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Guillain–Barré syndrome (GBS) after severe/critical COVID-19 or COVID-19 vaccination

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Abstract

Background The global COVID-19 pandemic was initiated by the appearance of the novel coronavirus SARS-CoV-2 in 2019, presenting a spectrum of clinical manifestations from asymptomatic cases to severe pneumonia and multi-organ dysfunction, with some cases leading to death induced by hyperinflammatory responses. Neurological manifestations have been reported in more than one-third of COVID-19 patients, particularly in severe instances. While vaccines are pivotal in combating infectious diseases and enhancing public health, reports have linked Guillain–Barré syndrome (GBS) to COVID-19 vaccination and infection. This study seeks to analyze four cases of GBS associated with COVID-19.

Methods Clinical and demographic data were collected from all patients diagnosed with GBS from a biobank, including patients with severe COVID-19 and those with autoimmune conditions resulting from COVID-19 infection or vaccination, who were referred to Alzahra University Hospital in Isfahan, Iran, between October 2020 and December. 2023.

Results Clinical and demographic data of affected patients are presented. This includes a unique family case involving a daughter who passed away due to GBS following AstraZeneca vaccination, her mother who succumbed to post-COVID-19 GBS, and her father who passed away from severe COVID-19 a year earlier.

Conclusions These cases provide valuable insights into investigating potential genetic or epigenetic influences on GBS and hyperinflammation. Furthermore, the occurrence of GBS following exposure to COVID-19 and vaccination suggests shared pathways of autoimmunity induction by SARS-CoV-2 and vaccines.

Keywords GBS, COVID-19 infection, COVID-19 vaccination, Autoimmunity

Introduction

In late 2019, SARS-CoV-2 emerged as a novel coronavirus responsible for a global pandemic [1], associated with a wide range of clinical manifestations, from asymptomatic cases to severe pneumonia, often leading to death due to a hyper-inflammatory response [2]. The severity of disease and clinical outcomes may be influenced by genetic factors [3, 4], such as HLA variants [5].

Common symptoms include cough, fever, shortness of breath, fatigue, and malaise [6]. More than one-third of patients with COVID-19 experience various neurological symptoms affecting both central and peripheral nervous systems (CNS and PNS), particularly in severe

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cases, likely attributed to cerebral hypoxia from respiratory insufficiency [7]. Symptoms related to the CNS include headaches, dizziness, altered levels of consciousness, acute cerebrovascular events, and epilepsy. PNS complications involve decreased or lost sense of smell (hyposmia/anosmia), decreased or lost sense of taste (hypogeusia/ageusia), skeletal muscle pain, and Guillain–Barre syndrome (GBS) [7, 8].

GBS, characterized by acute flaccid paralysis, is a post- or para-infectious neurological disorder that may follow-up per respiratory or gastrointestinal infections [9]. It typically presents with symptoms, such as weakness and tingling in the limbs, which can rapidly progress to paralysis, accompanied by reduced or absent reflexes [10]. GBS cases have been observed following COVID-19 infections and vaccinations, leading to ongoing investigations into whether these associations are causal [11]. Consequently, the primary causes of autoimmune diseases related to either the infection or the vaccine remain inconclusive [12] warranting further investigation into their associations [13, 14].

This study aims to collect the demographic and clinical data from patients diagnosed with GBS following severe COVID-19 infection or vaccination at Alzahra Hospital, Isfahan, Iran. In addition, DNA and RNA samples from the patients were stored for future research.

Materials and methods

Patient collection

We collected clinical and demographic data from all patients diagnosed with GBS from a biobank, including patients with severe COVID-19 and those with severe adverse effects resulting from COVID-19 infection or vaccination, who were referred to Alzahra University Hospital in Isfahan, Iran, between October 2020 and December 2023. The study excluded patients with pre-existing conditions, such as diabetes, chronic kidney disease, chronic liver disease, known primary or secondary immunodeficiency, malnutrition, and individuals who had undergone splenectomy.

All patients provided informed consent to participate in the study. A structured questionnaire was administered to gather the following demographic data: age, gender, vaccination details (including type of vaccine), timing of GBS onset, clinical characteristics, history of chronic diseases, and outcomes.

COVID-19 infection was confirmed based on clinical symptoms, a positive nasopharyngeal PCR test, or a CT scan of the lungs. The diagnosis of GBS was made by a neurologist based on the Brighton criteria [15] for clinical symptoms and the Rajabally criteria [16] for electrodiagnostic findings. EGRIS score was also done to predict the respiratory insufficiency and the need for intubation. The EGRIS score was also used to predict respiratory insufficiency and the need for intubation.

Sample collection and storage

Blood samples (totaling 10 mL) were collected from each participant using EDTA and anticoagulant blood tubes. Samples were processed for DNA, RNA extraction, and serum separation, and stored at − 70 °C for future analyses.

Ethical considerations

All study procedures adhered to the ethical principles and national standards for medical research in Iran (Ethics Code: IR.ARI.MUI.REC.1400.057).

Results

The demographic and clinical traits of individuals diagnosed with GBS are detailed in Table 1.

Patient 1

A 66 year-old male was admitted with symptoms of GBS. Despite having received three doses of the COVID-19 vaccine 8 months prior, he tested positive for COVID-19 via PCR. The patient was initially hospitalized due to weakness in all four limbs. The initial neurological examination revealed a Medical Research Council (MRC) scale score of 2/5 for proximal muscle strength and 3/5 for distal muscle strength in the lower extremities, while upper extremity strength was assessed at 1/5. He demonstrated impaired vibration sense in the lower extremities, and deep tendon reflexes (DTRs) were generally absent. No facial or bulbar weakness was noted.

After receiving intravenous immunoglobulin (IVIg) therapy, the patient showed improvement and was discharged. However, 3 day post-discharge, he developed fever, dry cough, shortness of breath, and pneumonia, necessitating readmission to the Intensive Care Unit (ICU) for severe COVID-19 infection. Over the next 6 months, his weakness gradually improved with physiotherapy.

Patient 2

A 44 year-old male sought medical attention for a 4 day history of tingling and weakness in all limbs. He had been diagnosed with GBS 2 years prior and he also tested positive for COVID-19. He was treated with IVIg for GBS. Although the patient's condition had been stable, it deteriorated over 10 days with recurrent GBS symptoms. His initial neurological examination revealed a MRC scale score of 2/5 in the lower extremities and 3/5 in the upper extremities. He exhibited impaired vibration sense in the lower extremities and disturbance of pinprick sensation in the lower extremities up to the knees. The DTRs in the

Table 1 Demographic data of GBS patients following severe COVID-19 infection or vaccination

Patient Code	Gender	Age	Vaccination	Type of vaccine	Time to GBS onset	Clinical characteristics	Chronic diseases	Final outcome
1	M	66	Yes	Sinopharm	During severe COVID-19	Numbness of hands and feet, quadriparesis, fever and chills, nausea, and vomiting	Renal calculi	Alive
2	M	45	No	No vaccine	Hospitalization due to GBS followed by severe COVID-19	Quadriparesis, drowsiness, fever, shortness of breath, muscle pain	2 years ago, suffering from GBS after COVID-19	Death
3	F	29	Yes	AstraZeneca	5 days after the second dose of the vaccine	Gastrointestinal symptoms, quadriparesis, respiratory distress, dizziness, leukopenia	Hypothyroidism	Death
4	F	61	No	No vaccine	20 days after COVID-19 infection	Paralysis of the lower extremities, upper limb weakness, distal paresthesia,	Osteoporosis	Death

lower extremities were absent, while DTRs in the upper extremities were + 1. COVID-19 sampling was collected upon his arrival at the hospital, and the PCR test was positive. He was treated with Remdesivir and IVIg therapy. Unfortunately, he succumbed to severe autonomic dysfunction.

Patient 3

A 29 year-old woman with a history of drug allergies (notably to azithromycin), gastrointestinal issues, food allergies, and hypothyroidism developed gastrointestinal symptoms, chest pain, nausea, vomiting, dizziness, and quadriparesis 5 days after receiving the second dose of the AstraZeneca vaccine. Upon examination, she exhibited impaired position and vibration sense in her lower extremities and generalized areflexia. Her initial neurological examination revealed a MRC scale score of 3/5 in proximal muscle strength and 2/5 in distal muscle strength. Electromyography demonstrated an acute inflammatory demyelinating polyneuropathy pattern. She tested negative for COVID-19, ruling out an active infection. After receiving IVIg, she showed improvement and was discharged from the hospital. However, 3 weeks after discharge, she was readmitted to the ICU due to respiratory distress. During this time, she also experienced quadriplegia and worsening limb function. Tragically, she passed away due to severe autonomic dysfunction.

Patient 4

A 61 year-old female with a history of osteoporosis presented with progressive lower limb weakness that ascended to upper limb weakness. She experienced sensory disturbances in her fingertips, areflexia, glove and stocking sensory loss, tachycardia, respiratory distress, and dysphagia. Her initial neurological examination revealed a Medical Research Council (MRC) scale score of 0/5 in the foot and 2/5 in the hand. Electromyography demonstrated an acute inflammatory demyelinating polyneuropathy pattern. Considering her clinical presentation and electromyography which was consistent with typical symptoms of GBS, she received treatment with IVIg. Due to respiratory distress, she was intubated in the ICU. During her ICU stay, the patient developed a sinus superinfection and purulent conjunctivitis. The Orbital CT scan was unremarkable. After some initial improvement, she developed urticaria and was treated with corticosteroids. Unfortunately, despite these interventions, she later passed away due to multi-organ dysfunction.

It is notable that the patient reported symptoms of gastritis, fever, and vomiting 20 days prior to her hospitalization. A CT scan performed at that time revealed lung involvement and a COVID-19 infection. In addition, it should be mentioned that Patient 4 was the daughter of Patient 3, who had lost her husband a year earlier due to severe COVID-19.

Discussion

GBS is a post- or para-infectious neurological disorder characterized by symptoms that often follow upper respiratory or gastrointestinal tract infections. In some cases, symptoms may manifest during the infection period itself [10]. Annually, GBS is reported to affect between 0.8 and 1.9 per 100,000 people in the general population [17]. GBS is widely recognized as the most

prevalent cause of acute flaccid paralysis globally [9]. Clinically, it is presented with weakness and tingling in the hands and feet, which rapidly spreads symmetrically, potentially leading to paralysis. GBS is often marked by reduced or absent reflexes and sometimes includes cranial nerve defects [18]. Different subtypes of GBS are classified based on clinical characteristics and electrophysiological results [19].

Various infections have been linked to GBS, including bacteria, such as *Campylobacter jejuni*, *Haemophilus influenzae*, and *Mycoplasma pneumoniae*, as well as viruses, such as cytomegalovirus, herpes simplex virus, Epstein–Barr virus, influenza, and Zika virus [20]. It has also been reported that GBS can occur following vaccination, the use of immune-inhibitor drugs, and certain surgical procedures [21]. Mechanisms such as molecular mimicry—where structural similarities between components of infectious organisms and peripheral nerve elements (notably gangliosides) exist—can lead to the production of autoantibodies and the activation of the complement cascade, contributing to the development of GBS [22], particularly in genetically susceptible individuals [20]. Furthermore, the incidence of GBS increases by 20% for every decade of age. Men are at a greater risk of developing GBS compared to women, which contrasts with trends seen in many other autoimmune diseases [9]. In addition, the presence of immune-related conditions in early life has been linked to a higher likelihood of developing GBS later on [23].

The emergence of the COVID-19 pandemic has coincided with an increase in the reported cases of GBS. This correlation may be related to the significant rise in COVID-19 infections worldwide, rather than indicating that COVID-19 causes more GBS cases than other illnesses. Initial reports of GBS linked to COVID-19 emerged from China, appearing as likely para-infectious disorders [13]. A systematic review conducted by De Sanctis et al. identified 18 cases of GBS occurring alongside COVID-19 symptoms, with cough and fever being the most frequently reported manifestations. In most of these cases, positive PCR tests for COVID-19 or pneumonia suggested that GBS might appear as a para-infectious disorder. However, considering the COVID-19 incubation period of 7–24 days before symptoms appear, the development of GBS after exposure to the virus raises the possibility of post-infectious GBS [24].

Conversely, research by Keddie et al., which analyzed the largest cohort to date, revealed no correlation between COVID-19 and GBS incidence at the regional level. They also noted that there were no unique characteristics of GBS linked to COVID-19 that differentiate it from GBS cases reported during non-pandemic times, indicating that the temporal association between SARS-CoV-2 infection and GBS is not significant [25].

Interestingly, cases of GBS following COVID-19 vaccination have also been observed. A prior study first linked vaccines to GBS during an influenza outbreak among U.S. military personnel [14]. It appears that protein impurities or other components present in vaccines might stimulate the production of antibodies against gangliosides [26], a possibility that could be mitigated by improving purification processes in future vaccine development [27]. Emerging evidence suggests a temporal association between GBS and adenovirus-vector COVID-19 vaccines, such as AstraZeneca. However, considering the multifactorial relationship between GBS and vaccines [28], the natural incidence of GBS, and the limitations of this study, further research with a control group is needed to establish any definitive causal association between GBS and COVID-19 vaccines [29].

In conclusion, while both para-infectious and post-infectious GBS have been associated with COVID-19, they differ in timing and context. Para-infectious GBS occurs during an active infection, while post-infectious GBS can develop after recovery. The risk of GBS following vaccination appears to be lower than that associated with COVID-19 infections, indicating a need for ongoing research to fully understand these relationships [30].

Here, four cases of GBS following severe or critical COVID-19 infection or vaccination were reported:

Case 1 presents a potential relationship between the onset of GBS and the COVID-19 infection that seems to be para-infectious disease, suggesting it may be a para-infectious condition likely related to the COVID-19 incubation period, which can range from 1 week to 24 days [13]. While this case indicates a possible connection between GBS and COVID-19, it is important to note that a definitive causal relationship has not been established [25], and it is also possible that this occurrence may be co-incidental. Although the patient received his booster vaccine 8 months prior, the efficacy of the Sinopharm vaccine decreases after about 6 months [31]. Furthermore, the waning immunity observed in older adults [32] highlights the potential for breakthrough infections even after vaccination. The patient developed severe COVID-19 symptoms 3 days after being discharged from the hospital and showed improvement following rehospitalization, IVIG therapy, and regular physiotherapy exercises.

Case 2 involved a male who experienced recurrent GBS following severe COVID-19 infection. The first episode of GBS also occurred during COVID-19 pandemic and the patient was positive for COVID-19 at that time. Recurrence is defined as occurring if the interval between episodes is ≥ 4 months after

incomplete recovery or ≥ 2 months after complete recovery [33]. The exact reasons and patterns of recurrence in GBS remain unclear; this phenomenon occurs in only 2–5% of GBS patients [33]. Unfortunately, our patient experienced severe illness similar to previously reported cases of recurrent GBS related to COVID-19 [34] and ultimately passed away due to severe autonomic dysfunction. This case may strengthen the possible causal relationship between GBS and COVID-19 infection.

Case 3 exhibited GBS symptoms 5 days after receiving the second dose of the AstraZeneca vaccine, which ultimately led to the patient's death from severe autonomic dysfunction, a significant cause of morbidity in patients with GBS [35]. The cause of GBS may be related to the binding of adenovirus-vector spike proteins to ganglioside receptors and the subsequent production of antibodies against these receptors [36].

Case 4 was the mother of Case 3, who tragically succumbed to GBS triggered by a COVID-19 infection. Her husband had passed away a year earlier due to severe COVID-19. This family presents unique cases that could provide insights into the genetic or epigenetic factors influencing the development of GBS and hyperinflammatory responses [37]. The occurrence of GBS following both COVID-19 infection and vaccination in this family suggests that both the vaccines and the SARS-CoV-2 virus might trigger autoimmunity through similar pathways, involving both autoimmune and autoinflammatory responses [11]. Notably, our research focused on GBS cases in individuals with severe or critical COVID-19, potentially linked to cytokine storms, which could act as a catalyst for the development of autoimmune conditions, such as GBS. This may partly explain the severity of GBS observed, with two out of three post-COVID-19 infection patients losing their lives.

Considering the variability in GBS progression among patients, the limited treatment options and their high costs, as well as the challenges of long-term disability and complaints [38], we propose the following pathway for better management of GBS during the COVID-19 pandemic:

1. Public awareness and education: Raise awareness of GBS symptoms within healthcare settings and the community, particularly related to COVID-19.
2. Screening protocols: Implement screening for neurological signs in patients presenting with respiratory or gastrointestinal symptoms.
3. Early recognition: Train healthcare providers to identify early signs of GBS using checklists or decision-support tools.
4. Diagnosis: Ensure rapid access to diagnostic tests such as nerve conduction studies and lumbar puncture for confirmation of GBS.
5. Admission to ICU: Establish criteria for timely ICU admission for patients exhibiting severe GBS symptoms or respiratory compromise.
6. Treatment indication and selection: Standardize treatment protocols based on severity, providing clear guidelines for IVIG or plasmapheresis initiation.
7. Monitoring disease progression: Regularly monitor neurological and respiratory function using standardized assessment tools.
8. Prediction of clinical course: Use clinical and electrophysiological criteria to predict GBS course and guide individualized management.
9. Management of complications and sequelae: Develop protocols for managing complications, ensuring access to rehabilitation services.
10. Post-infection and vaccination follow-up: Create follow-up plans for recovering patients, focusing on residual symptoms and psychological support.

This pathway can improve the management and outcomes of GBS patients during the COVID-19 pandemic and future pandemic by emphasizing early identification, coordinated care, and comprehensive follow-up.

Conclusion

Previous reports have highlighted a relationship between GBS, viral infections, and vaccination. During the COVID-19 pandemic, the various complications arising from COVID-19 infection and vaccination have revealed a stronger association with GBS. GBS may occur more frequently among patients with severe or critical COVID-19, and similar mechanisms may underpin the induction of GBS through both COVID-19 infection and vaccination. Since this study did not include a control group, further research with more cases is required to elucidate the mechanisms underlying GBS development, compare the incidence of GBS in vaccinated versus non-vaccinated individuals with similar characteristics, and explore the effects of genetic and epigenetic factors on its progression. Techniques such as whole-genome sequencing, targeted gene panels, transcriptomics, and epigenomic profiling (e.g., DNA methylation analysis) could be utilized to identify relevant factors. Furthermore, functional studies may help clarify the mechanisms through which these factors influence the immune response and contribute to GBS development. In light of the neurological adverse events following COVID-19 infection or vaccination, medical professionals should remain vigilant, as prompt diagnosis and comprehensive care are

crucial. Nonetheless, this recognition does not diminish the importance of vaccines in reducing mortality and improving the health of individuals and communities.

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Author contributions
Author contributions S.B: conceptualization, methodology, investigation, supervision, writing—original draft, writing—review & editing; B.A: data curation, formal analysis, validation, visualization, writing—review & editing; L.N-B: conceptualization, resources, project administration, writing—review & editing; B.B: funding acquisition, methodology, software, writing—original draft, writing—review & editing; F.kh: investigation, formal analysis, validation, writing—review & editing; S.A: data curation, visualization, resources, writing—original draft; R.Sh: conceptualization, supervision, writing—review & editing, validation.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
This study was approved by the Research Ethics Committee of Alzahra Research Center at Isfahan University of Medical Sciences (Approval Code: R.ARI.MUI.REC.1400.057).

Consent for publication
All patients provided informed consent to participate in the study and for the publication of the results.

Competing interests
The authors declare no competing interests.

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