Norovirus and C. Jejuni: Triggering a Guillain-Barré Syndrome Outbreak in Pune?

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Sir,

The recent cluster of Guillain-Barré Syndrome (GBS) cases in Pune has raised significant public health concerns, particularly due to its potential link with infectious agents like Norovirus and Campylobacter jejuni. In India, Norovirus is a leading cause of acute gastroenteritis across all age groups, and *C. jejuni* is frequently implicated in foodborne illnesses.¹⁻³ This potential link between GBS and these prevalent enteric pathogens warrants immediate attention, not only due to the acute neurological complications of GBS but also because of its potential to strain healthcare resources and impact long-term patient outcomes.⁴ Addressing this burgeoning public health challenge requires a swift and comprehensive response to mitigate further spread and protect vulnerable populations.

Guillain-Barré syndrome is an acute, immunemediated polyneuropathy characterized by rapidly progressive limb weakness and diminished reflexes.⁴ The hallmark of GBS is the body's immune system mistakenly attacking the peripheral nervous system, leading to demyelination and axonal damage.⁵ This autoimmune response is often triggered by a preceding infection, with *Campylobacter jejuni* and Norovirus being among the most commonly implicated pathogens.⁶ The clinical presentation of GBS typically involves ascending weakness, starting in the legs and progressing upwards, along with paresthesias (tingling sensations). In severe cases, it can lead to lifethreatening respiratory muscle paralysis.⁴ Diagnosis often relies on clinical findings, nerve conduction studies, and cerebrospinal fluid analysis.⁷ While most patients recover with appropriate medical care, including supportive measures and immunomodulatory therapies, some may experience long-term neurological deficits, such as persistent weakness or fatigue.⁸

Both Norovirus and C. jejuni are significant contributors to gastroenteritis globally, and their prevalence in India is well-documented.9,10 Norovirus, a highly contagious RNA virus, spreads primarily through the fecal-oral route, often via contaminated food and water, and person-to-person contact.¹¹ Campylobacter jejuni, a Gram-negative bacterium, is commonly associated with the consumption of undercooked poultry and contaminated water sources. These pathogens possess virulence factors that enable them to colonize the gastrointestinal tract and evade host immune responses, contributing to their pathogenicity.9 These infections may trigger an autoimmune response through molecular mimicry, where pathogen structures resemble nerve components, leading to immune-mediated nerve damage.¹² For instance, lipooligosaccharides in C. jejuni have been shown to mimic gangliosides in peripheral nerves.13

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The incidence of GBS varies globally, with estimates ranging from 0.6 to 4 cases per 100,000 population per year.¹⁴ In India, studies have reported an incidence of approximately 1.3 cases per 100,000 population.¹⁵ While GBS can affect individuals of any age, it is more common in adults and older individuals.⁴ Recognizing the potential for serious complications and the need for prompt medical intervention, understanding the epidemiological trends and risk factors associated with GBS is crucial for effective public health management.

Despite the growing concern, specific details regarding the recent GBS outbreak in Pune, such as the exact number of cases, demographics, and confirmatory laboratory findings, remain limited. However, reports suggest a notable increase in GBS cases presenting to hospitals in Pune within a short timeframe, raising concerns of a potential outbreak. Preliminary investigations indicate a possible link to Norovirus and C. jejuni infections, although the extent of this association remains to be fully established. It is crucial to acknowledge the limitations of the currently available information. Without comprehensive epidemiological data, it is challenging to draw definitive conclusions about the outbreak's magnitude and potential risk factors. Furthermore, the lack of widespread laboratory testing for Norovirus and C. jejuni in all suspected GBS cases hinders the ability to establish a conclusive link between these pathogens and the observed surge in neurological illness.

While the temporal association between reported gastroenteritis cases and the subsequent rise in GBS cases suggests a possible link to Norovirus and *C. je-juni*, alternative explanations must also be considered. Other infectious agents known to trigger GBS, such as cytomegalovirus, Epstein-Barr virus, and Zika virus, should be investigated.⁶ Additionally, the possibility of other environmental factors or triggers contributing to the outbreak cannot be ruled out.

Given the potential for further cases, immediate epidemiological investigations and public health interventions are imperative to prevent escalation of the GBS outbreak in Pune. These should include:

Enhanced surveillance: Active case finding and systematic data collection, including detailed clinical information, demographics, and potential exposures.

Laboratory confirmation: Widespread testing for Norovirus, *C. jejuni*, and other potential pathogens in GBS cases.

Environmental investigation: Assessing potential sources of contamination, such as food and water sources.

Serological studies: Conducting serological surveys to determine the extent of exposure and assess population immunity.

Genomic sequencing: Analysing the genetic makeup of circulating strains to identify variants and trace their origins.

Public awareness campaigns utilizing diverse channels (e.g., targeted health education programs, social media engagement, and collaboration with community leaders) should be launched to educate the community about GBS, its symptoms, and preventive measures, such as safe food handling and water hygiene.

The recent GBS outbreak in Pune highlights the urgent need for strengthened surveillance and preventive strategies against enteric infections. We urge public health authorities to take immediate action to investigate the outbreak thoroughly, identify sources of infection, and implement targeted control measures to contain its spread. This should include enhanced surveillance efforts, laboratory confirmation of pathogens, and environmental investigations. In the long term, strengthening surveillance systems for GBS and related pathogens is crucial for early detection and timely intervention. Furthermore, continued investment in research is essential to elucidate the complex interplay between these infections and GBS, paving the way for more effective preventive and therapeutic strategies. A collaborative and proactive approach involving healthcare professionals, public health agencies, and the community is paramount in safeguarding public health and mitigating the impact of future outbreaks.

REFERENCES

- Kambhampati A, Koopmans M, Lopman BA. Burden of norovirus in healthcare facilities and strategies for outbreak control. J Hosp Infect. 2015 Apr;89(4):296-301. DOI: https://doi. org/10.1016/j.jhin.2015.01.011 PMid:25726433
- Ahmed SM, Hall AJ, Robinson AE, Verhoef L, Premkumar P, Parashar UD, et al. Global prevalence of norovirus in cases of gastroenteritis: a systematic review and meta-analysis. Lancet Infect Dis. 2014 Aug;14(8):725-30. DOI: https://doi.org/10.1016/S1473-3099(14)70767-4 PMid:24981041
- Kirk MD, Pires SM, Black RE, Caipo M, Crump JA, Devleesschauwer B, et al. World Health Organization Estimates of the Global and Regional Disease Burden of 22 Foodborne Bacterial, Protozoal, and Viral Diseases, 2010: A Data Synthesis. PLoS Med. 2015 Dec;12(12):e1001921. DOI: https://doi.org/10. 1371/journal.pmed.1001921 PMid:26633831
- 4. Nguyen TP, Taylor RS. Guillain-Barre Syndrome. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 [cited 2025 Jan 24]. Available from: http://www.ncbi.nlm. nih.gov/books/NBK532254/
- 5. Berciano J. Pathophysiology and markers of very early classic Guillain-Barré syndrome. NeuroMarkers. 2024 Sep 1;1(1): 100003. DOI: https://doi.org/10.1016/j.neumar.2024.100003
- Finsterer J. Triggers of Guillain-Barré Syndrome: Campylobacter jejuni Predominates. Int J Mol Sci. 2022 Nov 17;23(22): 14222. DOI: https://doi.org/10.3390/ijms232214222
- Leonhard SE, Mandarakas MR, Gondim FAA, Bateman K, Ferreira MLB, Cornblath DR, et al. Diagnosis and management of Guillain-Barré syndrome in ten steps. Nat Rev Neurol. 2019 Nov;15(11):671-83. DOI: https://doi.org/10.1038/s41582-019-0250-9 PMid:31541214 PMCid:PMC6821638

- Elendu C, Osamuyi EI, Afolayan IA, Opara NC, Chinedu-Anunaso NA, Okoro CB, et al. Clinical presentation and symptomatology of Guillain-Barré syndrome: A literature review. Medicine (Baltimore). 2024 Jul 26;103(30):e38890. DOI: https://doi.org/10.1097/MD.00000000038890
- Kaakoush NO, Castaño-Rodríguez N, Mitchell HM, Man SM. Global Epidemiology of Campylobacter Infection. Clin Microbiol Rev. 2015 Jul;28(3):687-720. DOI: https://doi.org/10. 1128/CMR.00006-15 PMid:26062576 PMCid:PMC4462680
- Deb S, Mondal R, Lahiri D, Shome G, Roy AG, Sarkar V, et al. Norovirus-associated neurological manifestations: summarizing the evidence. J Neurovirol. 2023 Aug 1;29(4):492-506. DOI: https://doi.org/10.1007/s13365-023-01152-0
- Robilotti E, Deresinski S, Pinsky BA. Norovirus. Clin Microbiol Rev. 2015 Jan;28(1):134-64. DOI: https://doi.org/10.1128/ CMR.00075-14 PMid:25567225 PMCid:PMC4284304
- 12. Laman JD, Huizinga R, Boons GJ, Jacobs BC. Guillain-Barré syndrome: expanding the concept of molecular mimicry. Trends

Immunol. 2022 Apr;43(4):296-308. DOI: https://doi.org/10. 1016/j.it.2022.02.003 PMid:35256276 PMCid:PMC9016725

- Godschalk PCR, Kuijf ML, Li J, St. Michael F, Ang CW, Jacobs BC, et al. Structural Characterization of Campylobacter jejuni Lipooligosaccharide Outer Cores Associated with Guillain-Barré and Miller Fisher Syndromes. Infect Immun. 2007 Mar;75(3):1245-54. DOI: https://doi.org/10.1128/IAI.00872-06 PMid:17261613 PMCid:PMC1828588
- 14. Wachira VK, Farinasso CM, Silva RB, Peixoto HM, de Oliveira MRF. Incidence of Guillain-Barré syndrome in the world between 1985 and 2020: A systematic review. Global Epidemiology. 2023 Dec 1;5:100098. DOI: https://doi.org/10.1016/ j.gloepi.2023.100098 PMid:37638372 PMCid:PMC10445966
- Shrivastava M, Nehal S, Seema N. Guillain-Barre syndrome: Demographics, clinical profile & seasonal variation in a tertiary care centre of central India. Indian J Med Res. 2017; 145 (2):203-8. DOI: https://doi.org/10.4103/ijmr.IJMR_995_14 PMid:28639596 PMCid:PMC5501052