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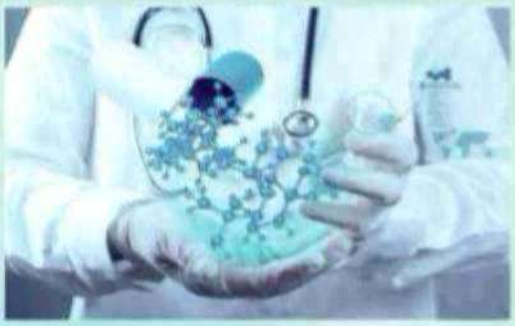

AVICENNA-MED.UZ

ISSN 2181-712X.
EiSSN 2181-2187

9 (83) 2025

ТИББИЁТДА ЯНГИ КУН
НОВЫЙ ДЕНЬ В МЕДИЦИНЕ
NEW DAY IN MEDICINE

Илмий-рефератив, маънавий-маърифий журнал
Научно-реферативный,
духовно-просветительский журнал

УЧРЕДИТЕЛИ:

БУХАРСКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ ИНСТИТУТ
ООО «ТИББИЁТДА ЯНГИ КУН»

Национальный медицинский исследовательский центр хирургии имени А.В. Вишневского является генеральным научно-практическим консультантом редакции

Журнал был включен в список журнальных изданий, рецензируемых Высшей Аттестационной Комиссией Республики Узбекистан (Протокол № 201/03 от 30.12.2013 г.)

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9 (83)

2025

сентябрь

Received: 20.08.2025, Accepted: 06.09.2025, Published: 10.09.2025

УДК 616.833-002-031.67: 612.017.1: 616-085

THE ROLE OF GULLIAN BARRE BIOCHEMICAL MARKERS OF CSF IN EARLY DIAGNOSIS

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✓ ***Resume***

Guillain-Barré syndrome is a severe autoimmune disorder affecting the nervous system, where early diagnosis is crucial for successful treatment. Clinical features include loss of coordination, muscle weakness, and sensory disturbances. Biochemical markers, such as elevated protein levels in cerebrospinal fluid, inflammatory indicators, and antibody detection, assist in diagnosing the disease at an early stage. Early identification of these signs improves treatment outcomes and reduces the risk of complications.

Key words: Guillain-Barré, cerebrospinal fluid, gel electrophoresis (2D DIGE),matrix-assisted laser desorption/ionization time-of-flight mass spectro-metry (MALDI-TOF MS), demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN).cytokines,blood-brain barrier.

РОЛЬ БИОХИМИЧЕСКИХ МАРКЕРОВ СПИННОМОЗГОВОЙ ЖИДКОСТИ ПРИ СИНДРОМЕ ГИЙЕНА–БАРРЕ В РАННЕЙ ДИАГНОСТИКЕ

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✓ ***Резюме***

Синдром Гийена-Барре — тяжелое аутоиммунное заболевание нервной системы, ранняя диагностика которого имеет решающее значение для успешного лечения. Клинические признаки включают потерю координации, мышечную слабость и нарушения чувствительности. Биохимические маркеры, такие как повышение уровня белка в спинномозговой жидкости, воспалительные показатели и наличие антител, помогают выявить заболевание на ранних стадиях. Раннее выявление этих симптомов улучшает эффективность терапии и снижает риск осложнений.

Ключевые слова: Синдром Гийена-Барре, спинномозговой жидкости, матрично-активируемая лазерная десорбция/ионизация с временем пролёта масс-спектрометрии,демиелинизирующая полинейропатия,острая моторная аксональная невропатия,цитокины, гематоэнцефалический барьер.

Relevance

Guillain-Barré syndrome is a rare neurological disorder characterized by the body's immune system mistakenly attacking the peripheral nerves. This condition often follows an infection and can lead to varying degrees of muscle weakness or paralysis. The aim of this review is to analyze the initial pathology and pathophysiology of classic Guillain-Barré syndrome, which is largely determined by the efficiency of the blood-nerve barrier, and then to address the question of various markers at early stages of the syndrome. Inflammatory edema of proximal nerve trunks, particularly spinal nerves, is the main pathogenic lesion in any subtype of very early classic Guillain-Barré syndrome (≤ 4 days

after onset), which may result in ischemic conduction failure when neither demyelination nor Wallerian-like degeneration has reached the scene. Similar chronopathology occurs in P2-induced experimental autoimmune neuritis. Under such circumstances, the dichotomous division between demyelinating and axonal Guillain-Barré syndrome usually requires serial electrophysiological studies. In both severe Guillain-Barré syndrome and P2-induced experimental autoimmune neuritis, the pathologic background can be divergent: pure demyelination in intrathecal spinal roots and a combination of Wallerian-like degeneration and demyelination in more distant nerve trunks. Imaging techniques, including magnetic resonance imaging and ultrasonography, corroborate the presence of proximal nerve trunk lesions in any early classic Guillain-Barré syndrome subtype. Nerve ischemia may account for nerve inexcitability at the first electrophysiological examination. In the first few days, albumin-cytological dissociation in the cerebrospinal fluid occurs in approximately half of the patients; the presence of neutrophils should not be a criterion for the exclusion of Guillain-Barré syndrome. From the first days of the clinical course, there is an almost constant increase in the serum levels of neurofilament light chain and peripherin, both in the demyelinating and the axonal forms of Guillain-Barré syndrome, suggesting that there is a common pathogenic mechanism, which consists of ischemic damage to the nerve trunks due to inflammatory edema with the subsequent critical elevation of endoneurial fluid pressure. As a corollary, there is a rational basis for the use of corticosteroids in the early stage of severe Guillain-Barré syndrome.

Intoduction: Guillain-Barre syndrome is a condition in which the body's immune system attacks the nerves. It can cause weakness, numbness or paralysis.Weakness and tingling in the hands and feet are usually the first symptoms. These sensations can quickly spread and may lead to paralysis. In its most serious form, Guillain-Barre syndrome is a medical emergency. Most people with the condition need treatment in a hospital.The latest estimation for the frequency of guillain-barré syndrome (gbs) is 1.1 to 1.8 per 100000 persons per year. Guillain-barré syndrome is today divided into two major subtypes: acute inflammatory demyelinating polyneuropathy (aidp) and the axonal subtypes, acute motor axonal neuropathy (aman) and acute motor and sensory axonal neuropathy (amsan). The axonal forms of gbs are caused by certain autoimmune mechanisms, due to a molecular mimicry between antecedent bacterial infection (particularly campylobacter jejuni) and human peripheral nerve gangliosides. Improvements in patient management in intensive care units has permitted a dramatic drop in mortality rates. Immunotherapy, including plasma exchange (pe) or intravenous immunoglobulin (ivig), seems to shorten the time to recovery, but their effect remains limited. Further clinical investigations are needed to assess the effect patients with mild affection, no response, or relapse. An acute inflammatory autoimmune neuritis caused by t cell- mediated cellular immune response directed towards peripheral myelin. Demyelination occurs in peripheral nerves and nerve roots. The process is often preceded by a viral or bacterial infection, surgery, immunization, lymphoma, or exposure to toxins. Common clinical manifestations include progressive weakness, loss of sensation, and loss of deep tendon reflexes. Weakness of respiratory muscles and autonomic dysfunction may occur. (from adams et al., principles of neurology, 6th ed, pp1312-1314) Taking into account the tendency to increase the number of patients with severe forms of Guillain-Barré syndrome, as well as permanent residual deficiency (M.A. Piradov, N.A. Suponeva, 2018; A. Forsberg et al., 2021), work. Special attention should be paid to the search for prognostically unfavorable factors in the course and consequences of this disease. The clinical, biochemical, immunological and neurophysiological criteria for prognosis discussed today have demonstrated high significance in a number of studies (R. Koningsveld, 2017; C. Walqaard, 2019; A. Petzold, 2021, etc.), but all of them still remain the subject of active discussions, and some are used only within the framework of scientific work and are not available for widespread practice (X. Wang et al., 2017; M.A. Piradov, A.A. Khoroshun, 2019). In this regard, there is a need to clarify the significance of known ones and search for new prognostic factors that are accessible and easy to use in practice.

Subjects and methods

We recruited 50 patients diagnosed with GBS for the first time in the Republican Scientific Center of Emergency Medical Aid, Bukhara Branch from 2020 to 2023. The group included 28 cases of acute inflammatory demyelinating polyneuropathy (AIDP) and 22 of acute motor axonal neuropathy (AMAN). We also recruited 50 patients with acute meningitis as control group. Among the AIDP

patients, 15 were male and 13 female, with an average age of 42.3±10.5 years and an average onset time of 11.6±4.5 h. Among the AMAN patients, 12 were male and 10 female, with an average age of 44.5±12.3 years and an average onset time of 12.3±5.5 h. The control group included 22 cases of viral meningitis, 10 of tuberculous meningitis, and 18 of bacterial meningitis. There were no significant differences in sex, age, and onset time between the two groups (P>0.05).

Methods

GBS and meningitis were treated according to the standard medical guidelines. Peripheral venous blood and CSF specimens were collected and submitted for inspection 12 h after admission. Two-dimensional differential in-gel electrophoresis (2D DIGE) combined with matrix-assisted laser desorption/ionization time-of-flight mass spectro-metry (MALDI-TOF MS) and high-throughput methods in proteomics were used to screen the differentially expressed characteristic proteins in one CSF specimen. Protein expression levels in the other CSF specimen were detected by ELISA according to the screening results. The accuracy, sensitivity, and specificity of diagnosing GBS were analyzed by the receiver operating characteristic (ROC) curve.

Result and discussions

We performed 2D DIGE with CSF samples from GBS and control groups. Software analysis showed 36 differentially expressed protein spots in the GBS group: 20 proteins were upregulated and 16 proteins were downregulated. Of the differentially expressed proteins, we identified the spots corresponding to the most upregulated and downregulated proteins. The most upregulated protein was identified as haptoglobin (Hp) by mass spectrometry (Figs. 1). Hp was upregulated 1.66 times in GBS (P<0.001). The most downregulated spot was identified by mass spectrometry as cystatin C, which was downregulated 1.05 times in GBS (P<0.001). Peptide mass fingerprinting of the differential protein spot 1 identified as the Hp by mass spectrometry with a sequence coverage rate of 34.6% and an expected value of 0.000).

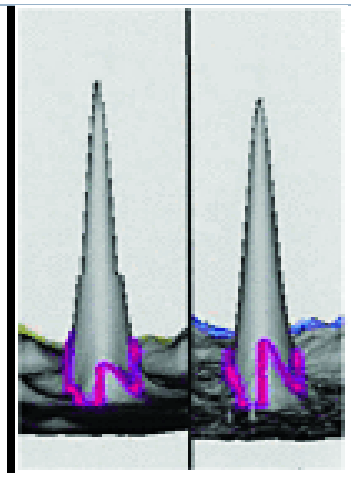

Figure 1. 3D reconstructed image of haptoglobin in the GBS (left) and control (right) groups. The spot was upregulated in GBS 1.66 times compared to the control group.

Conclusion

Overall, this work presents convincing evidence that the concentrations of Peripheral myelin protein 2 (P2) and αBC in the cerebrospinal fluid (CSF) serve as excellent prognostic biomarkers for the prognosis of Guillain-Barré Syndrome (GBS). The results of our study show a notable rise in both P2 and αBC levels from the time of admission to two weeks after the commencement of the disease. This increase is strongly associated with enhanced muscle strength in patients with Guillain-Barré syndrome (GBS). This correlation implies that elevated levels of these biomarkers are predictive of a more positive prognosis in GBS, representing a significant advancement in our comprehension of the disease.

LIST OF REFERENCES:

1. Davidson AI, Halstead SK, Goodfellow JA, Chavada G, Mallik A, Overell J, Lunn MP, McConnachie A, van Doorn P and Willison HJ: Inhibition of complement in Guillain-Barré syndrome: The ICA-GBS study. J Peripher Nerv Syst 1: 2017; 4-12 pp.
2. Uncini A, Shahrizaila N and Kuwabara S: Zika virus infection and Guillain-Barré syndrome: A review focused on clinical and electrophysiological subtypes. // J Neurol Neurosurg Psychiatry 88, 2017; 266–271 pp.
3. Hughes RA, Brassington R, Gunn AA and van Doorn PA: Corticosteroids for Guillain-Barré syndrome. // Cochrane Database Syst Rev 10: CD001446, 2016.
4. Ding Y, Han R, Jiang W, Xiao J, Liu H, Chen X, Li X and Hao J: Programmed death ligand 1 plays a neuroprotective role in experimental autoimmune neuritis by controlling peripheral nervous system inflammation of rats. // J Immunol 197: 3831-3840, 2016.
5. Halstead SK, Kalna G, Islam MB, Jahan I, Mohammad QD, Jacobs BC, Endtz HP, Islam Z and Willison HJ: Microarray screening of Guillain-Barré syndrome sera for antibodies to glycolipid complexes. // Neurol Neuroimmunol Neuroinflamm 3: e284, 2016.
6. Xu L, Gao TX, Chang SH, Jiang SM, Zhang LJ, Yang L. Role of lymphocyte-related immune-inflammatory biomarkers in detecting early progression of Guillain-Barré syndrome. // J Clin Neurosci. 2022;105:31-6.
7. Wu CL, Chao CH, Lin SW, Chien YY, Huang WY, Weng WC, et al. Case report: Plasma Biomarkers reflect immune mechanisms of Guillain–Barré syndrome. Front Neurol [Internet]. 2021 [cited 2023 Nov 15];12. Available from: https:// www.frontiersin.org/articles/10.3389/fneur.2021.720794
8. Stettner M, Zenker J, Klingler F, Szepanowski F, Hartung HP, Mausberg AK, et al. The role of peripheral myelin protein 2 in remyelination. // Cell Mol Neurobiol 2018;38:487-96.
9. Lim EMF, Nakanishi ST, Hoghooghi V, Eaton SEA, Palmer AL, Frederick A, et al. AlphaB-crystallin regulates remyelination after peripheral nerve injury. // Proc Natl Acad Sci 2017;114:1707–16.
10. Baumann N., Pham-Dinh D. Biology of oligodendrocyte and myelin in the mammalian central nervous system // Physiol Rev 2001;81:871-927.

Entered 20.08.2025