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Morvan Syndrome with CASPR2 and LGI1 Positivity Triggered by Anabolic Steroid Exposure: A Case Report

Case Report

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Abstract

Morvan syndrome is a rare autoimmune disorder involving both peripheral and central nervous systems. Patients typically present with continuous muscle twitching (myokymia), cramps, stiffness, fasciculations and autonomic dysfunction including hyperhidrosis, arrhythmias and blood pressure instability. Central nervous system involvement manifests as insomnia, psychiatric symptoms, memory impairment, and occasionally seizures. We report a 27-year-old man who presented with insomnia, severe myalgia, generalized myokymia, autonomic instability, and anxiety following anabolic steroid use. Nerve conductions revealed repetitive after-discharges and needle electromyography revealed myokymic discharges. Serum was strong positivity for CASPR2 and weak positivity for LGI [1] antibodies. The patient responded well to corticosteroids and intravenous immunoglobulin. This case report highlights a rare autoimmune neurological syndrome potentially triggered by anabolic steroid exposure.

Keywords: Morvan syndrome; CASPR2; Neuromyotonia; Anabolic steroids

Introduction

Morvan syndrome is a rare autoimmune neurological disorder characterized by peripheral nerve hyperexcitability, autonomic instability, and encephalopathy. It is often associated with antibodies to voltage-gated potassium channel complex (VGKC) proteins, including CASPR2 and LGI [1]. Morvan syndrome features pronounced peripheral nerve hyperexcitability, clinically manifesting as neuromyotonia. Patients often experience spontaneous muscle twitching (myokymia), muscle cramps, stiffness, and fasciculations.[1] Autonomic dysfunction is one of the hallmark features of Morvan syndrome and include hyperhidrosis and cardiovascular dysregulation such as tachycardia, bradycardia, or labile blood pressure.[2] Central nervous system manifestations include severe insomnia and increased psychomotor activity, often described as "agrypnia excitata"—a state of extreme and persistent sleeplessness with psychomotor and autonomic hyperactivity. Other features include hallucinations, confusion, agitation, irritability, psychosis, seizures, cognitive deficits and memory impairment.[3] Anabolic steroids may modulate immune responses and have been speculated to predispose susceptible individuals to autoimmune conditions and can also cause neuroexcitotoxicity and neuronal degeneration when given in supraphysiological doses.[4]

Case Report

A 27-year-old man presented with progressive severe generalized myalgia, paraspinal pain, excessive sweating, anxiety,

and insomnia for 2 months. Twenty days before admission, he developed multifocal twitching of limb muscles at rest. He had a History of anabolic steroid intake 10 weeks preceding the symptom onset. There were no other neurological or systemic symptoms. He had resting tachycardia (115/minute), normal blood pressure and hyperhidrosis. Cognitive functions, cranial nerves, and sensations were normal. Muscle strength, bulk, and reflexes were normal. Myokymia was present in limb muscles (lower > upper limb muscles). Motor and sensory nerve conduction parameters were normal. However, after-discharges were seen following compound muscle action potentials in median, ulnar, fibular and posterior tibial motor nerve conductions.(Figure 1) F – waves could not be identified due to the after-discharges.(Figure 2) Electromyography revealed myokymic discharges, sparse fasciculations and complex motor unit potentials in limb and paraspinal muscles.(Figure 3) Mixed nerve and cutaneous silent periods were normal in right abductor pollicis brevis. Heart rate variability was reduced at rest and during deep breathing.

Hemogram, liver function tests, renal function tests, thyroid function tests, antinuclear antibody (ANA) profile and serum creatine kinase were normal. CASPR2 antibodies were strongly positive, and LGI1 antibodies were weakly positive by cell-based assay. Mi antibody was positive on myositis panel. The Mi antibody positivity might be an incidental finding and its significance could not be ascertained. Magnetic resonance imaging of brain and spine were normal.

The patient received pulse-dose intravenous methylprednisolone (1g /day) for five days followed by intravenous immunoglobulin (2 g/kg over 5 days). There was a significant reduction in insomnia, autonomic dysfunction, and behavioral symptoms during the hospital stay. During follow up at two months patient had resolution of pain, autonomic dysfunction, myokymia. Repeat motor nerve conductions revealed disappearance of after-discharges with normal F-wave responses. (Figure 4). During follow-up, he was not initiated on further immunomodulation therapy and repeat antibody testing was not done. His last follow up was through telephonic consultation after 3 years of onset of symptoms. He currently has no recurrence of symptoms and is independent for all activities of daily living.

Discussion

This patient exhibited the classic presentation of Morvan syndrome with peripheral and central nervous system involvement and antibody positivity for both CASPR2 and LGI1. He had a probable trigger in the form of anabolic steroid exposure preceding the onset of Morvan syndrome. VGKC complex antibodies are detected in up to 72% of Morvan syndrome patients. Immunopathologically, anti-VGKC antibodies lead to downregulation of potassium ion channel function, resulting in lack of inhibition manifesting with neuronal hyperexcitability which is seen clinically as neuromyotonia, dysautonomia and CNS symptoms. [6,7] Peripheral neuronal

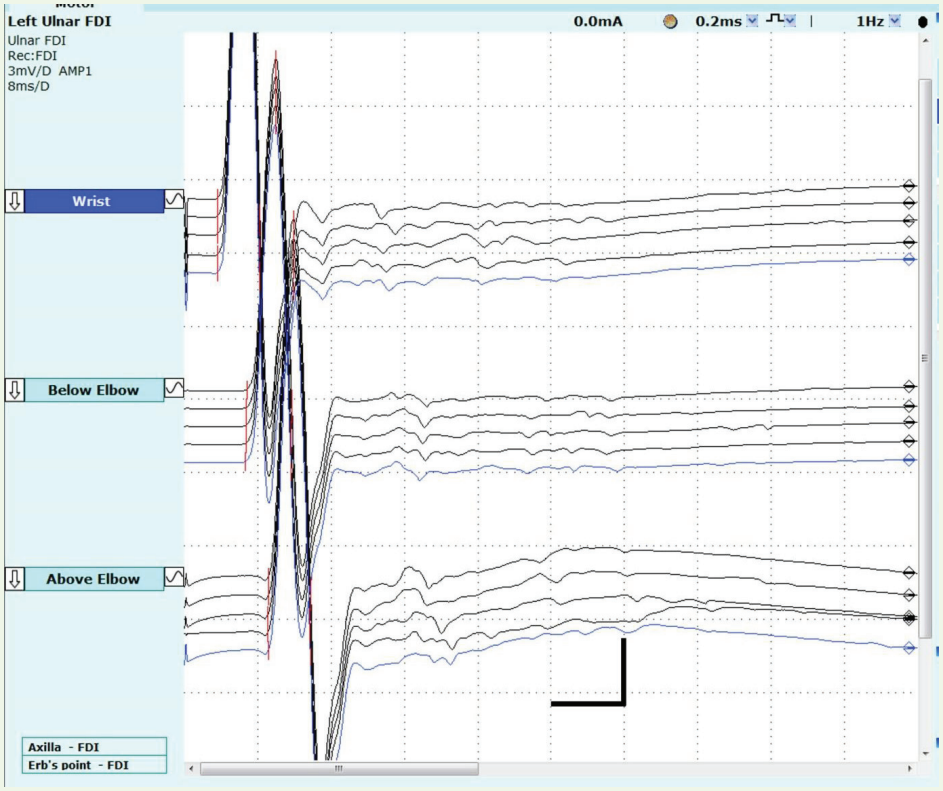

Figure 1: Motor nerve conduction study of left ulnar nerve with recording from first dorsal interosseus demonstrating the repetitive after discharges following the compound muscle action potentials at all stimulation sites. Sensitivity 3mv/d, Sweep speed 8 msec/division

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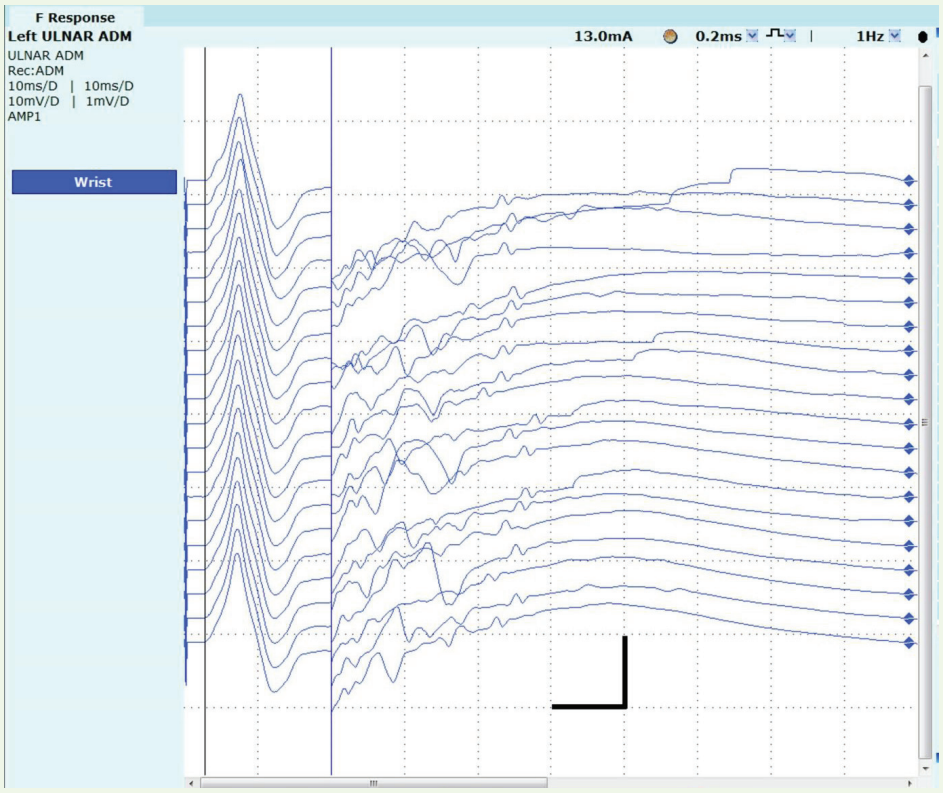

Figure 2: F wave study demonstrating the repetitive after-discharges obscuring the F – waves. Sweep 10 msec/division, Sensitivity for M wave10 mV/division and F-wave 1 mv/division

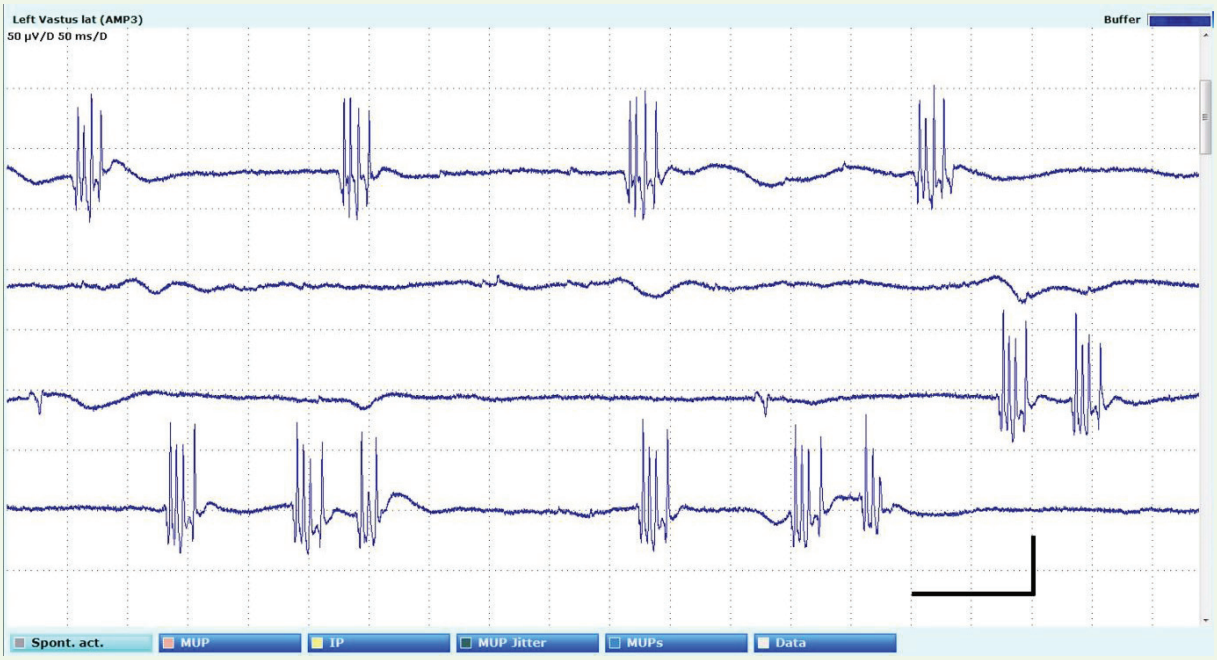

Figure 3: Needle electromyography in tibialis anterior depicting myokymic discharges. Sensitivity 50uV/d, sweep duration 1 second.

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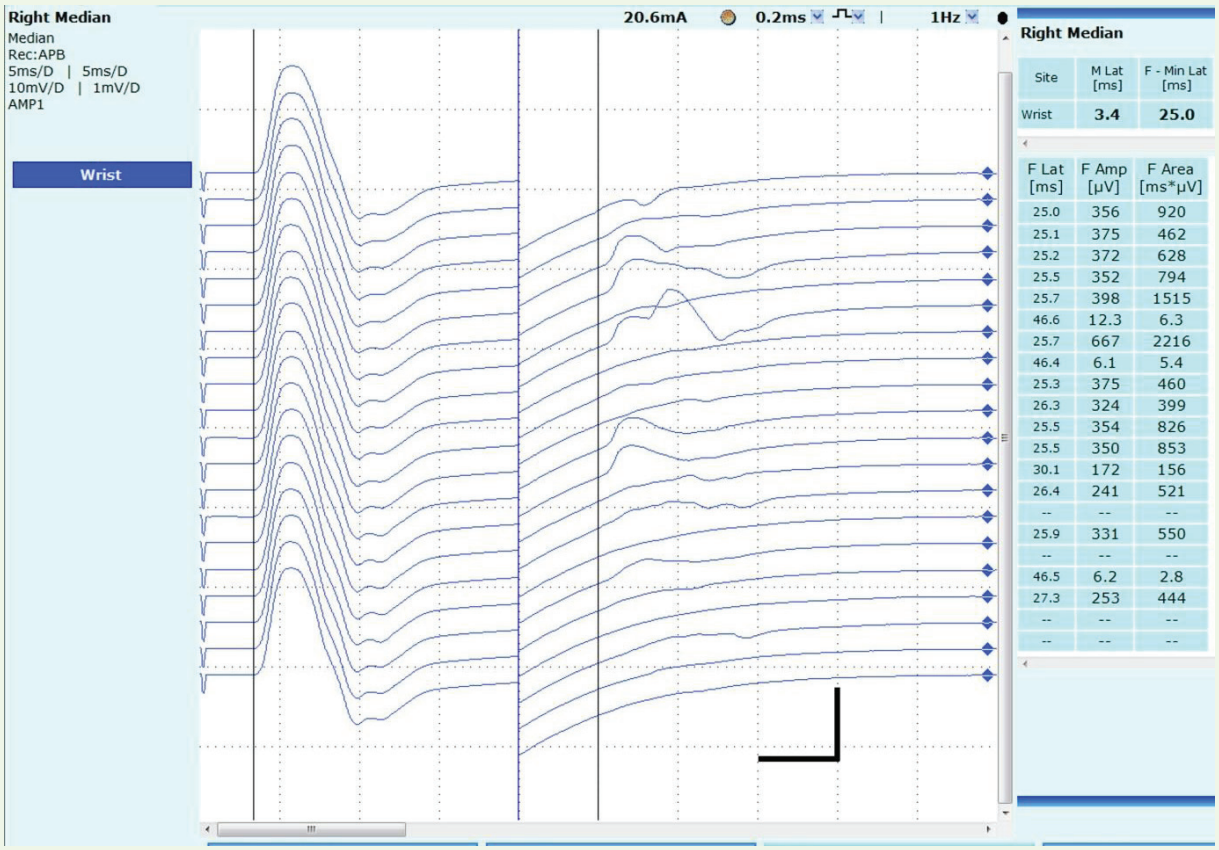

Figure 4: F wave study of right median nerve demonstrating the well demonstrated F waves with disappearance of after discharges during follow-up study.

hyperexcitability can be demonstrated with motor nerve conduction study which reveal repetitive after-discharges following the compound muscle action potential, often obscuring the F-wave. Needle EMG reveals myokymia, complex motor unit potentials and neuromyotonia. Early immunotherapy with corticosteroids and intravenous immunoglobulin therapy is critical for obtaining good clinical result. Anabolic steroids may disrupt immune tolerance by altering lymphocyte differentiation, cytokine production, and antibody generation, serving as potential triggers for autoimmune neurological syndromes.[4]

Conclusion

Morvan syndrome is a rare neurological disorder with both peripheral nerve and central nervous system involvement. This case report highlights the importance of recognizing atypical autoimmune triggers such as anabolic steroids and the importance of initiating early immunotherapy to ensure good neurological outcomes.

Conflicts of Interest

The authors declare no conflicts of interest.

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