volition. She reported negative thoughts, feeling that life was meaningless, and complained of severe somatic symptoms including widespread pain, dizziness, hand tremors, chest tightness, shortness of breath, restlessness, palpitations, abdominal bloating, and constipation. Insight was intact. Cognitive functions (orientation, memory, attention) were unimpaired.

Auxiliary examinations: Biochemical tests showed a potassium level of 3.34 mmol/L (normal range: 3.5–5.5 mmol/L). The Total IgE level was 293.55 IU/mL (normal range: 0–100 IU/mL). Routine cerebrospinal fluid (CSF) analysis revealed a weakly positive Pandy's test. The rapid CSF biochemical test showed a chloride (Cl-) level of 113.5 mmol/L (normal range: 120–130 mmol/L). The serum and CSF autoimmune encephalitis panel was negative for CSF, while the serum revealed positive findings: anti-Leucine-rich Glioma Inactivated 1 (LGI-1) IgG at a titer of 1:10 (+) and anti-Contactin-associated Protein-like 2 (CASPR2) IgG at a titer of 1:100 (++). The electromyography (EMG) showed normal motor nerve conduction in the bilateral tibial nerves, with observed afterdischarges. Both the H-reflex and F-wave of the bilateral tibial nerves also demonstrated afterdischarges. At rest, fibrillations and fasciculations were observed in the bilateral vastus medialis, bilateral gastrocnemius, and right tibialis anterior muscles. The electroencephalogram (EEG) revealed mild abnormalities, including irregular and slow alpha waves, with slightly increased slow waves. Routine blood and urine tests, tumor markers, a full infectious disease panel, and thyroid function tests revealed no significant abnormalities. Head MRI and ECG did not show any obvious abnormalities. The EMG findings in this case provide direct electrophysiological evidence of abnormal increased peripheral nerve excitability. Additionally, the immunological markers and CSF results

suggest an abnormality in the patient's immune system with possible central nervous system involvement. The positivity of specific antibodies is the core basis for the final diagnosis.

Treatment course: Upon admission, the patient was initially diagnosed with "anxiety disorder". A combination of pharmacological and non-pharmacological treatments was implemented. Oral medications included Duloxetine 40mg qd, 20mg qn; Tianeptine 10 mg tid; Alprazolam 0.2 mg bid, 0.4qn. Additionally, the patient received transcranial magnetic stimulation, biofeedback, and psychotherapy. After approximately 5 days of treatment, the patient's sleep and appetite showed slight improvement, but the widespread pain persisted. Due to the limited improvement in the patient's symptoms following anxiolytic treatment, and the inability of the anxiety disorder diagnosis to explain the patient's widespread pain and muscle twitching, our department promptly requested a neurology consultation. After completing additional tests, including electromyography (EMG) and sending autoimmune encephalitis-related antibody tests, the primary diagnosis was changed to "Morvan syndrome" five days after admission. The patient was transferred to the neurology department for continued treatment. The treatment plan was adjusted to include intravenous methylprednisolone sodium succinate 500 mg daily for 3 days, which was reduced to 240 mg daily based on the patient's condition. Carbamazepine 0.1 g was administered three times daily, and Tramadol hydrochloride extended-release tablets 0.1 g were given every 12 hours. Potassium citrate granules were given 1.46 g three times a day. It is noteworthy that on the first day of steroid use and the first day of steroid dose reduction, the patient experienced significant worsening of anxiety and depressive symptoms, restlessness, irritability, and even suicidal behavior. Therefore, the sleep medication was switched to Clonazepam 0.5 mg in the morning and 1 mg at bedtime, along with Zolpidem tartrate 10 mg at bedtime. Magnesium valproate 0.25 g was added in the morning and evening to control the patient's mood. Following active treatment, the patient's symptoms significantly improved after 15 days of hospitalization. The widespread pain resolved, the patient reported an improved mood, and there were no complaints of restlessness, palpitations, or hand tremors. On physical examination, there was no muscle twitching, and muscle contraction was normal. The patient's sleep improved, with an average of 6–7 hours of sleep per night. The patient was discharged in an improved condition. After discharge, the patient was prescribed oral Prednisone 70 mg in the morning, with a reduction of one tablet every 2 weeks until discontinued. In addition, the medications mentioned earlier for improving mood and sleep were continued. The patient did not follow up regularly at our hospital after discharge. Six months post-discharge, during a telephone follow-up, the patient reported no recurrence of widespread pain or other discomforts, felt an improvement in mood, and had discontinued all treatment medications on her own.

Diagnostic criteria: Core symptoms: Persistent widespread pain, abnormal limb sensations, refractory sleep disturbances, and significant weight loss. Physical examination: Muscle twitching in the limbs, with noticeable muscle contractions. Auxiliary examinations: The patient's electromyography (EMG) showed signs of increased peripheral nerve excitability (afterdischarges were observed in the tibial nerve and its F-waves and H-reflexes, as well as fibrillations and fasciculations in both lower limbs). Cerebrospinal fluid (CSF) routine: Pandy's test weakly positive. Serum and autoimmune encephalitis panel: Positive for anti-Leucine-rich Glioma Inactivated 1 (LGI-1) IgG and anti-Contactin-associated Protein-like 2 (CASPR2) IgG antibodies.

Discussion

For patients with Morvan syndrome without associated tumors, treatments such as corticosteroids, intravenous immunoglobulin (IVIG), and plasmapheresis generally show good efficacy. Regarding the use of IVIG, however, it is important to note that caution is required in interpreting serum and cerebrospinal fluid autoantibody results, as passively transferred antibodies from the preparation may lead to false-positive or misleading findings.[7] Despite the general effectiveness of these standard immunotherapies, patients with concomitant tumors often have poorer treatment outcomes and prognosis.[8] A 2025 case report showed that two patients with positive anti-CASPR2 antibodies significantly improved in peripheral nerve hyperexcitability and central nervous system symptoms after treatment with Efgartigimod, a novel FcRn inhibitor. The serum antibody levels quickly decreased, and the treatment was well tolerated.[9] This drug works by accelerating the degradation of IgG antibodies and has traditionally been used for treating myasthenia gravis. However, its long-term efficacy for Morvan syndrome still requires validation with larger sample sizes.[9] In addition, some reports based on traditional Chinese medicine theories and syndrome differentiation treatments have also shown promising efficacy, suggesting another potential targeted treatment

direction.[10] It is worth noting that the patient in this case experienced worsened emotional symptoms, including suicidal behavior, on both the first day of steroid pulse therapy and the first day after steroid dose reduction. This is consistent with the well-documented feature of corticosteroid use and dose changes potentially triggering anxiety, depression, or psychotic symptoms.[11] It also reminds us to be cautious of the potential neuropsychiatric imbalances that may result from rapid immunomodulatory treatment and highlights the importance of gradual dose escalation and slow tapering of corticosteroid medications.

In this case, the patient was initially diagnosed only with "anxiety disorder", and the prominent and specific somatic symptoms were categorized as somatization caused by emotional issues during the initial consultation. The examination results revealed clear evidence of objective organic abnormalities, thus not meeting the criteria for Somatic Symptom Disorder. Most patients with Morvan syndrome in the initial consultation are typically classified under psychiatric conditions, such as anxiety disorders or sleep disorders.[12] Therefore, the diagnostic and treatment process described in this case serves as a warning to psychiatrists: when patients present with symptoms and signs similar to those reported in this case, which cannot be fully explained by psychiatric disorders, consideration should be given to the possibility of Morvan syndrome.

Finally, several limitations of this case report must be acknowledged. First, as a single-case study, the findings regarding clinical presentation and treatment response may not be generalizable to the broader population of patients with Morvan syndrome. Second, the simultaneous administration of corticosteroids, antiepileptic drugs (carbamazepine, valproate), and analgesics makes it difficult to isolate the specific therapeutic contribution of each agent. This polypharmacy serves as a confounding factor in evaluating the efficacy of immunotherapy alone. Third, although the patient showed significant improvement, the follow-up period was relatively short (6 months) and conducted via telephone. Given the risk of recurrence and the potential for delayed emergence of tumors such as thymoma, longer-term monitoring is required to fully assess the prognosis and rule out paraneoplastic etiology.

Ethics Approval

Ethical approval was not required for the publication of this case report by the Ethics Committee of the Second Hospital of Lanzhou University.

Informed Consent Patient Statement

The patient signed the written informed consent, agreed to the publication of the medical case and understands that this report is intended for scientific and educational purposes.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that they have no competing interests in this work.

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