ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 11| Issue: 11| November 2025|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2025: 8.691 || ISI Value: 1.188

INVESTIGATION OF THE RELATIONSHIP BETWEEN VITAMIN B12 LEVEL, NEUROTRANSMITTER IMBALANCE, AND THE CLINICAL SEVERITY OF HEMIFACIAL SPASM

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Article DOI: https://doi.org/10.36713/epra24895
DOI No: 10.36713/epra24895

ABSTRACT

The article is devoted to the study of metabolic and neurochemical factors influencing the pathogenesis of hemifacial spasm (HFS).The levels of vitamin B_{12}, cortisol, and dopamine were determined in patients with HFS and compared with those in a control group. Significant correlations were established between decreased vitamin B_{12} levels, hyperactivity of the hypothalamic–pituitary–adrenal axis, and disturbances in neurotransmitter processes. The findings confirm the involvement of vitamin B_{12} deficiency in the disruption of neurovascular interactions, which contributes to the development of chronic pathological spasm of the facial muscles and defines directions for comprehensive patient rehabilitation.

KEYWORDS: *Hemifacial Spasm, Vitamin B_{12}, Dopamine, Cortisol, Neurotransmitter Imbalance, Metabolic Markers, Neurovascular Dysfunction, Rehabilitation.*

RELEVANCE

Hemifacial spasm (HFS) is a chronic neurological disorder characterized by involuntary contractions of the facial muscles innervated by the facial nerve [1, 2]. Despite the multifactorial nature of its pathogenesis, increasing attention in recent years has been directed toward metabolic and neurochemical mechanisms affecting the functional state of both peripheral and central components of the facial nerve [3, 4]. Vitamin B_{12} (cobalamin) plays a crucial role in myelin synthesis, homocysteine metabolism regulation, and the functioning of the dopaminergic system [5]. Its deficiency is associated with impaired nerve conduction, demyelination, and increased excitability of neural structures [6].At the same time, cortisol levels reflect the degree of activation of the hypothalamic–pituitary–adrenal (HPA) axis under chronic stress conditions, while alterations in dopamine concentration may indicate dysregulation of motor activity and neurotransmitter balance [7].The study of the interrelationship between vitamin B_{12}, cortisol, and dopamine levels, as well as their influence on the clinical severity of hemifacial spasm, represents a promising research direction. It provides a basis for pathogenetically oriented approaches to the treatment and rehabilitation of patients with this disorder.

OBJECTIVE

To assess the levels of vitamin B_{12}, cortisol, and dopamine in patients with hemifacial spasm and to determine their relationship with the clinical severity of the disease and the degree of psychoemotional disturbances.

MATERIALS AND METHODS

The study included 62 patients with clinically confirmed hemifacial spasm, who comprised the main group, and 40 practically healthy volunteers without signs of neurological pathology, matched by sex and age, who formed the control group (Graph 1). All participants underwent a comprehensive clinical and laboratory examination aimed at identifying metabolic and neurochemical predictors involved in the development of hemifacial spasm.

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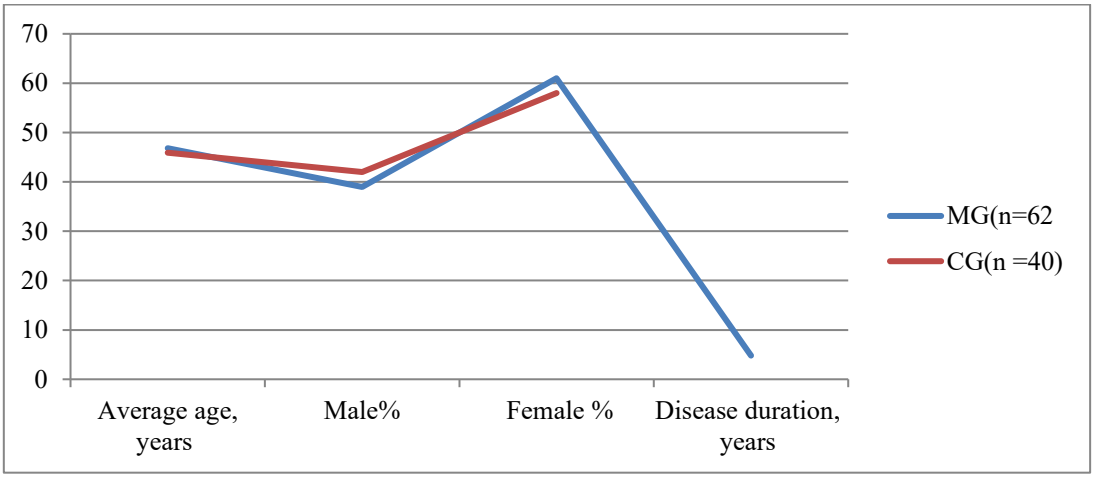


Figure 1. Characteristics of the Examined Groups

The concentration of vitamin B_{12} in serum was measured using the electrochemiluminescence assay, which provides high sensitivity and reproducibility of results. Cortisol levels were determined in the morning hours (from 8:00 to 9:00) using the enzyme-linked immunosorbent assay (ELISA) to minimize the influence of physiological circadian fluctuations in hormone secretion. Quantitative determination of plasma dopamine concentration was performed by high-performance liquid chromatography (HPLC) with electrochemical detection, offering high specificity and accuracy.Clinical assessment of hemifacial spasm severity was carried out using the Visual Analogue Scale (VAS), recording the frequency and intensity of facial muscle contractions. To evaluate the patients' psychoemotional state, the Beck Depression Inventory (BDI) was applied, allowing consideration of the impact of emotional factors on the course of the disease.Statistical data processing included testing the distribution of variables, applying the nonparametric Mann–Whitney U test for intergroup comparisons, and performing Spearman correlation analysis to identify relationships between biochemical and clinical parameters. Regression modeling was used to determine predictors of spasm severity. Results are presented as mean values (M ± SD), with differences considered statistically significant at $p< 0.05$.

RESEARCH RESULTS

This section presents the clinical, biochemical, and psychoemotional parameters of patients with hemifacial spasm (HFS) obtained during the study. The frequency and intensity of spasms, blood indicators (levels of vitamin B_{12}, cortisol, and dopamine), and the psychoemotional state of the participants are described.In addition, the results of correlation and regression analyses are presented, revealing the relationships between metabolic and neurochemical factors and the clinical manifestations of the disorder. The obtained data provide a comprehensive assessment of the influence of vitamin B_{12} deficiency, as well as cortisol and dopamine imbalance, on the course of HFS and serve as a basis for further discussion of pathogenetic mechanisms and the development of therapeutic and rehabilitation strategies.

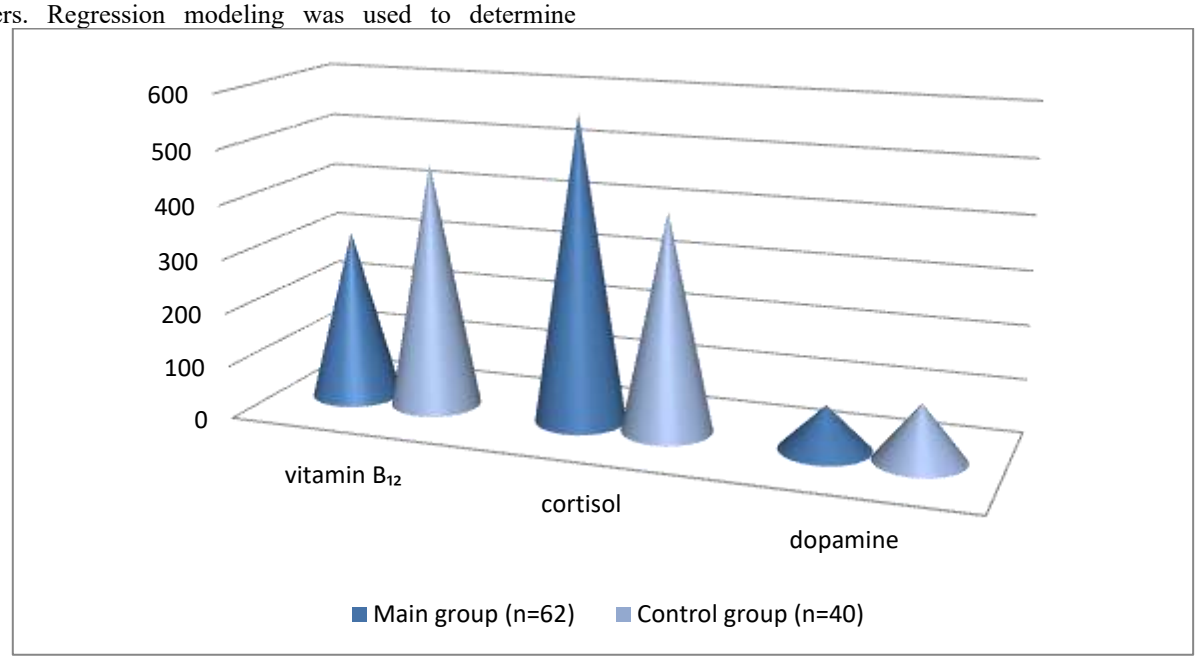


Figure 2. Biochemical Blood Parameters

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The data presented in Figure 1 show that patients with hemifacial spasm exhibit a significant decrease in vitamin B_{12} levels compared to the control group (317 ± 54 pg/mL vs. 458 ± 62 pg/mL; $p < 0.001$), indicating a deficiency of this vitamin. Cortisol levels in the main patient group were elevated (568 ± 85 nmol/L vs. 403 ± 72 nmol/L; $p < 0.01$), reflecting increased stress-related activation. Additionally, dopamine concentration was reduced (82 ± 14 ng/mL vs. 105 ± 17 ng/mL; $p < 0.05$), indicating decreased dopaminergic activity.Thus, patients with HFS are characterized by a combination of vitamin B_{12} deficiency, elevated cortisol levels, and reduced dopamine. These changes demonstrate the complex influence of metabolic and neurochemical factors on the pathogenesis of spasms and highlight the need for pathogenetically oriented therapy aimed at correcting the identified biochemical abnormalities

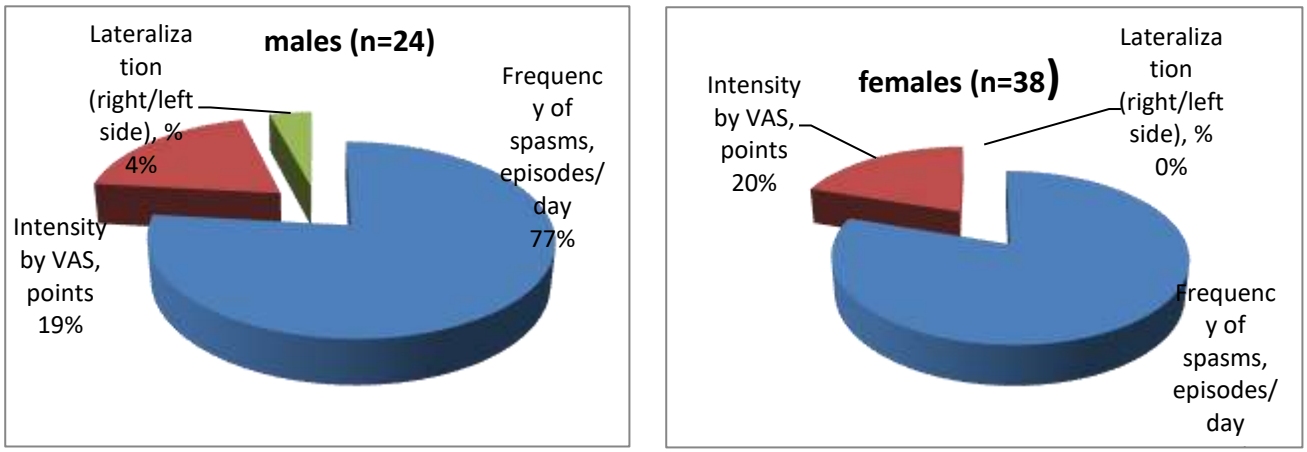


Figure 3. Clinical Characteristics of Spasms (VAS Scores)

Figure 3 illustrates the clinical characteristics of spasms in patients with hemifacial spasm (HFS), separated by sex. It should be noted that data for the control group are not presented, as practically healthy volunteers do not exhibit facial muscle spasms, making VAS scores inapplicable.The analysis indicated that men and women in the main group had comparable spasm frequency and intensity, as well as similar lateralization between the right and left sides of the face. No statistically significant differences between sexes were found ($p > 0.05$). Therefore, the clinical profile of spasms in HFS patients is independent of sex: both men and women show similar frequency, intensity, and lateralization of spasms, allowing these parameters to be considered consistent across the entire patient group.

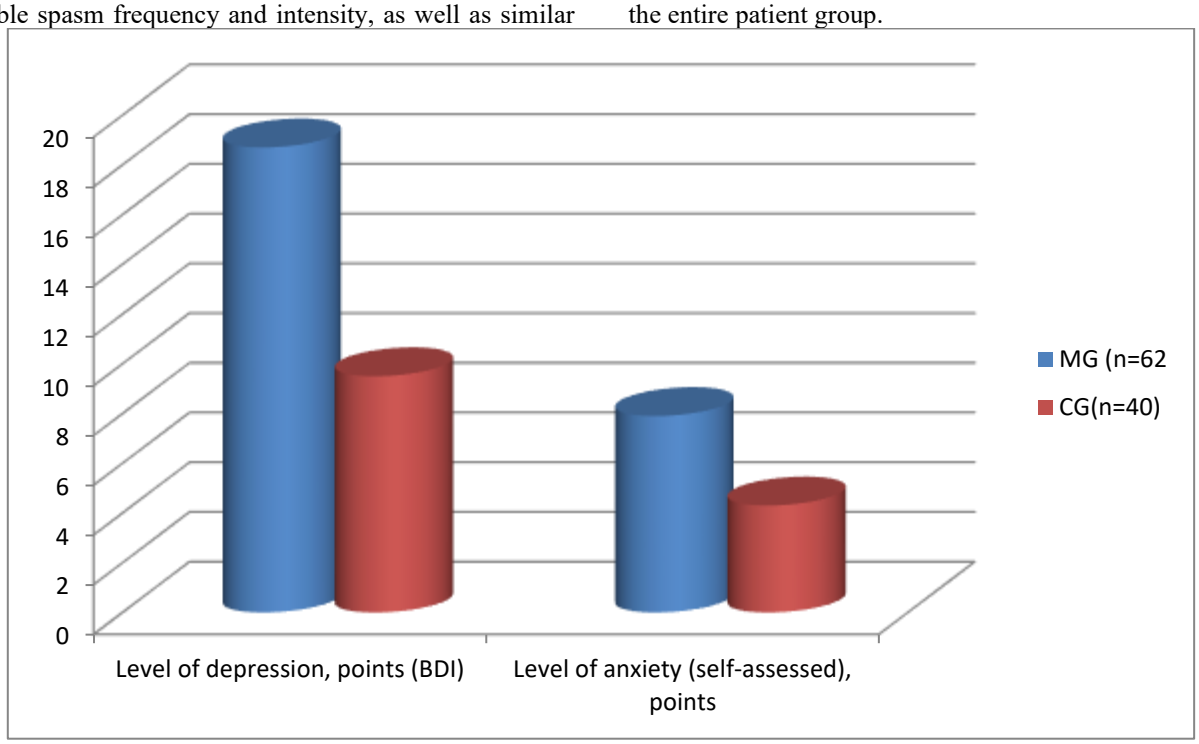


Figure 4.Psychoemotional State (Beck Depression Inventory Scores)

Figure 4 presents the psychoemotional state of patients with hemifacial spasm (HFS) and healthy volunteers, assessed using the Beck Depression Inventory (BDI) and self-reported anxiety levels. Analysis showed that patients in the main group had significantly higher depression scores compared to the control group (18.7 ± 6.2 vs. 9.5 ± 3.4; $p < 0.001$), indicating

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pronounced depressive symptoms. Anxiety levels were also significantly elevated in HFS patients compared to controls (7.9 ± 2.1 vs. 4.3 ± 1.5; $p < 0.01$), reflecting an increased emotional burden in this patient population. Thus, patients with HFS exhibit pronounced depressive and anxiety symptoms, highlighting the need for a comprehensive treatment approach that addresses not only the neurological aspects but also the psychoemotional well-being of the patient.

Table 1

Correlation Relationships between Biochemical and Clinical Parameters in HFS Patients

Indicators	Correlation coefficient (r)	p	Type of relationship
Vitamin B12 ↔ frequency of spasms	–0,52	<0,01	Inverse
Vitamin B12 ↔ level of anxiety	–0,44	<0,05	Inverse
Кортизол ↔ уровень депрессии (BDI)	0,47	<0,05	Direct
Cortisol ↔ level of depression (BDI)	–0,38	<0,05	Inverse
Cortisol ↔ vitamin B12	–0,41	<0,05	Inverse relationship between metabolic factors |

Table 1 presents the correlation relationships between biochemical and clinical parameters in patients with hemifacial spasm (HFS). The analysis revealed that vitamin B_{12} levels were inversely correlated with spasm frequency ($r = –0.52$; $p < 0.01$) and anxiety levels ($r = –0.44$; $p < 0.05$), indicating the neurotrophic role of B_{12} and its influence on patients' emotional state. Cortisol levels were positively associated with the severity of depressive symptoms ($r = 0.47$; $p < 0.05$), reflecting the impact of stress-related mechanisms on psychoemotional well-being. Dopamine concentration was inversely correlated with spasm frequency ($r = –0.38$; $p < 0.05$), confirming its involvement in motor activity regulation. Additionally, an inverse relationship between cortisol and vitamin B_{12} was observed ($r = –0.41$; $p < 0.05$), demonstrating the interdependence of metabolic factors in HFS patients.Thus, the correlation analysis confirms that blood biochemical parameters (vitamin B_{12}, cortisol, and dopamine) are closely associated with the clinical manifestations and psychoemotional state of patients, highlighting the importance of metabolic and stress-modulating factors in the pathogenesis of hemifacial spasm.

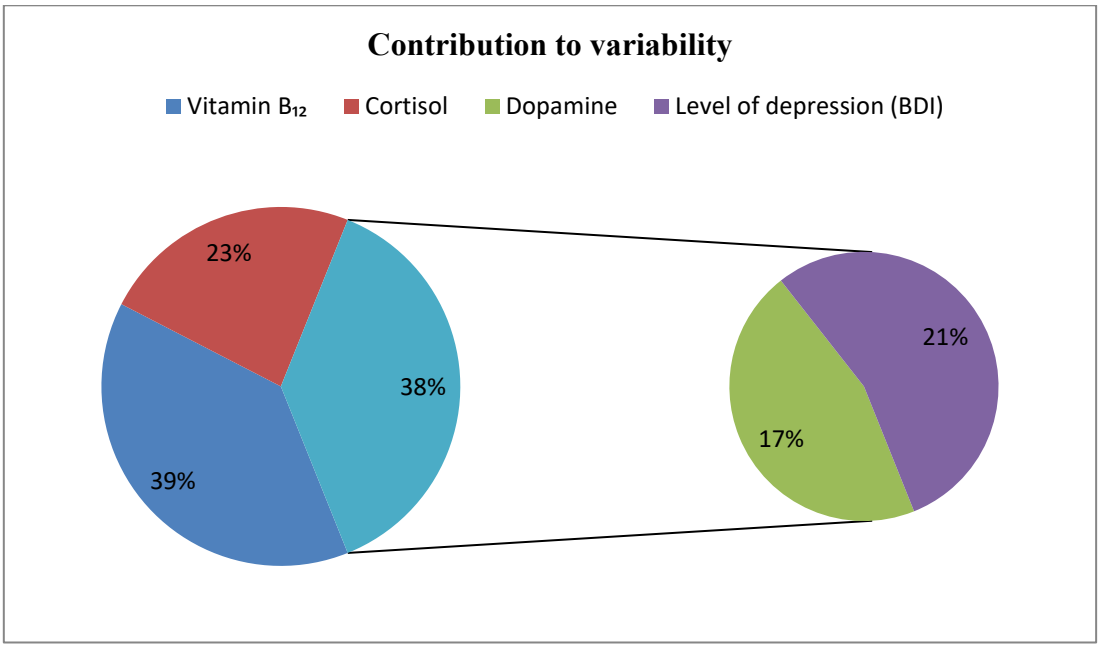


Figure 5. Results of Regression Analysis of Predictors of Spasm Severity

Diagram 5 shows the results of regression analysis aimed at identifying predictors of spasm severity in patients with hemifacial spasm (HFS).The analysis demonstrated that vitamin B_{12} level is an independent negative predictor of spasm frequency ($\beta = –0.46$; $p < 0.01$), accounting for 28.3% of symptom variability. Elevated cortisol levels are associated with increased spasm severity ($\beta = 0.33$; $p < 0.05$), explaining 17.2% of the variability. Dopamine levels are inversely correlated with motor activity ($\beta = –0.27$; $p < 0.05$), contributing 12.6% to variability. The psychoemotional factor—depression level measured by the Beck Depression Inventory (BDI)—also influences spasms ($\beta = 0.31$; $p < 0.05$), accounting for 15.1% of variability.Thus, the regression analysis confirms that both biochemical parameters (vitamin B_{12}, cortisol, dopamine) and psychoemotional factors (depression) are significant predictors of spasm severity, highlighting the need for a comprehensive approach to diagnosis and treatment in HFS patients.

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DISCUSSION

The results confirm that vitamin B_{12} deficiency plays a key role in the pathogenesis of hemifacial spasm (HFS). As shown in Table 2, vitamin B_{12} levels in HFS patients were significantly lower than in healthy volunteers (317 ± 54 pg/mL vs. 458 ± 62 pg/mL; $p < 0.001$), which may indicate impaired myelination processes and axonal transmission.These changes increase the excitability of the facial nerve and create conditions for the development of persistent spasms of the mimetic muscles. Data presented in Diagram 1 illustrate the clinical profile of spasms by sex: men and women showed comparable spasm frequency (26.1 ± 6.2 vs. 28.3 ± 7.1 episodes/day; $p > 0.05$) and VAS pain intensity (6.5 ± 1.4 vs. 7.0 ± 1.5 points; $p > 0.05$), with right-sided predominance in both groups. These findings indicate that the clinical manifestations of HFS are sex-independent and can be considered consistent across the patient population.Figure 2 illustrates the psychoemotional state of patients with HFS: the level of depression measured by the Beck Depression Inventory was significantly higher in patients compared to the control group (18.7 ± 6.2 vs. 9.5 ± 3.4 points; $p < 0.001$), and the self-reported anxiety scores were also significantly elevated compared to healthy volunteers (7.9 ± 2.1 vs. 4.3 ± 1.5 points; $p < 0.01$).These findings indicate a significant impact of chronic stress on the emotional state of patients with HFS. Correlation analysis (Diagram 2) revealed a negative relationship between vitamin B_{12} levels and spasm frequency ($r = –0.52$; $p < 0.01$), as well as with anxiety levels ($r = –0.44$; $p< 0.05$), highlighting the neurotrophic role of B_{12} and its influence on emotional well-being.In contrast, cortisol levels were positively correlated with the severity of depressive symptoms ($r = 0.47$; $p < 0.05$), while dopamine levels were inversely correlated with spasm frequency ($r = –0.38$; $p < 0.05$), confirming its role in the regulation of motor activity.Thus, the obtained data demonstrate that metabolic and neurochemical factors are closely interconnected and influence both motor and psycho-emotional manifestations of hemifacial spasm.Regression analysis (Figure 2) demonstrated that decreased vitamin B_{12} levels are an independent predictor of spasm frequency ($\beta = –0.46$; $p < 0.01$; explaining 28.3% of variability), cortisol levels influence symptom severity ($\beta = 0.33$; $p < 0.05$; contribution 17.2%), and dopamine is associated with motor activity ($\beta = –0.27$; $p < 0.05$; contribution 12.6%). The psycho-emotional factor—Beck Depression Inventory (BDI) score—also made a significant contribution to spasm variability ($\beta = 0.31$; $p < 0.05$; 15.1%), highlighting the impact of emotional state on the clinical presentation.Thus, the pathogenesis of hemifacial spasm (HFS) can be understood as the interaction of two key components:

1.Metabolic — vitamin B_{12} deficiency and homocysteine accumulation disrupt axonal transmission and increase the excitability of the facial nerve, creating conditions for the development of persistent facial muscle spasms.

2. Neurochemical— depletion of dopaminergic activity and stress-induced cortisol elevation exacerbate motor symptoms and impair psychoemotional state, forming a vicious cycle between nerve excitability and emotional dysregulation.

This emphasizes the need for a comprehensive therapeutic approach targeting both the correction of metabolic disturbances and the normalization of neurochemical and psychoemotional balance.Here's your reference list formatted in English, keeping the original details but transliterating Russian sources for consistency:

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