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Guillain-Barré Syndrome incidence in the light of COVID-19 infection and vaccines

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Abstract

Introduction and Objective. Guillain-Barré Syndrome is considered a neurological disease that attacks the peripheral nerves in an autoimmune mechanism. Although the exact etiology is uncertain, most cases are preceded by infections or vaccines. The novel coronavirus – SARS-CoV-2 – responsible for COVID-19 pandemic has brought new concerns about GBS cases in the context of morbidity and vaccination. The aim of the review is to present GBS characteristics and research the topics of GBS manifesting after SARS-CoV-2 infection, as well as vaccination, in order to summarize the available data and to assess the link between SARS-CoV-2 and GBS.

Review Methods. This scoping review was conducted with use of PubMed, Cochrane, and Google Scholar databases and 'https://gov.uk' website. The sources ranged mainly from 2019–2024.

Brief description of the state of knowledge. The clinical presentation of GBS depends on a variant, although similar in both COVID-19-related GBS and non-COVID-19-related GBS. The most prevalent GBS variant is acute inflammatory demyelinating polyradiculoneuropathy, with men of older age are more likely to develop the disease. Regarding pathogenesis, a few possible mechanisms have been suggested for SARS-CoV-2 to reach the nervous system, including ACE2 and NRP. For vaccines, similarity between vaccine contents and gangliosides may generate immune cross-reactivity, which induces anti-ganglioside autoantibodies production. The antibodies target neuronal antigens and cause damage to the nervous system.

Summary. GBS cases associated with both COVID-19 vaccines and infection have been observed. Adenovirus-vectored vaccines have been reported as higher-risk in comparison with mRNA-based vaccines; most GBS cases occurred after the first dose of the vaccine. COVID-19-related GBS is probably developed in a post-infection mechanism.

Key words

vaccination, COVID-19, Guillain-Barré Syndrome

INTRODUCTION

Guillain-Barré Syndrome (GBS) is a rapidly progressing immune-mediated polyradiculoneuropathy that may result in paresis of all limbs, and cranial and respiratory muscles [1]. Although the etiology and pathophysiology of GBS remain unclear, it was discovered, however, to be commonly emerging after certain infections caused by, among others, *Campylobacter jejuni (C. jejuni)*, cytomegalovirus (CMV), Zika virus and Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2). A number of subtypes are classified according to pathology, clinical presentations, and neurophysiological features. The incidence of each type varies according to the region and season of the year. The diagnostic process is complicated and requires the exclusion of other diseases with similar manifestations. Treatment involves plasmapheresis, a method for purifying blood plasma, or intravenous immunoglobulin preparations. In cases of respiratory failure, mechanical ventilation is administered. Coronavirus disease 2019 (COVID-19), a disease caused by SARS-CoV-2, began in China in December 2019. Within a few months, the disease spread globally, leading to a pandemic outbreak in 2020. SARS-CoV-2 infection impacts various organs having even the potential to elicit multi-organ failure [2]. While respiratory impairment is the primary symptom of COVID-19, neurological symptoms have also been perceived [3], one of which is the neurological consequences associated with COVID-19 is GBS [1]. During the pandemic, new vaccines against SARS-CoV-2 were developed. They were proven efficient, although multiple cases of GBS were reported to have emerged after SARS-CoV-2 vaccinations.

The aim of the review is to present current data on GBS occurring after COVID-19 infection or vaccination.

MATERIALS AND METHOD

The scoping review includes multiple publications that provided information about GBS and COVID-19. 80 research articles using the PubMed, Cochrane, and Google Scholar databases, excluding languages other than English were analyzed. Key words used to search for data included 'Guillain-Barré Syndrome', linked to additional words 'epidemiology', 'variants', 'subtypes', 'diagnosis', 'treatment', 'vaccination' and 'COVID-19'. After thorough research, 34 articles were selected for inclusion in the review.

Epidemiology. Population-based research in North America and Europe shows that the incidence of GBS varies from 0.81–1.91 cases per 100,000 person-years, with a median

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of 1.11. The risk of developing GBS is higher in males than females, and studies report that the incidence rises by 20% for every 10 years of age [4]. According to a study, the seasonal fluctuation in GBS incidence is inconsistent in some areas, such as the high summer incidence of acute axonal motor neuropathy (AMAN) in Northern China, which differed from other regions in East Asia, where it predominated in winter. There were also greater geographical disparities in seasonal fluctuation in GBS, where Western nations, the Middle East, and East Asia also have a high winter predominance, whereas the Indian subcontinent and Latin America had a high summer prevalence. Seasonal fluctuation in Western countries may be caused by an increased number of upper respiratory tract infections during winter, which has also been suggested in other studies. These differences might be associated with the frequency of prodromal infections throughout the year, but they also might be connected with general factors, such as genetic predisposition, hygiene among the community, or earlier developed immunity [5]. Research by Latov found that *C. jejuni* infection was present in around 30% of GBS patients, with geographical and seasonal variation ranging from 4.8% – 71.7% [6]. Mortality rates in GBS range between 1% – 13% mainly as a result of cardiovascular and respiratory problems; however, for most people, GBS is a treatable condition with a generally good prognosis [7].

Etiology. The actual etiology and pathophysiology of GBS remain unclear. Initially, GBS was considered a single illness with distinguished pathophysiology [8]. Bacterial and viral infections are the most prevalent cause of GBS, occurring in 75% of cases. The International GBS Outcome Study (IGOS) revealed that GBS was caused by *C. jejuni* in 30% of cases, based on 768 accessible biosamples. Moreover, 72% of respondents reported symptoms from past infections, with no significant difference between those who tested positive or negative for a recent illness and 6% of GBS patients had a history of multiple infections (Tab. 1) [9].

Subtypes and diagnosis. Several subtypes of GBS are distinguished, based on pathology, clinical manifestations, and neurophysiological characteristics:

1) *acute inflammatory demyelinating polyradiculoneuropathy (AIDP)* – the most frequently recognized type in Europe and North America. It mainly has demyelinating characteristics, and is associated with better prognosis than axonal types of GBS;
2) *AMAN* – an acute and progressive ascending flaccid quadriparesis, typically aggravated by respiratory failure and accompanied by minimum sensory signs. Characterized by worse recovery prognosis with a high death rate;
3) *acute motor sensory axonal polyneuropathy (AMSAN)* – a similar form to AMAN, but additionally has sensory involvement;
4) *Miller-Fisher syndrome (MFS)* – presents with ophthalmoparesis, areflexia, and ataxia, distinguishing itself from other forms [8]. It is linked to antibodies targeting the GQ1b ganglioside found in oculomotor neurons [10];

Bickerstaff brainstem encephalitis (BBE) – similar to MFS, but in this form symptoms of impaired consciousness additionally appear, as the immune system attacks the pontine reticular formation. The most severely afflicted individuals have respiratory failure and autonomic dysfunction [11].

The different variants are presented in Table 1 [8].

Table 1. Triggers of GBS reported in the literature [9]

Trigger kind	Direct trigger	Frequency
Bacteria	Campylobacter jejuni	+++
	Mycoplasma pneumoniae	++
	Haemophilus influenzae	+
	Escherichia coli	+
Viruses	SARS-CoV-2	+++
	Cytomegaly	+++
	Zika	+++
	Dengue	++
	Influenza-A (H1N1)	+
	Epstein Barr	+
	Hepatitis-E	+
	Measles	+
	Enterovirus D68	+
Vaccinations	SARS-CoV-2	+++
	Influenza	+
	Polio	+
	Rabies	+
	Hepatitis-A, -B	+
Others		

+++ – highly prevalent; ++ – moderate prevalence; + – rare; SARS-CoV-2 – Severe Acute Respiratory Syndrome Coronavirus 2

Diagnostic criteria. Diagnosis of GBS is based on patient's history, neurological and electrophysiological examinations, and cerebrospinal fluid (CSF) parameters. In order to diagnose GBS, other diseases manifesting similar symptoms to GBS must be ruled out first. These include CNS, anterior horn cells, nerve roots, peripheral nerves, neuromuscular junction or muscles disorders. The criteria for diagnosis are both vast and specific, however, the fundamental symptoms are progressive bilateral weakness in arms and legs, and decreased or absent tendon reflexes in affected limbs along the clinical course [12].

Treatment. GBS should be treated as a medical emergency in its early stages of progression. Approximately 25% of patients experience bulbar weakness, which can lead to upper airway blockage and intubation. Respiratory muscle weakness can lead to gradual respiratory failure in around 20% of patients. Autonomic dysfunction, present in around 25% of individuals, may lead to extreme changes in heart rate and blood pressure, leading to circulatory failure. Before considering specialized GBS treatment, all critical functions must be ensured [11].

Corticosteroids have been used for many years, with claims and counterclaims concerning their effectiveness. It was only after they were examined in major national and later worldwide double-blind randomized controlled trials that it became clear that their apparent efficacy is false [13].

Plasma exchange is proven to be effective is now widely acknowledged as a first-line therapy for GBS. Multiple trials found that it reduced the time for patients to walk without

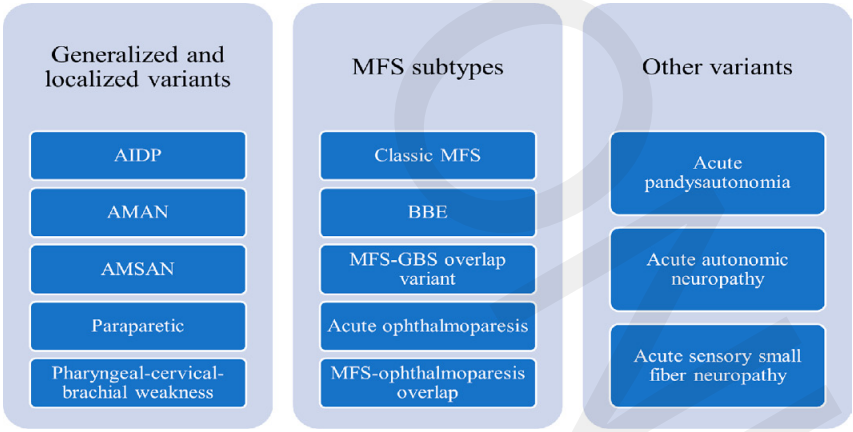

Figure 1. Different variants of GBS (8). AIDP – acute inflammatory demyelinating polyradiculoneuropathy; AMAN – acute axonal motor neuropathy; AMSAN – acute motor sensory axonal polyneuropathy; MFS – Miller-Fisher syndrome; BBE – Bickerstaff brainstem encephalitis; GBS – Guillain-Barré Syndrome.
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assistance, and the time on the ventilator for ventilated patients, when compared to supportive treatment. This finding was also supported by the Cochrane review [14]. It was not verified, however, in a placebo-controlled experiment due to the ethical difficulties of executing such trial in critically sick patients.

Because plasma exchange has become the standard treatment, new options had to be evaluated against it. Some experiments indicated that a single course of intravenous immunoglobulin (IVIg) was at least as effective as plasma exchange, which was also backed by a Cochrane review [13]. As a result, IVIg is widely acknowledged as a first-line therapy. Administering a second IVIg treatment was no more effective, and was linked to more serious side-effects [14].

No other GBS therapies have been found to have any significant effect [15].

COVID-19

Infection. COVID-19, induced by SARS-CoV-2, resulted in a global pandemic in 2020. Aside from severe respiratory symptoms, COVID-19 may cause neurological injury, including myositis, myasthenia gravis, stroke, encephalitis, acute meningitis, and GBS [16]. During the COVID-19 pandemic, the incidence of GBS may have been reduced due to a potential decrease in infectious disease transmission as a result of COVID-19 preventative measures. According to a study of GBS patients in UK hospitals, between March – May 2020, the number of GBS patients decreased compared to the same months in 2016–2019. This could have been due to fewer social interactions and better hand hygiene reducing the spread of other etiologic agents, e.g. respiratory pathogens and ***C. jejuni*** [17].

More than 200 cases of GBS following COVID-19 infection were documented in Europe, America, India, Brazil, China, and the Middle East. In 2021, a meta-analysis found that the prevalence of GBS among COVID-19 patients was 15 per 100,000 population-years [16].

Pathogenesis of COVID-19-related GBS. There are some possible mechanisms for COVID-19 to affect neurological system: 1) direct neurotropism and neuroinvasion of SARS-CoV-2, a secondary effect linked to prothrombotic and vascular effects on the central nervous system (CNS) or peripheral nervous system (PNS), activated by viral infection; 2) secondary effect of systemic inflammatory response on viral infection; 3) autoimmune or post-infectious immune-mediated effect as a response to viral infection [18].

It has been proposed that SARS-CoV-2 enters the CNS via the olfactory bulb using angiotensin-converting enzyme 2 (ACE2). In more recent research, neuropilin (NRP) – the mediator of neuronal guidance and angiogenesis – has been suggested as an entry point for SARS-CoV-2, accelerating the viral invasion of the neurological system. Moreover, NRPs contribute significantly to COVID-19-related hyperinflammatory processes in the lungs, nervous system, kidneys, liver, pancreas, and heart. The effects arise from interactions with certain areas of the SARS-CoV-2 spike protein, along with vascular endothelial growth factor receptors (VEGFR1/2), underscoring the significance of multi-organ neuroimmunopathological damage caused by COVID-19 [19].

GBS is most likely caused by a post-infectious immune system imbalance resulting from COVID-19 [17]. Autoreactive T or B lymphocytes are thought to be stimulated by a molecular mimicry mechanism in which infecting viruses share epitopes similar to some peripheral nerve components [20].

Clinical features of GBS associated with COVID-19. Some researchers compared clinical features in COVID-19 GBS patients and non-COVID-19 GBS patients. The pattern of neurological abnormalities was similar, with the most common GBS variant in both groups being AIDP [21]. The majority of reported patients were males over 50 years of age, reflecting the demographics of COVID-19 cases early in the pandemic. Male gender and older age are therefore risk factors for severe COVID-19, and the prevalence of GBS increases with age [22]. Mohammad Aladawi et al. reported 109 individuals with an average age of 56.07 years, with SARS-CoV-2-associated GBS,. From all cases in the study, the most prevalent variants were a classic sensorimotor GBS and acute demyelinating polyneuropathy. The latency period since viral symptoms until neurological symptoms appeared was 12 days, on average [23]. The average interval between SARS-CoV-2 infection and the onset of GBS symptoms was 2–3 weeks [24].

During the analysis of GBS symptoms in COVID-19, some individuals developed hyperreflexia rather than hyporeflexia, particularly with the AMAN subtype [25]. CSF findings revealed no difference in white blood cell count, although post-COVID-19 GBS patients had a greater protein content in CSF, which has been related to increased permeability of the blood-brain barrier. SARS-CoV-2 ribonucleic acid (RNA) in CSF was absent in all examined patients, while anti-ganglioside antibodies were rarely identified [21]. Bentley et al. also investigated concomitant GBS and COVID-19. In their study, only 18.8% of patients tested positive for anti-ganglioside antibodies, which are frequently related to GBS. This outcome differs from the typical GBS presentation associated with molecular mimicry, indicating that the cytokine release storm may play a significant role at the onset of COVID-19-related GBS [3].

Considering the long interval between SARS-CoV-2 infection and GBS symptoms, absence of CSF pleocytosis, and negative PCR in CSF for SARS-CoV-2, a post-infection mechanism, can be suggested as being more likely than parainfectious [20]. GBS can develop in COVID-19 patients who are completely asymptomatic or have mild symptoms. Moreover, the severity of GBS does not correspond to the severity of COVID-19. Although the course of the disease is rather severe, with approximately one-third of SARS-CoV-2-associated GBS patients requiring mechanical ventilation and intensive care unit (ICU) hospitalization, the immunomodulating treatment has proven to be effective. With IVIg and plasmapheresis treatment, unfavourable results are rare [26].

Vaccination. Vaccines are known to be one of the factors causing GBS. Among the vaccinated population, a higher incidence of GBS has been documented primarily following the administration of the H1N1 swine flu vaccine (1.6 per 1,000,000), as well as following oral adenovirus vaccines and vaccinations against hepatitis B, tetanus, polio, meningitis, and rabies [27, 28]. In the literature, there are reports of GBS incidence following the worldwide COVID-19 vaccinations, although the exact mechanism remains unclear. Some theories have emerged since the COVID-19 pandemic. The initial immunological response elicited by the COVID-19 vaccination may initiate an autoimmune process, wherein antibodies might cross-react via molecular mimicry with glycoproteins on the myelin sheath of peripheral nerve axons, potentially resulting in GBS. Adenovirus-vectored COVID-19 vaccines are recombinant vaccines utilizing a nonreplicating adenovirus vector that encodes the SARS-CoV-2 spike protein to elicit an immunological antibody response. Immune cross-reactivity, induced by the resemblance between vaccine constituents and peripheral nerve components (gangliosides) may lead the immune system to target analogous proteins. Thus, anti-ganglioside autoantibodies target neuronal antigens, resulting in neurological damage in vaccinated people [29].

Research in India found that over 50% of GBS patients tested positive for antiganglioside antibodies, with IgG anti-GT1b being the most frequent observed. Antibodies did not correspond with illness severity, but their absence was associated with dysautonomia and the need for ventilatory support. Antiganglioside antibodies assessment did not correlate with patients' outcome or prognosis [30]. Other factors, such as particular autoantibodies and vaccination adjuvants, may also play a significant role in the autoimmune response [31]. A variety of adverse effects have been described, including fatigue, fever, myalgia, and more severe conditions after receiving vaccinations. Adenovirus-vectored COVID-19 vaccines – ChAdOx1 nCoV-19 (Oxford-AstraZeneca) and Ad.26.COV2.S (Janssen), were proven to elevate the risk of GBS [29].

Abara et al. discovered that GBS reporting after Ad26.COV2.S vaccination was about 9–12-fold more likely than after BNT162b2 (Pfizer-BioNTech) or mRNA-1273 (Moderna) vaccination during 21- and 42-day post-vaccination intervals. In addition, reported GBS cases following Ad26.COV2.S vaccinations were 2–3 times higher than predicted based on background rates at 21- and 42-days post-vaccination, respectively. GBS occurred more frequently after adenovirus immunization than after mRNA (messenger ribonucleic acid) vaccination [32].

Ogunjimi et al. summarized the characteristics of GBS patients following COVID-19 vaccination. Out of 138 cases, the majority were males (59.4%), with the mean age of all patients being 56.8±16.1 years. The average time between vaccination and GBS development was 13.0±6.9 days. Elevated CSF protein levels were found in 87 (96%) of the 91 patients who underwent lumbar puncture, with the mean value of 221 mg/dL. Most patients received the ChAdOx1 nCoV-19 vaccine [33].

As of 24 July 2021, 130 cases of GBS related to immunization with Ad26.COV2.S were reported in the US and the observed-to-expected rate increased in all age categories, except for those aged 18–29 years [31]. Selia and Samia Chowdhury analyzed case reports with a total number of 67 patients from 16 different countries who developed GBS after COVID-19 vaccination. According to their study, some of the initial features were frequently observed, whereas others were reported only occasionally. The most prevalent manifestations were sensory symptoms, limb weakness and cranial nerve palsy.

GBS followed by COVID-19 vaccination was observed in different age groups. Between the ages of 18 and 44, most of the patients were men, with women accounting for around 4 times fewer cases. On the other hand, most cases in men were present in the group aged between 45–65, but in that group women accounted for almost the same amount of cases. [34]. The incidence of GBS following COVID-19 immunization is recorded at 8.1 per 1.000.000 vaccinations, markedly above the rate of GBS in the general population. Virtually, every case report of GBS after COVID-19 immunization occurred after the first dosage [29]. A study by Zhu et al. suggests that patients with a history of radiculitis may be more prone to GBS after COVID-19 immunization. Therefore, extended vaccination intervals and ongoing evaluation of potential increased risk are advisable in such cases [35].

As of 8 December 2021, there have been 472 cases of GBS in the UK following ChAdOx1 nCoV-19 immunization, as well as 27 cases of MFS. Following the BNT162b2 immunization, there have been 69 cases of GBS and 2 of MFS, and 7 cases of GBS after the mRNA-1273 vaccination. During this time, 24.9 million first doses and 24.1 million second doses of the ChAdOx1 nCoV-19 vaccine, 24.8 million first doses and 21.2 million second doses of the BNT162b2 vaccine, and 1.5 million first doses and 1.4 million second doses of the mRNA-1273 vaccine were administered [36].

SUMMARY

GBS is a condition caused by several infections, including COVID-19. GBS in individuals with SARS-CoV-2 infection has clinical and electrophysiological characteristics similar to classical forms, with the only difference being the absence of SARS-CoV-2 RNA in CSF, which means there is no viral attack against nerve roots; however, the full mechanism remains unknown [26].

There is a small increase in the risk of developing GBS after COVID-19 vaccination. The emergence of a post-vaccination neurological condition may arise from the production of host antibodies that cross-react with proteins found in peripheral myelin. These antibodies may be produced in direct reaction to the SARS-CoV-2 spike protein, but a more general immune response, such as to adenovirus vector components, is also possible. However, the discovery of a comparable condition in the context of SARS-CoV-2 infection supports an immune response to the spike protein [35].

There is some evidence for a link between COVID-19 infection and/or vaccination and the occurrence of GBS, however, more research is needed on this topic.

REFERENCES

1. Leonhard SE, Cornblath DR, Endtz HP, et al. Guillain-Barré syndrome in times of pandemics. J Neurol Neurosurg Psychiatry. 2020;91(10):1027–1029. https://doi.org/10.1136/jnnp-2020-324230
2. Malekpour M, Khanmohammadi S, Meybodi MJE, et al. COVID-19 as a trigger of Guillain-Barré syndrome: A review of the molecular mechanism. Immunity, Inflamm Dis. 2023;11(5):e875. https://doi.org/10.1002/iid3.875
3. Bentley SA, Ahmad S, Kobeissy FH, et al. Concomitant Guillain–Barré Syndrome and COVID-19: A Meta-Analysis of Cases. Medicina (Kaunas). 2022;58(12):1835. https://doi.org/10.3390/medicina58121835
4. Shahrizaila N, Lehmann HC, Kuwabara S. Guillain-Barré syndrome. Lancet. 2021;397(10280):1214–1228. https://doi.org/10.1016/s0140-6736(21)00517-1
5. Webb AJS, Brain SAE, Wood R, et al. Seasonal variation in Guillain-Barré syndrome: A systematic review, meta-analysis and Oxfordshire cohort study. J Neurol Neurosurg Psychiatry. 2015;86(11):1196–1201. https://doi.org/10.1136/jnnp-2014-309056
6. Latov N. Campylobacter jejuni Infection, Anti-Ganglioside Antibodies, and Neuropathy. Microorganisms. 2022;10(11):2139. https://doi.org/10.3390/microorganisms10112139
7. Busl KM, Fried H, Muehlschlegel S, et al. Guidelines for Neuroprognostication in Adults with Guillain–Barré Syndrome. Neurocrit Care. 2023;38(3):564–583. https://doi.org/10.1007/s12028-023-01707-3
8. Malek E, Salameh J. Guillain-Barre Syndrome. Semin Neurol. 2019;39(5):589–595. https://doi.org/10.1055/s-0039-1693005
9. Finsterer J. Triggers of Guillain-Barré Syndrome: Campylobacter jejuni Predominates. Int J Mol Sci. 2022;23(22):14222. https://doi.org/10.3390/ijms232214222
10. Laman JD, Huizinga R, Boons G-J, et al. Guillain-Barré syndrome: expanding the concept of molecular mimicry. Trends Immunol. 2022;43(4):296–308. https://doi.org/10.1016/j.it.2022.02.003
11. Leonhard SE, Papri N, Querol L, et al. Guillain-Barré syndrome. Nat Rev Dis Prim. 2024;10(1): 97. https://doi.org/10.1038/s41572-024-00580-4
12. Leonhard SE, Mandarakas MR, Gondim FAA, et al. Diagnosis and management of Guillain-Barré syndrome in ten steps. Nat Rev Neurol. 2019;15(11):671–683. https://doi.org/10.1038/s41582-019-0250-9
13. Hughes RA, Brassington R, Gunn AA, et al. Corticosteroids for Guillain-Barré syndrome. Cochrane Database Syst Rev. 2016;(10):CD001446. https://doi.org/10.1002/14651858.cd001446.pub5
14. Hughes RAC, Swan AV, van Doorn PA. Intravenous immunoglobulin for Guillain-Barré syndrome. Cochrane Database Syst Rev. 2006;(1):CD002063. https://doi.org//10.1002/14651858.CD002063.pub3
15. Pritchard J, Hughes RA, Hadden RD, et al. Pharmacological treatment other than corticosteroids, intravenous immunoglobulin and plasma exchange for Guillain-Barré syndrome. Cochrane Database Syst Rev. 2016;(11):CD008630. https://doi.org/10.1002/14651858.cd008630.pub4
16. Palaiodimou L, Stefanou MI, Katsanos AH, et al. Prevalence, clinical characteristics and outcomes of Guillain-Barré syndrome spectrum associated with COVID-19: A systematic review and meta-analysis. Eur J Neurol. 2021;28(10):3517–3529. https://doi.org/10.1111/ene.14860
17. Davidy T, Zmira O. Peripheral nervous system manifestations associated with COVID-19. Autoimmunity, COVID-19, Post-COVID19 Syndrome and COVID-19 Vaccination. Tel Aviv, 2023.
18. Stafstrom CE, Jantzie LL. COVID-19: Neurological Considerations in Neonates and Children. Children. 2020;7(9):133. https://doi.org/10.3390/children7090133
19. Saleki K, Mojtahedi Z, Ulrichs T, et al. Editorial: Neuroimmune cell signaling in COVID-19. Front Immunol. 2024;15:1429908. https://doi.org/10.3389/fimmu.2024.1429908
20. Zito A, Alfonsi E, Franciotta D, et al. COVID-19 and Guillain-Barré Syndrome: A Case Report and Review of Literature. Front Neurol. 2020;11:909. https://doi.org/10.3389/fneur.2020.00909
21. Şirin NG. Guillain-Barré Syndrome Associated with COVID-19: Is There A Causal Relation or Just A Coincidence? Noropsikiyatri Ars. 2023;60(4):295–297. https://doi.org/10.29399/npa.28547
22. Caress JB, Castoro RJ, Simmons Z, et al. COVID-19-associated Guillain-Barré syndrome: The early pandemic experience. Muscle and Nerve. 2020;62(4):485–491. https://doi.org/10.1002/mus.27024
23. Aladawi M, Elfil M, Abu-Esheh B, et al. Guillain Barre Syndrome as a Complication of COVID-19: A Systematic Review. Can J Neurol Sci. 2022;49(1):38–48. https://doi.org/10.1017/cjn.2021.102
24. Radišić V, Ždraljević M, Perić S, et al. Is there a difference between GBS triggered by COVID-19 and those of other origins? Egypt J Neurol Psychiatry Neurosurg. 2022;58(1):54. https://doi.org/10.1186/s41983-022-00486-6
25. Tawakul AA, Al-Doboke AW, Altayyar SA, et al. Guillain-Barré syndrome in the COVID-19 pandemic. Neurol. Int. 2021;14(1):34–48. https://doi.org/10.3390/neurolint14010003
26. Sansone P, Giaccari LG, Aurilio C, et al. Post-Infectious Guillain-Barré Syndrome Related to SARS-CoV-2 Infection: A Systematic Review. Life (Basel). 2021;11(2):167. https://doi.org/10.3390/life11020167
27. Lunn MP, Cornblath DR, Jacobs BC, et al. COVID-19 vaccine and Guillain-Barré syndrome: let's not leap to associations. Brain. 2021;144(2):357–360. https://doi.org/10.1093/brain/awaa444
28. Introna A, Caputo F, Santoro C, et al. Guillain-Barré syndrome after AstraZeneca COVID-19-vaccination: A causal or casual association? Clin Neurol Neurosurg. 2021;208:106887. https://doi.org/10.1016/j.clineuro.2021.106887
29. Oliveira AM, Varela Ramos P, Durão-Carvalho G, et al. Guillain-Barré Syndrome After a SARS-CoV-2 Vaccine. Cureus. 2024;16(4):e57705. https://doi.org/10.7759/cureus.57705
30. Zhu C, Wang H, Zhang Y, et al. Experience of treatment in critical Guillain-Barre Syndrome case after COVID-19 vaccination. Int J Dev Neurosci [Internet]. 2024;84(4):342–348. https://doi.org/10.1002/jdn.10325
31. Chen Y, Xu Z, Wang P, et al. New-onset autoimmune phenomena post-COVID-19 vaccination. Immunology. 2022;165(4):386–401. https://doi.org/10.1111/imm.13443
32. Abara WE, Gee J, Marquez P, et al. Reports of Guillain-Barré Syndrome After COVID-19 Vaccination in the United States. JAMA Netw Open. 2023;6(5):e2315740. https://doi.org/10.1001/jamanetworkopen.2022.53845
33. Ogunjimi OB, Tsalamandris G, Paladini A, et al. Guillain-Barré Syndrome Induced by Vaccination Against COVID-19: A Systematic Review and Meta-Analysis. Cureus. 2023;15(4):e37578. https://doi.org/10.7759/cureus.37578
34. Chowdhury S, Chowdhury S. Association of Guillain-Barré syndrome following COVID-19 vaccination. Int J Immunopathol Pharmacol. 2023;37:3946320231199349. https://doi.org/10.1177/03946320231199349
35. Allen CM, Ramsamy S, Tarr AW, et al. Guillain-Barré Syndrome Variant Occurring after SARS-CoV-2 Vaccination. Ann Neurol. 2021;90(2):315–318. https://doi.org/10.1002/ana.26144
36. UK Health Security Agency. Information for healthcare professionals on Guillain-Barré Syndrome (GBS) following COVID-19 vaccination. https://www.gov.uk/government/publications/covid-19-vaccination-guillain-barre-syndrome-information-for-healthcare-professionals/information-for-healthcare-professionals-on-guillain-barre-syndrome-gbs-following-covid-19-vaccination (access 2025.01.24).