RECOVERY RATE AND LONG-TERM PROGNOSIS OF GUILLAIN-BARRE SYNDROME

PhD, Associate Professor **Abdukadirova D.T.**, **Abdulhamidov M.A.**

Andijan State Medical Institute, Department of Neurology

Abstract: Guillain—Barre syndrome (pronounced gee-YAN buh-RAY) is a disorder in which the body's immune system attacks the nerves. It can lead to weakness, tingling, or paralysis. Weakness and tingling (paresthesia) in the hands and feet are usually the initial symptoms. These sensations can rapidly spread throughout the body, resulting in paralysis. The most severe forms of Guillain—Barré syndrome are considered medical emergencies. Most patients with this condition require hospitalization. Guillain—Barré syndrome is rare, and its exact cause is unknown. However, in approximately two-thirds of patients, signs of infection are observed within six weeks prior to the onset of Guillain—Barré symptoms. These infections may include respiratory or gastrointestinal tract infections, including COVID-19. The Zika virus can also trigger Guillain—Barré syndrome. Currently, there is no definitive cure for Guillain—Barré syndrome. Nevertheless, several treatment methods can alleviate symptoms and accelerate the recovery process. Most patients recover completely from the disease, although some severe cases may result in death.

Keywords: Guillain–Barre syndrome, immune system, nervous system, weakness, tingling (paresthesia), paralysis, weakness of the hands and feet, inpatient treatment, infection, gastrointestinal infection, COVID-19, Zika virus.

Analysis of clinical, neurological, autonomic, immunological, and electrophysiological parameters in patients with Guillain-Barré syndrome demonstrated the multifactorial nature of the disease and its complex course depending on sex and clinical form. Among 30 patients with Guillain-Barré syndrome (GBS) (16 men and 14 women), the most common complaints were weakness and paresthesias in the limbs, accompanied by gait instability, low back and leg pain, and dizziness. These symptoms were statistically significantly more pronounced in patients with MGBS compared to those with SGBS (p < 0.005; p < 0.001), indicating that the degree of peripheral nerve damage and the activity of immune processes are among the key determinants of clinical severity in GBS. Assessment of neurological status revealed the predominance of pyramidal and lower motor neuron (LMN) syndromes in patients with GBS, recorded overall in 62.5% and 65.0% of cases, respectively. Vestibular and extrapyramidal disorders also accounted for a substantial proportion (40% and 23.3%). All four major neurological syndromes pyramidal, LMN, vestibular, and extrapyramidal—were more frequent and more severe in men (Group I) than in women (Group II): 81%, 64%, 55%, and 27% versus 50%, 60%, 20%, and 10%, respectively (p < 0.05). According to a three-point scale, the severity of CNS syndromes was also higher in men: during the course of GBS, pyramidal, vestibular, and extrapyramidal syndromes scored 1.48, 0.96, and 0.85 points, respectively, compared with 1.34, 0.69, and 0.42 points in women (p < 0.05). These findings provide scientific evidence for sex-related specific features of neurological impairment in GBS.

Analysis of immunological parameters confirmed the leading role of autoimmune processes in GBS. Levels of anti-GM1 and anti-GQ1b antibodies, as well as proinflammatory cytokines such as IL-6 and TNF- α , varied depending on disease activity and clinical form. In patients with



MGBS, anti-GM1 levels were significantly higher than in those with SGBS: in subgroups I-A and II-A they were 61.2 ± 4.1 and 47.4 ± 3.8 U/mL, respectively, whereas in SGBS subgroups I-B and II-B they were 29.6 ± 3.2 and 19.8 ± 2.5 U/mL (p < 0.05). Correlation analysis revealed a significant inverse relationship between anti-GM1 titers and functional status (Yuki–Nachamkin and Hughes scales) (r = -0.57; p = 0.008), a moderate positive correlation with IVIG administration (r = 0.32; p = 0.03), and a very strong positive correlation with early rehabilitation (r = 0.96; p < 0.01). These results confirm a direct and close relationship between peripheral nervous system damage, immune status, early therapeutic strategies, and the course and prognosis of Guillain–Barré syndrome.

ENMG findings demonstrated significant differences in the mechanisms of nerve fiber injury between the demyelinating (AIDP) and axonal (AMAN, AMSAN) subtypes of Guillain-Barré syndrome (GBS). In the AIDP group, a marked prolongation of distal latency (5.2 \pm 1.1 ms), reduction in motor conduction velocity (43 \pm 3.2 m/s), decreased amplitude (4.3 \pm 1.1 mV), and prolongation of the F-wave (39 \pm 4.6 ms) confirmed the predominance of demyelinating processes. In the AMAN and AMSAN forms, a more pronounced reduction in amplitude (2.1 \pm 0.7 and 2.6 ± 0.8 mV, respectively) indicated severe axonal damage. In subgroup analysis, men showed a predominance of reduced motor conduction velocity and prolonged distal latency, whereas women mainly exhibited decreased amplitudes and impaired sensory fiber conduction. These findings indicate the presence of a sex-related spectrum of demyelinating and axonal processes in GBS. The state of the autonomic nervous system was also an integral component of the clinical picture of GBS. Autonomic dystonia syndrome (ADS), hyperventilation syndrome, gastrointestinal dysfunction, dysuric symptoms, psycho-vegetative paroxysms (panic attacks), and localized hyperhidrosis were recorded at very high frequencies in both Groups I and II. In Group I, the prevalence of hyperventilation (41.1%), gastrointestinal dysfunction (38.4%), dysuric symptoms (32.9%), and panic attacks (20.5%) was significantly higher than in Group II (21.1%, 26.3%, 18.4%, and 7.9%, respectively) (p < 0.01-0.001). These vegeto-visceral disorders not only increase the clinical severity of GBS but also significantly reduce patients' physical activity and quality of life.

Analysis of comorbid conditions showed that 61.5% of patients with GBS had one or more concomitant diseases, with an average of 2.36 ± 0.7 comorbidities per patient. The most common comorbid conditions were arterial hypertension (24.3%), metabolic syndrome (28.8%), gastrointestinal diseases (33.3%), cerebrovascular pathology (24.3%), and type 2 diabetes mellitus (15.3%). In women with MGBS, cerebrovascular pathology and metabolic syndrome were identified in 75% and 85% of cases, respectively, indicating that the "definite" clinical form of GBS often coexists with multisystem metabolic and cardiovascular background diseases.

Assessment using the Hughes Functional Grading Scale revealed that, in the early stage of GBS, most patients presented with severe paralysis (HFGS scores 4–5: bedridden and unable to walk independently). Against the background of early comprehensive therapy based on IVIG and plasmapheresis, a stable positive dynamics of Hughes scores was observed at 4 and 12 weeks: the proportion of severe paralysis decreased, while the frequency of transition to mild paresis and normal functional status increased. Although recovery dynamics were slightly faster and residual deficits less frequent in men compared with women, the beneficial effect of early treatment and rehabilitation was clearly evident in both sexes.



Thus, it can be concluded that Guillain–Barré syndrome (GBS) is a complex neuroimmun disorder characterized by the coexistence of various autoimmune and autonomic–visceral processes and sensitivity to sex and clinical form.

The clinical severity of the disease is closely associated with the extent of peripheral and central nervous system involvement, the activity of autoantibodies (anti-GM1) and cytokines, autonomic nervous system imbalance, and the comorbid background. At the same time, the timely use of IVIG and plasmapheresis, combined with early rehabilitation as part of a comprehensive therapeutic approach, reduces the severity of peripheral paralysis and autonomic disturbances in patients with GBS, thereby improving recovery rates and long-term prognosis. Accordingly, the results of this study led to the following key conclusions:

- The severity of clinical manifestations of GBS is closely related to the degree of peripheral nerve damage, immune activity (anti-GM1 antibodies and cytokines), autonomic system imbalance, and sex-related factors.
- In male patients, pyramidal, LMN, vestibular, extrapyramidal, and peripheral paralysis syndromes, as well as autonomic disturbances, occur more frequently and with greater severity compared with female patients.
- In demyelinating (AIDP) forms of GBS, conduction slowing predominates, whereas in axonal (AMAN/AMSAN) forms, a marked reduction in amplitude is dominant; these forms are associated with significant differences in clinical course, recovery rate, and prognosis.
- Comorbid conditions (arterial hypertension, metabolic syndrome, gastrointestinal diseases, diabetes mellitus, etc.) act as background factors that increase the clinical severity of GBS, especially in older patients and in women, where they are often accompanied by multisystem involvement.
- Early comprehensive immunotherapy with IVIG and plasmapheresis, combined with early rehabilitation, accelerates functional recovery, reduces the severity of peripheral paralysis and autonomic disturbances, and decreases the proportion of residual complications.

As a result, the evaluation and management of patients with Guillain–Barré syndrome require an individualized and systematic approach that comprehensively considers clinical, neurological, autonomic, immunological, and electrophysiological parameters, while also accounting for sex differences and comorbid background conditions. The findings of this study provide a scientific basis for improving diagnostic and therapeutic algorithms for GBS in clinical neurology, optimizing early rehabilitation programs, and enhancing long-term functional outcomes for patients.

References:

- 1. Van Doorn P.A., Ruts L., Jacobs B.C., et al. European Academy of Neurology/Peripheral Nerve Society guideline on diagnosis and treatment of Guillain-Barré syndrome. European Journal of Neurology. 2023;30:3646–3674.
- 2. Yusari I.G.A.A.A., Sudira P.G., Samatra D.P.G.P. Clinical characteristics of Guillain-Barré syndrome in COVID-19: a systematic review and meta-analysis. Egyptian Journal of Neurology, Psychiatry and Neurosurgery. 2023;59:40.



- 3. Ali B.M., Hasan M.M., Rahman M.S., et al. Guillain-Barré syndrome: a review of immunotherapy, rehabilitation, and long-term outcomes. International Journal of Community Medicine and Public Health. 2023;10(12):5038–5043.
- 4. Amin W., Arshad S., Khalid A., et al. Clinical outcome of Guillain-Barré syndrome in the intensive care unit of a pediatric tertiary care hospital. Biological and Clinical Sciences Research Journal. 2023;4(1):570.
- 5. Kudratov F.O. Clinical and neurophysiological characteristics of patients with Guillain-Barré syndrome. Innovative Development in Research and Education. 2023;2(22):257–260.
- 6. Ismoilov B.A., Karimov J.K., et al. In the early stages of Guillain-Barré syndrome, gangliosides (GM1 autoantibodies and GM1) in the blood serum. Innovative Models in Education Systems. 2023;3(34).
- 7. Zhang Y., Liu X., Chen J. Guillain-Barré syndrome with compressive myelopathy: a case report. Intelligent Pharmacy. 2023;1(4):283–285.
- 8. Rodrigues L.D., Silva A.C., Pereira R.M., et al. Pediatric Guillain-Barré syndrome: a case series. Research, Society and Development. 2023;12(13):e99121344236.
- 9. Finsterer J., Scorza F.A. Immunological, clinical, and epidemiological features of Guillain-Barré syndrome associated with SARS-CoV-2. Journal of Medical Virology. 2023;95:e5380946.
- 10. Ayraler A. COVID-19 infection and Guillain-Barré syndrome: a case report. Ordu University Medical Journal. 2023;10(3):143–147.
- 11. Wang L., Chen Y., Li S., et al. Knowledge mapping and bibliometric analysis of Guillain-Barré syndrome research from 2013 to 2023. Frontiers in Neurology. 2023.
- 12. Hughes R.A.C., Cornblath D.R., et al. Predictors of mechanical ventilation in axonal subtypes of Guillain-Barré syndrome. Canadian Journal of Neurological Sciences. 2023;50:221–227.
- 13. Khan F., Amatya B., Galea M.P. Plasma exchange versus intravenous immunoglobulin for severe Guillain-Barré syndrome: a systematic review. eNeurologicalSci. 2023;31:100468.
- 14. Martin T., Lefèvre G., Arnaud L. Guillain-Barré syndrome revealing systemic lupus erythematosus: a case report. Rheumatology. 2023;62(Suppl 3).
- 15. Ahmed R., Malik A., Iqbal M., et al. Clinical, demographic and neurophysiological features of Guillain-Barré syndrome. The Professional Medical Journal. 2023;30(6):742–748.

