Sarcouncil Journal of Internal Medicine and Public Health

ISSN(Online): 2945-3674

Volume- 04| Issue- 02| 2025

Letter to the Editor **Received:** 11-02-2025| **Accepted:** 23-02-2025 | **Published:** 04-03-2025

Before Ciprofloxacine is Accused of Causing Neuropathy in Gastroenteritis, GBS should be Off the Table

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Keywords: ciprofloxacin, gastroenteritis, Guillain-Barre syndrome, neuropathy, side effect

COMMENTARY

The interesting case report by Refaeian, *et al*., about a 42-year-old man with arterial hypertension and diverticulitis attracts attention, but revisions are required. Ten days after taking ciprofloxacin for gastroenteritis for 10 days the index patient developed progressive quadriparesis with lower-limb predominance, but without sensory disturbances [Refaeian, A. *et al*., 2023]. Because nerve conduction studies (NCSs) revealed mixed axonal and demyelinating polyneuropathy and cerebrospinal fluid (CSF) studies suggested Guillain-Barre syndrome, intravenous immunoglobulins (IVIGs) were administered, resulting in partial but significant recovery until discharge [Refaeian, A. *et al*., 2023]. Despite these findings, the patient was diagnosed with ciprofloxacin-induced polyneuropathy [Refaeian, A. *et al*., 2023]. The study attracts attention, but some issues should be he addressed.

The first point is that we disagree with the diagnosis of ciprofloxacin-induced neuropathy because the causal relationship between ciprofloxacin and neuropathy remained unproven [Refaeian, A. *et al*., 2023]. There are several arguments against a causal connection. First, the latency between administration of ciprofloxacin and the onset of quadriparesis was short (10 days) [Refaeian, A. *et al*., 2023], arguing against a cumulative toxic effect. Secondly, the patient suffered from chronic diverticulitis, which in turn can be responsible for neuropathy [Igarashi, M. *et al*., 1981]. Thirdly, the disease course and the therapeutic effect of IVIG's argue against ciprofloxacin as the causative agent of neuropathy. Partial recovery of weakness and neuropathy after IVIGs at discharge is more suggestive of GBS than ciprofloxacin-induced neuropathy, as subsequent full recovery is expected in a shorter time after discontinuation.

The second point is that the microbial pathogen responsible for gastroenteritis has not been identified and not reported. To assess whether the index patient had ciprofloxacin-induced neuropathy or GBS due to gastroenteritis, it is essential to know the pathogen as typically either vaccinations or specific pathogens such campylobacter jejunii, myocoplasma pneumoniae, cytomegalyvirus, or others [Mateen, F. J. *et al*., 2011] trigger the development of GBS.

The third point is the contradiction between the statement "CSF studies were negative for GBS" and the statement that "the patient had elevated levels of CSF protein" [Refaeian, A. *et al*., 2023]. One of the diagnostic criteria for GBS according to the Brighton criteria is an increased CSF protein content without pleocytosis (dissociation-cytoalbuminique) [Finsterer, J. *et al*., 2022]. Because the patient had gastroenteritis shortly before the onset of motor neuropathy, progressive course, dissociation cyto-albuminique, and responded positively to IVIGs, the diagnosis of GBS is more likely than ciprofloxacin-induced neuropathy.

The fourth point is that the patient received duloxetine and pregabalin for quadriparesis without experiencing neuropathic pain. It is incomprehensible why these two medications were administered to a patient with quadriparesis but no sensory impairment. We should know the reason for administering these two drugs.

A fifth point is that CSF studies were not repeated. In GBS, dissociation cytoalbuminique often only becomes more pronounced several days after the onset of clinical symptoms of GBS. It is also imperative to tule out any infectious cause of radiculitis by performing CSF culture, viral panel, and fungal culture.

Although there is evidence that ciprofloxacin and fluoroquinolone can cause neuropathy, there are also studies that have not found an increased risk of neuropathy from these medications. Since ciprofloxacin has also been reported to cause optic

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DOI- https://doi.org/10.5281/zenodo.14962914

neuropathy and small fiber neuropathy, we should know whether there was evidence for optic neuropathy or small fiber neuropathy in the index patient. It should also be ruled out that the patient suffered from myasthenia, as there are reports that ciprofloxacin worsen myasthenia. Since quinolones can be complicated by hyperglycemia [Althaqafi, A. *et al*., 2021], we should also know whether ciprofloxacin caused an increased HbA1c or not.

In conclusion, before diagnosing ciprofloxacin-induced neuropathy in the index patient, GBS should have been definitively excluded. The clinical presentation and treatment response suggest GBS rather than ciprofloxacin-induced neuropathy.

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Source of support: Nil; **Conflict of interest:** Nil.

Cite this article as:
Finsterer, J. "Before Ciprofloxacine is Accused of Causing Neuropathy in Gastroenteritis, GBS should be Off the Table." *Sarcouncil Journal of Internal Medicine and Public Health* 4.2 (2025): pp 5-6.

Publisher: SARC Publisher