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Adenovirus as a plausible trigger of Guillain-Barré syndrome: response to the letter to the editor

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Dear Editor,

We would like to thank the authors for their thoughtful and constructive comments regarding our manuscript.[1,2] We appreciate the opportunity to clarify several important points raised in their letter. We fully agree that establishing a causal relationship between a specific infectious agent and Guillain-Barré syndrome (GBS) is inherently challenging, particularly in the context of case-based observations.

We agree that GBS is most commonly associated with a range of infectious triggers, including *Campylobacter jejuni*, SARS-CoV-2, and other viral pathogens.[3] Adenovirus infections are known to have a broad clinical spectrum in children, ranging from mild disease to severe systemic involvement, particularly in the presence of comorbidities.[4] Due to the word limitations of the case report format, these detailed microbiological or virological evaluations were not fully elaborated in the original manuscript, although they were systematically performed. As outlined below, a comprehensive microbiological evaluation was undertaken in both cases. Blood and cerebrospinal fluid cultures were negative, and a respiratory viral panel including SARS-CoV-2, influenza viruses, respiratory syncytial virus, rhinovirus, enterovirus, metapneumovirus, parainfluenza viruses, and seasonal coronaviruses, detected only adenovirus. Stool cultures were also negative. Neither patient had a history of recent vaccination. Testing for Zika virus was performed in only Case 2 and was negative, while dengue testing was not performed due to the absence of epidemiological risk in our non-endemic setting. Cerebrospinal fluid testing was negative for varicella-zoster virus, herpes simplex virus, and cytomegalovirus, and TORCH panel testing showed no evidence of infection. Respiratory molecular testing was negative for *Mycoplasma pneumoniae* and other atypical bacterial pathogens. While we acknowledge that it is not possible to definitively exclude all potential infectious triggers, the temporal association and the absence of alternative identifiable causes support adenovirus as a plausible antecedent infection rather than a proven causal factor. In this regard, our intention was not to imply causality, but to report a clinically relevant temporal association observed after a reasonably comprehensive exclusion of common alternative triggers.

Cerebrospinal fluid analysis in our patients was consistent with the diagnosis of GBS. In Case 2, classical albuminocytologic dissociation was clearly demonstrated. In Case 1, although interpretation was limited due to a traumatic

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lumbar puncture, elevated protein levels in the absence of pleocytosis were supportive in the appropriate clinical context.[5]

Regarding imaging, although contrast-enhanced spinal magnetic resonance imaging could have provided additional supportive findings in Case 1, the examination was technically limited due to poor cooperation. In this context, the diagnosis was based on the clinical presentation and electrophysiological findings, which remain the cornerstone of GBS diagnosis in routine clinical practice, particularly when imaging is inconclusive or technically limited.

In conclusion, we fully agree that adenovirus should not be considered a definitive cause of GBS without careful exclusion of other triggers. Our aim was to highlight adenovirus as a plausible antecedent infection in the appropriate clinical context. Adenovirus infections are known to mimic immune-mediated conditions such as Kawasaki disease.[6] In addition, adenovirus has been associated with immune-mediated neurological complications such as acute disseminated encephalomyelitis, supporting a potential mechanistic basis for such associations.[7] We believe that our findings contribute to the limited literature on this association and underscore the need for further studies. Importantly, the accumulation of well-characterized case reports and case series remains essential to better define such rare associations and to generate hypotheses for future mechanistic and epidemiological studies.

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Author contribution

The authors confirm contribution to the paper as follows: Study conception and design: GT, KA, IO, GCC; data collection: KA; analysis and interpretation of results: GT, KA, IO, GCC; draft manuscript preparation: GT, KA ABC. All authors reviewed the results and approved the final version of the manuscript.

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Conflict of interest

The authors declare that there is no conflict of interest.

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