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Letter to the Editor

Before Shigella is Blamed for Guillain-Barré Syndrome, All Other Causes must be Reliably Ruled Out

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Letter to the Editor

We read with interest the article by Kaur *et al*. speculating on the role of Shigella as the causative agent of a recent GBS outbreak in Pune [1]. It was noted that Shigella, due to its neuroinvasive potential and its proven involvement in the development of diseases of the central and peripheral nervous systems (CNS, PNS), could also be responsible for GBS [1]. This hypothesis was supported by the citation of some cases in which GBS was attributed to Shigella infection [1]. These considerations are valuable, but several points warrant discussion.

First, it is essential to rule out all other possible causes before considering Shigella as a cause of GBS. Since Shigella is only a rare cause of GBS, more common causes must be thoroughly ruled out. These include infections with Campylobacter jejuni (the most common cause of GBS), Haemophilus influenzae, Mycoplasma pneumoniae, E. coli, the varicella-zoster virus, the human immunodeficiency virus, the Zika virus, the Epstein-Barr virus, the influenza virus, the SARS-CoV-2 virus, hepatitis A, B, C, D, and E viruses, cytomegalovirus, rubella, West-Nile virus, Japanese encephalitis virus, and chikungunya [2]. In addition, vaccinations, immunological disorders, trauma, and sepsis must be considered as triggers of GBS [2]. Shigella has rarely been described as a trigger of GBS [3, 4]. In Khour's study, Shigella was suspected as the cause in only 19 cases of GBS. However, the infection Shigella boydii could be confirmed by stool culture in only a single case [4]. Leaver *et al*. reported a patient with polyneuropathy following a Shigella infection. However, it was unclear whether the polyneuropathy was already present before the infection [5].

The second point is that neurological manifestations of a Shigella infection do not necessarily mean that patients with a Shigella infection will also develop GBS. Convulsions, seizures, loss of consciousness, encephalopathy, and Ekiri syndrome are CNS manifestations of Shigella infection, but not disorders of the PNS. Since GBS is a PNS disorder - with the exception of Bickerstaff brainstem encephalitis - the fact that a Shigella infection triggers a CNS disorder does not mean that it also causes GBS.

The third point is that Shigella is a widespread infection that occurs worldwide, yet there are only a few cases of GBS that have been linked to Shigella. Given the high prevalence of Shigella infections, particularly in the tropics and in developing countries, one would expect a significantly higher incidence of GBS among those with confirmed Shigella infections. The small number of GBS cases reported to date in association with Shigella suggests that there is no causal relationship.

The fourth point is that in all cases where GBS has been linked to Shigella [3, 4], alternative and more common causes have not been sufficiently ruled out. Before Shigella can be held responsible for causing GBS, alternative causes must be systematically ruled out. In addition, an animal model must be developed that unequivocally confirms that infection with any strain of Shigella causes polyradiculitis.

Overall, Shigella can only be held responsible for GBS if a causal relationship has been demonstrated by sufficient clinical and experimental data.

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