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Case Report

Gq1b IgG-positive myeloradiculoneuropathy with spinal cord demyelination and autonomic dysfunction

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ABSTRACT

Here, we present a case of a 43-year-old woman who developed acute-onset progressive weakness in both lower limbs, with a distinct sensory level at D3-D4, and bowel/bladder incontinence, demonstrating an unusual overlap of central and peripheral nervous system involvement. Magnetic resonance imaging (MRI) revealed linear hyperintensities from C6 to D3 spinal levels while nerve conduction studies confirmed peripheral demyelination, creating a diagnostic challenge. Serological testing identified strongly positive GQ1b IgG antibodies with equivocal GQ1D reactivity, while comprehensive panels ruled out neuromyelitis optica, myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD), and paraneoplastic aetiologies. The patient showed dramatic improvement following IVIG therapy, supporting an antibody-mediated pathophysiology affecting both spinal cord and peripheral nerves. This case showcases the heterogeneity of presentation of GQ1b-associated disorders by demonstrating its potential to cause concurrent central demyelination and peripheral neuropathy. Our findings underline the importance of considering atypical presentations in anti-ganglioside antibody disorders, where immunotherapy may be effective. This type of presentation of anti GQ1b reactive disease is rarely documented in the existing literature.

Keywords: Guillain-Barré syndrome variant, Anti-GQ1b antibody, Spinal cord demyelination, IVIG responsiveness, Central-peripheral nervous system overlap, Autoimmune myelitis

INTRODUCTION

Anti-GQ1b IgG antibodies are diagnostic biomarkers for MFS (Miller Fisher Syndrome) and Bickerstaff brainstem encephalitis, both considered peripheral nervous system (PNS) disorders.[1] Rare reports describe GQ1b positivity in pharyngeal-cervical-brachial weakness or encephalitis, but spinal cord demyelination remains sparsely documented.

We present a case of GQ1b IgG-positive myeloradiculoneuropathy with autonomic dysfunction, challenging the traditional presentations. An overlap syndrome that responded to immunotherapy.

CASE REPORT

A 43-year-old woman with no known comorbidities, presented with rapidly progressive weakness in both lower limbs causing inability to ambulate, accompanied by a distinct band-like loss of pain and temperature sensation below the D3-D4 dermatomal level, developing within 24 hours. Concurrent bowel and bladder dysfunction manifested as urinary retention requiring catheterization and faecal incontinence was also there. There was history of dysuria since last 3 days for which she was on antibiotics. She had no history of fever, diarrheal episodes, vaccinations, or trauma.

Neurological examination revealed preserved higher mental and cranial nerve function. Motor assessment documented symmetric weakness: upper limbs graded 4/5 on the Medical Research Council scale, lower limbs 3/5 with proximal predominance. Deep tendon reflexes were absent at the patellar and achilles tendons but were present normally in the upper extremities. Sensory testing revealed a sharp sensory level at T3-T4 for pinprick and temperature perception, with diminished vibration sense and proprioception in the lower extremities. Autonomic dysfunction was evidenced by bladder incontinence and faecal incontinence. Plantar responses were flexor bilaterally.

Diagnostic evaluation included spinal MRI, which revealed linear STIR hyperintensities in the ventral cord extending contiguously from C6 to D3, involving <50% cross-sectional area (Figure 1), without contrast enhancement. Brain magnetic resonance imaging (MRI) was unremarkable. Nerve conduction studies confirmed a demyelinating polyradiculoneuropathy with prolonged F-wave latencies (median nerve: 38 ms; normal <31 ms) and slowed conduction velocities (peroneal nerve: 32 m/s; normal >40 m/s). Cerebrospinal fluid analysis revealed normal cell counts (2 lymphocytes/µl), protein (38 mg/dl), and glucose, absent oligoclonal bands, and no albuminocytological dissociation. Serological testing demonstrated strongly positive anti-GQ1b IgG antibodies (ELISA 1:6400) with equivocal GQ1D reactivity, while comprehensive autoimmune panels (anti-AQP4, anti-MOG, paraneoplastic antibodies) and extended ganglioside antibodies (GM1, GD1a, GD1b, GT1b) returned negative. Urinary culture revealed growth of Escherichia coli (pan sensitive to antibiotics).

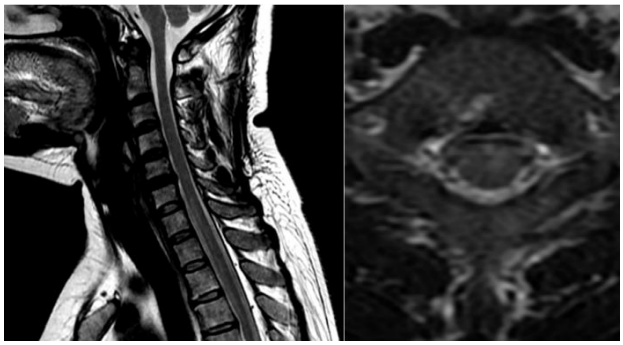

Figure 1: Sagittal STIR MRI showing ventral cord hyperintensity extending from C6 to D3.

She was managed with intravenous methylprednisolone (1 g daily ×3 days) followed by IV immunoglobulin (0.4 g/kg/day ×5 days), with concurrent ceftriaxone (2 g/day ×7 days) for UTI. Within 72 hours, motor function improved significantly (lower limb power: 4/5), enabling supported ambulation. By day 7, bladder function recovered sufficiently for catheter removal, though intermittent bowel incontinence persisted. The patient achieved a satisfactory level of independence activities of daily living and was discharged on day 10 with residual neurogenic bowel dysfunction.

Three-month follow-up documented sustained motor recovery (lower limb power 5/5) with resolution of sensory deficits. Urodynamic studies confirmed normal bladder function, while chronic constipation requiring laxative regimens persisted. This case represents an atypical presentation GQ1b IgG-positive overlap syndrome with concurrent central demyelination and peripheral neuropathy, expanding the phenotypic spectrum of anti-ganglioside antibody disorders.

DISCUSSION

This case presents an uncommon neurological phenotype that challenges the traditional presentations of anti-GQ1b antibody syndromes. While GQ1b IgG positivity typically manifests as Miller Fisher syndrome with its classic triad of ophthalmoplegia, ataxia, and areflexia, our patient showed a striking combination of spinal cord demyelination and peripheral neuropathy with autonomic dysfunction. The ventral cord localization suggests potential antibody targeting of gray matter gangliosides (e.g., anterior horn cells, autonomic nuclei), expanding the known pattern of involvement of GQ1b IgG, which usually involve only peripheral nervous system.[2,3] The co-occurrence of central demyelination with peripheral nerve involvement suggests a possible overlap syndrome where anti-GQ1b antibodies may have cross-reacted with shared epitopes in both central and peripheral neural tissues.[4] This hypothesis is supported by the patient's dramatic response to IVIG therapy, which typically targets antibody-mediated peripheral nerve disorders but showed efficacy in this case with concurrent central nervous system involvement.

The pathogenic mechanisms underlying this unique presentation may involve molecular mimicry triggered by the preceding urinary tract infection, with bacterial antigens potentially sharing structural similarities with both spinal cord and peripheral nerve gangliosides. The strongly positive GQ1b IgG antibodies, while typically associated with peripheral nerve targets, may have exhibited atypical binding to spinal cord gray matter antigens, particularly in autonomic regions. This could explain the prominent bowel and bladder dysfunction that persisted despite improvement in motor symptoms. The equivocal GQ1D reactivity raises additional questions about potential antibody cross-reactivity with other gangliosides present in spinal cord tissue. The absence of albuminocytological dissociation in cerebrospinal fluid, typically seen in classic Guillain-Barré syndrome, further underlines the atypical nature of this case and suggests a distinct pathophysiological process.[5]

Critical differential diagnoses were systematically excluded through comprehensive testing. Neuromyelitis optica spectrum disorder was ruled out by negative anti-AQP4 antibodies and the presence of ventral predominant

short-segment spinal cord lesions rather than longitudinally extensive transverse myelitis. Anti-MOG antibody-associated disease was considered unlikely in the setting of negative serology and the atypical clinical presentation. Paraneoplastic aetiologies were excluded through negative antibody panels and absence of malignancy on imaging. Classic Guillain-Barré syndrome variants were insufficient to explain the spinal cord involvement and discrete sensory level. Vitamin B12 deficiency and other metabolic causes were excluded through normal laboratory testing. The occurrence of central and peripheral nervous system involvement with isolated GQ1b IgG positivity makes this case particularly interesting.

The excellent response to IVIG suggests that antibody-mediated mechanisms predominated in both the central and peripheral nervous system pathology, supporting the use of immunotherapy even when atypical features are present. The partial response to steroids followed by more dramatic improvement with IVIG may indicate that humoral immunity played a greater role than cellular mechanisms in disease pathogenesis. The persistence of bowel dysfunction despite motor recovery highlights the vulnerability of autonomic pathways to immune-mediated injury and suggests that early, aggressive immunotherapy may be warranted in similar cases to prevent long-term autonomic sequelae. This case emphasizes the importance of considering antibody testing even in presentations that deviate from classic syndromes. The residual neurogenic bowel dysfunction observed during follow-up points to the need for prolonged monitoring and rehabilitation in such overlap syndromes, as autonomic recovery often lags motor improvement. Whether similar cases represent a distinct subgroup within the spectrum of anti-ganglioside antibody disorders, which requires modified treatment approaches, is a question that remains to be answered.

CONCLUSION

This case represents an expansion of the recognized clinical spectrum of GQ1b IgG-associated neurological disorders, demonstrating that anti-ganglioside antibodies can produce a unique overlap syndrome combining central demyelination and peripheral neuropathy. The persistence of bowel dysfunction despite motor recovery highlights the selective vulnerability of spinal autonomic pathways in this novel phenotype, suggesting that ganglioside antibody targets may extend beyond conventional peripheral nerve antigens. This case underlines the importance of comprehensive imaging and antibody testing in atypical Guillain-Barré-like presentations, as accurate diagnosis has direct therapeutic implications. The efficacy of IVIG in this context supports its use even when central nervous system involvement is present. Clinicians should keep a high index of suspicion for such overlap syndromes when evaluating patients with mixed central and peripheral neurological deficits, as timely immunotherapy may significantly improve outcomes.

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