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CASE REPORT

When the Eyes Speak First: A Rare Case of Anti-GQ1b Syndrome in a Young Adult

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ABSTRACT

Anti-GQ1b syndrome, a rare autoimmune neurological disorder, presents a diagnostic challenge due to its overlapping central and peripheral nervous system features. We report a case of a 21-year-old male who presented with binocular diplopia, cranial nerve involvement, and limb weakness following a recent febrile illness. Despite initial unresponsiveness to steroid therapy, the patient showed remarkable improvement with intravenous immunoglobulin (IVIg), and a delayed anti-GQ1b antibody test confirmed the diagnosis. This case emphasizes the importance of early clinical suspicion and aggressive immunotherapy to prevent long-term complications.

KEYWORDS

• Anti-GQ1b syndrome • Bickerstaff brainstem encephalitis • Miller Fisher syndrome • Ophthalmoplegia • IVIg therapy • Autoimmune neuropathy • Diplopia Central and peripheral demyelination

INTRODUCTION

Anti-GQ1b syndrome encompasses a spectrum of post-infectious autoimmune neuropathies, most notably **Miller Fisher Syndrome (MFS)** and **Bickerstaff Brainstem Encephalitis (BBE)**. The unifying biomarker is the anti-GQ1b antibody, which targets gangliosides abundant in cranial nerves and peripheral nerves, particularly those involved in eye movement and facial expression.

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Despite its rarity, recognizing this syndrome early is crucial due to its potential to progress rapidly and cause serious morbidity such as **blindness, quadriparesis, dysphagia**, or **respiratory failure**. Prompt treatment with IVIg or plasmapheresis can be lifesaving

Case Presentation:

A **21-year-old previously healthy male** presented with a **3-day history** of:

- Progressive binocular diplopia (initially horizontal, now constant)
- Frontal headache and retro-orbital pain
- Mild photophobia
- No history of trauma or previous neurological illness

Notably, he had a **fever and sore throat 7 days prior**, lasting 3 days, which resolved spontaneously.

Initial Evaluation (at an outside hospital):

- He was evaluated for possible sinusitis or meningitis.
- Basic labs and CT head were unremarkable.
- He was started on symptomatic treatment but returned 3 days later with **worsening diplopia**, **generalized weakness**, and **new leg cramps**.

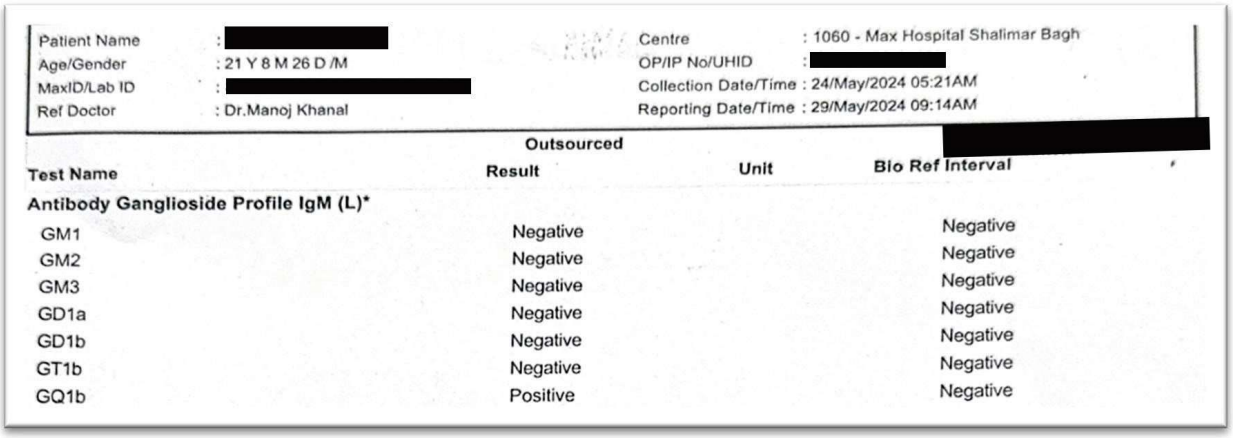
On Presentation to Our Center:

Vital Signs: Afebrile, HR 82 bpm, BP 124/76 mmHg, SpO_2 98% RA **Neurological Examination:**

- **Cranial Nerves:**
  - Bilateral lateral rectus palsy (suggestive of abducens nerve dysfunction)
  - Bilateral LMN facial weakness (forehead spared)
  - Normal pupils and visual acuity, no ptosis
- **Motor:**
  - Mild symmetric weakness in lower limbs (MRC 4/5 distally)
- **Reflexes:**
  - Hyperreflexia in lower limbs
- **Sensation & Coordination:** Normal
- **Gait:** Mildly unsteady due to diplopia

Investigations:

- **MRI Brain and Orbits with contrast:** Normal
- **CSF Analysis:** Clear, acellular, normal glucose and protein
- **Visual Evoked Potentials (VEP):** Mild bilateral optic neuropathy
- **Nerve Conduction Studies (NCV):** Mild demyelinating sensorimotor polyneuropathy in lower limbs
- **ANA:** Positive
- **Anti-GQ1b Antibody:**
- **Other Labs:** CBC, ESR, CRP, renal/liver function—normal



Patient Name : [redacted]
Age/Gender : 21 Y 8 M 26 D /M
MaxID/Lab ID : [redacted]
Ref Doctor : Dr.Manoj Khanal

Centre : 1060 - Max Hospital Shalimar Bagh
OP/IP No/UHID : [redacted]
Collection Date/Time : 24/May/2024 05:21AM
Reporting Date/Time : 29/May/2024 09:14AM

Outsourced [redacted]

Test Name	Result	Unit	Bio Ref Interval
Antibody Ganglioside Profile IgM (L)*			
GM1	Negative		Negative
GM2	Negative		Negative
GM3	Negative		Negative
GD1a	Negative		Negative
GD1b	Negative		Negative
GT1b	Negative		Negative
GQ1b	Positive		Negative

Management and Clinical Course:

Given the presence of cranial nerve involvement, mild polyneuropathy, hyperreflexia, and preceding viral illness, a **central demyelinating disorder** was considered. The patient was initially started on **IV methylprednisolone**

(1g/day for 5 days) for suspected autoimmune encephalitis/demyelination.

However, there was **no clinical improvement**, prompting re-evaluation of the diagnosis.

A diagnosis of **Bickerstaff Brainstem Encephalitis (BBE)**, part of the **anti-GQ1b spectrum**, was suspected due to:

- Ophthalmoplegia
- Facial weakness
- Polyneuropathy
- Reflex changes

The patient was then started on **IVIg (0.4 g/kg/day for 5 days)**.

Clinical Response:

- By Day 3: Diplopia started resolving; energy levels improved
- By Day 5: Able to ambulate normally, facial tone improved
- Discharged on Day 7 in stable condition with only **residual diplopia on extreme gaze**
- **Anti-GQ1b antibodies returned positive on Day 10 post-discharge**, confirming diagnosis

DISCUSSION

Anti-GQ1b syndrome represents a unique overlap between central and peripheral nervous system autoimmunity. The GQ1b ganglioside is heavily expressed in the **oculomotor, abducens, and trochlear nerves**, as well as in proprioceptive fibers and brainstem structures, explaining the spectrum of symptoms.

Spectrum Includes:

- **Miller Fisher Syndrome (MFS):** Ophthalmoplegia, ataxia, areflexia
- **Bickerstaff Brainstem Encephalitis (BBE):** Ophthalmoplegia, altered consciousness, hyperreflexia or pyramidal signs
- **Overlap with Guillain-Barré Syndrome (GBS):** May progress to limb weakness or respiratory involvement

Our patient did not exhibit altered mental status, but the presence of hyperreflexia, cranial nerve dysfunction, and facial palsy placed him within the **BBE end of the spectrum**.

Key Diagnostic Considerations:

- Normal MRI and CSF do **not exclude** BBE
- VEP and NCV may reveal subclinical demyelination
- Positive **anti-GQ1b antibodies** are diagnostic, but clinical treatment should **not be delayed** while awaiting results

Therapeutic Insights:

- IVIg is more consistently effective than steroids in anti-GQ1b syndromes
- Delay in treatment may lead to irreversible neurological deficits
- Plasmapheresis is an alternative if IVIg is contraindicated or ineffective

CONCLUSION

This case underscores the importance of **clinical suspicion** in diagnosing anti-GQ1b syndrome, especially in young patients presenting with rapid-onset ophthalmoplegia and combined central and peripheral neurological signs following an infection.

A normal MRI or CSF does **not rule out serious neuroimmunological disease**. Early initiation of IVIg therapy can dramatically alter the disease course, as seen in our patient who made a near-complete recovery.

Physicians should consider **anti-GQ1b antibody syndromes** in any patient with:

- Diplopia with cranial nerve involvement
- Recent viral illness
- Evidence of both CNS and PNS dysfunction

Learning Points:

- **Don't dismiss diplopia**—it can be the first sign of serious demyelinating disease.
- **Consider anti-GQ1b syndrome** when both CNS and PNS signs coexist, especially post-infection.
- **Start IVIG early**—waiting for serology confirmation may cost precious neurological function.

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