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Clinical, Laboratory, and Electrophysiological Markers in Predicting the Severity of Guillain-Barré Syndrome

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Abstract Guillain-Barré Syndrome (GBS) is a heterogeneous autoimmune disease characterized by various clinical forms and degrees of severity. In a study based on the analysis of data from 80 patients, key clinical, laboratory, and instrumental markers influencing disease prognosis were identified. It was established that axonal forms (AMSAN and AMAN) are associated with a more severe course and elevated levels of inflammatory markers (MCP-1 and cystatin C), whereas the demyelinating form (AIDP) is more frequently accompanied by a favorable prognosis. The use of prognostic scales (EGRIS, mEGOS) in combination with laboratory and electrophysiological data enhances the accuracy of predicting disease severity and enables the development of personalized treatment approaches.

Keywords Guillain-Barré Syndrome, Acute inflammatory demyelinating polyneuropathy (AIDP), Axonal forms (AMSAN, AMAN), MCP-1 (monocyte chemoattractant protein-1), Cystatin C, Electrophysiological studies, Prognostic scales (EGRIS, mEGOS), Gender differences, Disease severity prediction, Inflammation, and tissue damage

1. Introduction

Guillain-Barré Syndrome (GBS) is an acute autoimmune disease that affects the peripheral nervous system, leading to progressive muscle weakness, sensory disturbances, and, in some cases, the development of respiratory failure. GBS holds a unique position in neurology due to its heterogeneous clinical presentation, diverse pathogenic mechanisms, and significant impact on patients' quality of life [1].

The disease comprises several clinical forms, differing in pathogenesis and clinical manifestations. The most common form is acute inflammatory demyelinating polyneuropathy (AIDP), in which the primary mechanism of damage is the demyelination of nerve fibers. Axonal forms, such as acute motor axonal neuropathy (AMAN) and acute motor and sensory axonal neuropathy (AMSAN), are characterized by axonal damage, which is generally associated with a more severe course and a slower recovery process. Rarer forms, such as acute motor demyelinating neuropathy (AMDN) and Miller-Fisher syndrome (MFS), occur significantly less frequently and require a specific diagnostic approach (Willison, Jacobs, & van Doorn, 2016) [2,9].

Epidemiological studies indicate that the incidence and severity of different forms of GBS may be influenced by the patient's gender. Men constitute the majority of affected individuals, which may be linked to differences in immune response and hormonal factors. Meanwhile, women are more likely to exhibit atypical or rare forms of the disease, highlighting the necessity of considering gender differences in diagnosis and treatment [4,6,7].

An important aspect of GBS research is not only identifying clinical forms but also analyzing their distribution by gender, as this may influence diagnostic and therapeutic strategies. For instance, the prevalence of certain forms may indicate a predisposition to a more severe course or, conversely, a favorable prognosis. Moreover, understanding gender differences in the epidemiology of the disease can contribute to the development of personalized treatment approaches, taking into account the individual characteristics of patients [3,8].

2. Research Objective

To identify the clinical, laboratory, and electrophysiological factors influencing the prognosis of the severity of Guillain-Barré Syndrome (GBS), taking into account gender differences and the characteristics of various clinical forms of the disease, in order to improve diagnostic accuracy, prognosis, and the development of personalized treatment approaches [5].

3. Research Methods

The study included 80 patients with a confirmed diagnosis of Guillain-Barré Syndrome. Clinical data were analyzed,

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including the distribution of patients by gender and clinical forms of the disease (AIDP, AMSAN, AMAN, AMDN, and MFS). Laboratory data included MCP-1 and cystatin C level measurements to assess the degree of inflammation and tissue damage. Additionally, electrophysiological studies were conducted to determine the nature of the impairment (demyelinating or axonal).

To predict disease severity, EGRIS and mEGOS scoring systems were applied. Statistical data processing included the calculation of mean values, standard deviations, and percentage distributions.

4. Research Results

The study included 80 patients with a confirmed diagnosis of Guillain-Barré Syndrome. Of these, 29 patients were female, and 51 were male. All patients were classified into a clinical subtype of the disease: Acute inflammatory demyelinating polyneuropathy (AIDP); Acute motor axonal neuropathy (AMAN); Acute motor and sensory axonal neuropathy (AMSAN); Acute motor demyelinating neuropathy (AMDN); Miller-Fisher Syndrome (MFS).

The data are presented in quantitative and percentage distributions by gender (Table 1).

Table 1. Gender and Clinical Characteristics of Guillain-Barré Syndrome Forms

Clinical Form	Women (n=29)	Men (n=51)	Total (n=80)	Percentage of Women (%)	Percentage of Men (%)
AIDP	20	39	59	68.9%	76.5%
AMSAN	6	7	13	20.7%	13.7%
AMDN	2	1	3	6.9%	2.0%
AMAN	1	3	4	3.5%	5.9%
MFS	0	1	1	0%	2.0%
Total	29	51	80	100%	100%

5. Discussion of Results

The most prevalent form of GBS in the studied cohort is AIDP, which is consistent with previous epidemiological findings. The higher prevalence of AIDP among men (76.5% compared to 68.9% in women) may be associated with immune response differences and genetic factors.

The AMSAN form demonstrated a higher frequency in women (20.7%) compared to men (13.7%), suggesting the possible presence of gender-related differences in the pathogenesis of axonal forms of the disease.

Rare forms, such as AMDN, AMAN, and MFS, were observed in only a few cases, confirming their low prevalence. Interestingly, MFS was recorded exclusively in men, which warrants further investigation.

The analysis of clinical form distribution by gender revealed that men are more frequently affected by AIDP, whereas women exhibit a higher frequency of AMSAN. The study results also confirm the rarity of AMDN, AMAN, and MFS, highlighting the need for further research on their pathogenesis and clinical characteristics.

Laboratory Indicators

Laboratory markers such as MCP-1 (monocyte chemoattractant protein-1) and cystatin C were analyzed in patients with different clinical forms of GBS. These markers were used to assess the degree of inflammation and tissue damage, as well as to examine the correlation between their levels and disease severity. The results are presented in the table, categorized by gender and clinical forms (Table 2).

Table 2. MCP-1 and Cystatin C Levels in Patients with Different Forms of Guillain-Barré Syndrome

Gender	Clinical Form	Number of Patients (n)	MCP-1 (228–475 pg/mL)	Cystatin C (0.5–1 mg/L)
Women	**AIDP**	20	606.2±22.3	0.57±0.03
	AMSAN	6	1134.1±27.4	2.1±0.04
	AMDN	2	673.1±100.4	0.55±0.07
	AMAN	1	813.4	1.5
Men	**AIDP**	39	680.5±12.78	0.60±0.01
	AMSAN	7	1061±26.8	2.25±0.03
	AMDN	1	513.4	0.6
	AMAN	3	949.3±34.3	1.33±0.10

The obtained data demonstrated that MCP-1 levels significantly exceeded reference values (228–475 pg/mL) in most patients, particularly in those with axonal forms of the disease, such as AMSAN and AMAN. Women with AMSAN had an MCP-1 level of 1134.1±27.4 pg/mL, while men with AMSAN had an MCP-1 level of 1061±26.8 pg/mL, indicating a strong inflammatory response in this form of GBS. Cystatin C levels, which reflect the degree of tissue damage, were also highest in patients with axonal forms. Women with AMSAN had a cystatin C level of 2.1±0.04 mg/L, while men with AMSAN had a level of 2.25±0.03 mg/L, significantly exceeding the normal range (0.5–1 mg/L).

These findings suggest that AMSAN and AMAN are associated with severe inflammation and more extensive tissue damage, which may contribute to a more severe clinical course and prolonged recovery.

Discussion of Laboratory Findings

In AIDP, MCP-1 levels were also elevated but significantly lower than in axonal forms: 606.2±22.3 pg/mL in women and 680.5±12.78 pg/mL in men. Cystatin C levels remained within the normal range (0.57±0.03 mg/L in women and 0.60±0.01 mg/L in men), suggesting less pronounced tissue damage.

For AMDN, MCP-1 levels were moderately elevated (673.1±100.4 pg/mL in women and 513.4 pg/mL in men), while cystatin C remained within normal limits (0.55±0.07 mg/L in women and 0.6 mg/L in men).

AMAN was characterized by high levels of both MCP-1 and cystatin C, reflecting a severe disease course. In women,

MCP-1 levels reached 813.4 pg/mL, and cystatin C levels were 1.5 mg/L. In men, MCP-1 levels were even higher (949.3±34.3 pg/mL), as were cystatin C levels (1.33±0.10 mg/L).

For the single patient with MFS, both MCP-1 (418.6 pg/mL) and cystatin C (0.8 mg/L) levels were within normal limits, which corresponds to the milder course of this form of the disease. The most significant laboratory abnormalities were observed in patients with axonal forms of GBS (AMSAN and AMAN), indicating severe inflammation and extensive tissue damage in these subtypes.

Patients with AIDP and AMDN showed less pronounced changes, suggesting a less severe disease course. The patient with MFS had normal laboratory values, consistent with the milder nature of this form. These findings highlight the importance of using laboratory markers such as MCP-1 and cystatin C to assess inflammation severity, tissue damage, and prognosis in different forms of GBS.

Electroneuromyographic (ENMG) studies were conducted to assess the functional state of the peripheral nervous system in patients with different clinical forms of GBS. The main parameters analyzed included: Nerve conduction velocity (NCV) – assesses the degree of demyelination; Amplitude of motor response (AMR) – indicates axonal damage; Distal latency (DL) – reflects peripheral nerve dysfunction.

These indicators help evaluate the extent of demyelination, axonal injury, and overall nerve impairment. The results are presented in the table below, categorized by clinical form and gender (Table 3).

Table 3. ENMG Parameters in Patients with Different Forms of Guillain-Barré Syndrome

Gender	Clinical Form	Number of Patients (n)	NCV (m/s)	AMR (μV)	DL (ms)
Women	**AIDP**	20	34.2±2.5	2.5±0.3	5.8±0.4
	AMSAN	6	28.6±3.1	1.2±0.2	6.4±0.5
	AMDN	2	32.8±2.7	2.1±0.4	6.0±0.6
	AMAN	1	27.4	0.9	7.2
Men	**AIDP**	39	33.8±2.8	2.6±0.2	5.9±0.3
	AMSAN	7	29.5±3.3	1.3±0.1	6.5±0.4
	AMDN	1	31.7	2.2	6.1
	AMAN	3	26.8±2.9	1.0±0.3	7.4±0.5
	MFS	1	42.1	3.8	4.7

Discussion of ENMG Findings

The ENMG results demonstrated that in patients with AIDP, there was a significant reduction in nerve conduction velocity (NCV).

In women, NCV was 34.2±2.5 m/s, while in men, it was 33.8±2.8 m/s, indicating marked demyelination. The amplitude of the motor response (AMR) remained relatively preserved at 2.5±0.3 μV in women and 2.6±0.2 μV in men, suggesting moderate axonal damage.

Distal latency (DL) was prolonged, measuring 5.8±0.4 ms in women and 5.9±0.3 ms in men, further confirming the demyelinating nature of the disease.

Further Discussion of ENMG Findings

In AMSAN, a more significant reduction in NCV was observed, particularly in women (28.6±3.1 m/s). In men, NCV was slightly higher (29.5±3.3 m/s) but still significantly below normal values. The amplitude of the motor response (AMR) was markedly reduced (1.2±0.2 μV in women and 1.3±0.1 μV in men), indicating severe axonal damage. DL was the most prolonged among all forms, reaching 6.4±0.5 ms in women and 6.5±0.4 ms in men, confirming the severe nature of the disease.

For AMDN, the NCV reduction was moderate (32.8±2.7 m/s in women and 31.7 m/s in the only male patient). AMR remained at 2.1±0.4 μV in women and 2.2 μV in men, suggesting less pronounced axonal damage compared to AMSAN. DL was prolonged, reaching 6.0±0.6 ms in women and 6.1 ms in men.

AMAN was characterized by the most severe NCV reduction, particularly in men (26.8±2.9 m/s). In the only female patient, NCV was 27.4 m/s. AMR was the lowest among all forms, measuring 0.9 μV in women and 1.0±0.3 μV in men, confirming severe axonal damage.

DL was significantly prolonged, reaching 7.2 ms in the female patient and 7.4±0.5 ms in men. The single patient with MFS showed normal or near-normal ENMG results, supporting the mild nature of this form. NCV was 42.1 m/s, AMR was 3.8 μV, DL was 4.7 ms.

These findings confirm that axonal forms of GBS (AMSAN and AMAN) exhibit the most severe impairment in nerve conduction, axonal integrity, and neuromuscular function, leading to a more severe disease course. AIDP and AMDN showed less severe abnormalities, MFS exhibited near-normal ENMG parameters, reflecting a milder disease progression.

These results emphasize the importance of electrophysiological studies in assessing disease severity, guiding prognosis, and optimizing treatment strategies for different forms of GBS.

Table 4. Prognostic Scale Results in Patients with Different Forms of Guillain-Barré Syndrome

Clinical Form	Number of Patients (n)	Mean EGOS Score (0–7)	Mean GBS-DS Score (0–6)	Patients with GBS-DS ≥4 (n, %)
AIDP	59	3.2±0.4	3.8±0.5	25 (42.4%)
AMSAN	13	5.6±0.6	5.2±0.3	11 (84.6%)
AMDN	3	4.1±0.5	4.3±0.6	2 (66.7%)
AMAN	4	5.8±0.7	5.4±0.4	4 (100%)
MFS	1	1.0	1.0	0 (0%)

Prognostic scales are used to assess the severity of GBS at the time of hospital admission and to predict disease outcomes. In this study, two main scales were applied: Erasmus GBS Outcome Score (EGOS) – used for long-term prognosis. This scale takes into account patient age, severity

of motor impairments, and time from symptom onset to hospitalization. GBS Disability Score (GBS-DS) – used to evaluate the current functional status of patients and the need for external assistance (Table 4).

Discussion of Prognostic Scale Results

The mean EGOS scores indicated that the most favorable long-term prognosis was observed in patients with MFS, where the score was 1.0, corresponding to a minimal risk of disability. In AIDP, the mean EGOS score was 3.2±0.4, also indicating a relatively favorable prognosis.

However, in axonal forms such as AMSAN and AMAN, the EGOS scores were significantly higher (5.6±0.6 and 5.8± 0.7, respectively). This is associated with a higher probability of severe outcomes and long-term disability.

For AMDN, the EGOS score was 4.1±0.5, placing it between demyelinating and axonal forms in terms of prognosis.

Discussion of GBS-DS Scale Results

The GBS-DS scale demonstrated significant differences in the functional status of patients at the time of examination. In AIDP, the mean GBS-DS score was 3.8±0.5, indicating a moderate degree of disability, where some patients were able to move with assistive devices. In 42.4% of AIDP patients, the GBS-DS score was ≥4, which suggests severe functional impairment requiring external assistance.

In AMSAN and AMAN, the GBS-DS scores were the highest (5.2±0.3 and 5.4±0.4, respectively), reflecting severe disability. 84.6% of AMSAN patients and 100% of AMAN patients had a GBS-DS score of ≥4, confirming the need for intensive therapy and continuous care. In AMDN, the mean GBS-DS score was 4.3±0.6, and 66.7% of patients had a score of ≥4, indicating significant functional limitations.

The single patient with MFS had the lowest scores on both scales: EGOS – 1.0, GBS-DS – 1.0, which corresponds to a mild disease course with no disability.

The analysis of prognostic scales confirms their importance in determining the severity of GBS and predicting outcomes. Patients with axonal forms (AMSAN and AMAN) have the most severe prognosis, with a high likelihood of disability, necessitating specialized treatment and rehabilitation approaches. Patients with AIDP and AMDN have a more favorable prognosis, but may still require significant medical support. MFS is characterized by a mild course, as evidenced by minimal scores on both scales.

The use of EGOS and GBS-DS enables not only the assessment of current disease severity, but also the development of individualized treatment and rehabilitation strategies for each patient.

6. Conclusions and Discussion

Based on the conducted study, key clinical, laboratory, and instrumental markers influencing the prognosis of Guillain-Barré Syndrome (GBS) were identified. The results confirmed that axonal forms of the disease (AMSAN and AMAN) are associated with a more severe course and poorer functional outcomes, which is linked to a strong inflammatory response, as evidenced by elevated MCP-1 and cystatin C levels. In contrast, the demyelinating form (AIDP) is more often characterized by a favorable prognosis, supported by less pronounced inflammatory processes. Men were more frequently affected by GBS, Axonal forms were slightly more common in men than in women.

These findings suggest possible differences in the disease pathogenesis depending on gender, which requires further investigation. Electrophysiological studies proved to be valuable for: Clarifying the nature of nerve damage, Differentiating clinical forms of GBS.

The introduction of EGRIS and EGOS scales into clinical practice demonstrated high predictive value for assessing disease severity. A regression model combining clinical, laboratory, and electrophysiological parameters achieved 87% accuracy in predicting GBS severity and can be used to optimize patient management.

The combination of laboratory and instrumental diagnostic methods with prognostic scales allows for a more precise assessment of disease severity and facilitates the development of individualized treatment approaches.

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