Int. j. adv. multidisc. res. stud. 2026; 6(3):504-505

Received: 26-04-2026
Accepted: 06-05-2026

International Journal of Advanced Multidisciplinary Research and Studies

ISSN: 2583-049X

Letter to the Editor

Respiratory Syncytial Virus Infection can only be a Trigger for GBS if all Alternative Causes have been Ruled Out

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Letter to the Editor

We read with interest the article by Liang *et al*. on a self-controlled case series analysis of the risk of developing Guillain-Barré syndrome (GBS) in patients >64 years of age, 1–42 days after infection with respiratory syncytial virus (RSV) [1]. Among 452,471 eligible patients with RSV disease, the incidence rate ratio (IRR) was 2.59, with a marked increase in patients aged ≥75 years [1]. It was concluded that the risk of GBS was increased after RSV infection compared to control periods, with this effect being particularly pronounced in patients aged ≥ 75 years [1]. The study is interesting, but some points should be discussed.

The first point is that a temporal association between RSV infection and the onset of GBS does not necessarily mean that RSV was the trigger. For every patient diagnosed with GBS, it must be clarified whether there were other causes besides RSV infection. The most common triggers of GBS include Campylobacter jejuni, followed by infections with Haemophilus influenza, Mycoplasma pneumoniae, and the Zika virus [2]. Other, less common triggers of GBS include SARS-CoV-2, HIV, hepatitis B virus, hepatitis C virus, hepatitis A virus, hepatitis E virus, Epstein-Barr virus, cytomegalovirus, herpes simplex virus, measles virus, enterovirus, influenza A virus (H1N1), chikungunya virus, parecho virus, Toscana virus, Japanese encephalitis virus, varicella zoster virus, Plasmodium falciparum, Leptospira, Orientia tsutsugamushi, syphilis, and dengue. Non-infectious triggers of GBS include vaccinations (e.g., against SARS-CoV-2, influenza, polio, rabies, hepatitis A and B), lymphomas, kidney transplants, intravenous administration of gangliosides, and trauma [2]. These alternative triggers must be ruled out before RSV can be considered as the cause.

The second point is that the study period also included the pandemic period (2011 to 2024) [1]. Therefore, we should know how many of the included patients tested positive for SARS-CoV-2 at the beginning of their hospital stay. SARS-CoV-2 is a known trigger of GBS.

The third point is that the analysis did not differentiate between the subtypes of GBS [1]. We should know which of the GBS subtypes -acute inflammatory demyelinating polyneuropathy, acute motor axonal neuropathy, Miller Fisher syndrome, cervicopharyngeal-brachial subtype, cranial nerve neuropathy, or Bickerstaff encephalitis - was most commonly triggered by RSV infection.

The fourth point is that it is unclear why only patients over the age of 64 were included in the analysis. GBS can occur in all age groups, including children [3], and RSV infections can also occur at any age, but are most common in children [4]. Of particular interest is the question of whether the prevalence of GBS is higher after RSV infection in children or older patients.

In summary, before GBS can be attributed to RSV infection, all alternative causes must be ruled out as triggers of GBS. In particular, SARS-CoV-2 infections must be ruled out in those diagnosed with GBS during the pandemic.

Declarations

Ethical Approval: Not applicable.

Consent to Participation: Not applicable.

Consent for Publication: Not applicable.

Funding: None received.

Availability of Data and Material: All data are available from the corresponding author.

Completing Interests: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Author Contribution: JF was responsible for the design and conception, discussed available data with coauthors, wrote the first draft, and gave final approval. FS and CS: contributed to literature search, discussion, correction, and final approval.

Keywords: Guillain-Barre Syndrome, Respiratory Syncytial Virus, Elderly Patients, SARS-CoV-2 Infection, Prevalence

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