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Letter to the Editor

Before GBS is Attributed to a Preceding Shigella Infection, Alternative Infectious, Toxic, Autoimmune, and Traumatic causes must be Ruled out and the Diagnosis of GBS Confirmed

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Letter to the Editor

We read with interest the article by Kaur *et al*. regarding *Shigella* as a potential trigger for Guillain-Barré syndrome (GBS) [1]. The authors proposed the following: Testing for *Shigella* should be integrated into the diagnostic workup for GBS, particularly during pandemic outbreaks; drinking water should be systematically monitored for pathogens and improved globally; research into cross-reactivity between *Shigella* LOS epitopes and human gangliosides should be advanced; and new insights should be sought to bridge knowledge gaps regarding the immunological mechanisms underlying *Shigella*-associated GBS [1]. While these observations are highly valuable, certain points warrant further discussion.

First, characterizing the specific GBS subtypes caused by *Shigella* infections is crucial for elucidating the underlying pathophysiology. A particularly important question is whether the radiculitis results from damage to the axolemma in the internodal regions or from impairment of Schwann cell membranes. To identify the structure potentially targeted by antibodies originally directed against *Shigella* epitopes, one must first determine the specific neuronal structure that serves as the actual target for these antibodies. Electrophysiological studies can be helpful in this regard; therefore, all patients with suspected GBS should undergo nerve conduction studies - at the very least to distinguish between axonal damage, demyelination, or a combination of both.

A second point is that infections such as shigellosis are typically treated with fluoroquinolones (e.g., ciprofloxacin), macrolides (e.g., azithromycin), cephalosporins (e.g., ceftriaxone), or other agents [2]. Since fluoroquinolones have been reported to trigger neuropathies and GBS [3], it is crucial to know which antibiotics the patients received during the GBS outbreak in Puna. To avoid overlooking neuronal toxicity resulting from the administration of neurotoxic antibiotics or concomitant substances, all medications administered during the hospital stay must be taken into account and factored into pathophysiological considerations.

A third point is that establishing a causal link between *Shigella* and GBS requires the GBS diagnosis to be supported by appropriate investigations and to meet currently accepted diagnostic criteria (Brighton criteria). Patients in whom the diagnosis is merely suspected or cannot be confirmed beyond doubt should be excluded from such studies. Since *Shigella* can also cause neuropathies without nerve root involvement [4], it is important to differentiate between radiculopathy, plexopathy, polyneuropathy, and transmission disorders.

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