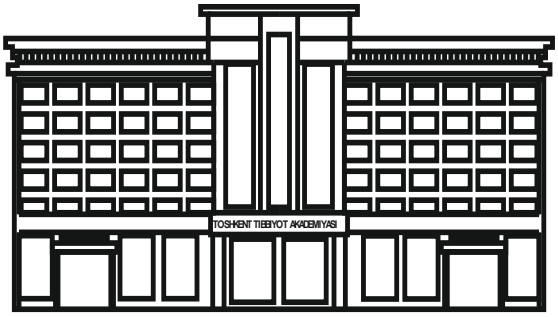
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THE ROLE OF PSYCHOMETRIC SCALES IN THE DEVELOPMENT OF SOMATOFORM AUTONOMIC DYSFUNCTION IN CHRONIC CEREBRAL ISCHEMIA

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Abstract*. This study analyzed psychometric indicators (COMPASS-31, PSS-10, HADS-A) of somatoform autonomic dysfunction (SAD) in patients with chronic cerebral ischemia (CCI). A total of 118 participants were divided into three groups. Intergroup differences were evaluated using the Kruskal–Wallis test, while correlations between indicators were assessed with Spearman's correlation. The results demonstrated significant differences across all scales (p<0.001) and positive correlations between psychometric measures.*

Keywords: *chronic cerebral ischemia, somatoform autonomic dysfunction, COMPASS-31, PSS-10, HADS-A*

Introduction. Chronic cerebral ischemia (CCI) is one of the most pressing problems of modern neurology, characterized by progressive cognitive, emotional, and somatovegetative disturbances that significantly reduce patients' quality of life [1]. A key component of this pathology is the involvement of autonomic regulation, which has been shown to play a decisive role in both the course and prognosis of cerebrovascular diseases [2]. According to recent studies, autonomic dysfunction is observed in the majority of patients after acute or chronic ischemic brain damage and is associated with worse clinical outcomes [3].

Somatoform autonomic dysfunction (SAD) represents a clinically heterogeneous syndrome manifesting as diverse vegetative complaints not fully explained by structural damage. SAD has been increasingly recognized as a comorbid and prognostically significant condition in patients with CCI [1,3]. Reliable detection and quantification of autonomic disorders have been facilitated by standardized questionnaires, in particular the COMPASS-31 scale, which has been validated as a refined and concise instrument for assessing autonomic symptoms in both research and clinical settings [4].

Despite growing evidence of the clinical relevance of autonomic dysfunction in CCI, the biochemical correlates of SAD remain poorly understood. Stress-related neuroendocrine markers such as cortisol and β-endorphin are of special interest, as they reflect the activity of the hypothalamic-pituitary-adrenal axis and endogenous opioid system, respectively. Dysregulation of these systems has been linked to autonomic imbalance, impaired stress adaptation, and unfavorable outcomes in cerebrovascular pathology [2,3].

Thus, investigation of the combined biochemical and clinical associations of SAD in patients with CCI appears to be of high scientific and practical importance. This work aims to contribute to the understanding of these mechanisms by analyzing both biochemical markers and standardized clinical parameters, in order to identify potential diagnostic and therapeutic targets.

Relevance. Autonomic dysfunction decreases quality of life and worsens prognosis in CCI patients [9]. The availability of simple and reliable methods to identify SAD is highly valuable in clinical practice. Determining interrelations among psychometric scales improves diagnostic accuracy and facilitates the development of treatment strategies [10,11].

Materials and Methods. **Study design and participants.** This cross-sectional observational study was conducted at Tashkent State Medical Institute in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants. A total of **118 subjects** were included and divided into three groups:

Group 1 (n = 48): patients with chronic cerebral ischemia (CCI) and somatoform autonomic dysfunction (SAD);

Group 2 (n = 40): patients with CCI without SAD;

Group 3 (n = 30): healthy controls.

Inclusion criteria were age >40 years, confirmed diagnosis of stage I–II CCI by clinical and neuroimaging data (Groups 1–2), and ability to complete psychometric questionnaires.

Exclusion criteria included acute cerebrovascular event within 6 months, severe psychiatric illness, endocrine disorders affecting cortisol metabolism (e.g., Cushing's, Addison's disease), systemic inflammatory conditions, chronic glucocorticoid or opioid use, pregnancy or lactation.

Assessments. Biochemical analysis: Serum cortisol and β-endorphin levels were measured in morning fasting venous blood samples using ELISA.

Psychometric scales: Autonomic symptoms were assessed with the COMPASS-31, perceived stress with PSS-10, and anxiety/depression with the HADS (A and D subscales). SAD was determined according to COMPASS-31 cutoff values.

Statistical analysis. Data were analyzed using SPSS 26.0. Continuous variables were presented as mean ± SD or median (IQR), categorical as n (%). Between-group comparisons were performed with the Kruskal–Wallis test; Dunn's post-hoc test was applied when appropriate. Correlations were assessed with Spearman's method. Independent predictors of SAD were identified using logistic regression with odds ratios (OR) and 95% confidence intervals (CI). A p-value <0.05 was considered statistically significant.

Results

Table 1.

Distribution of participants by group and sex

Group	Total participants (n)	Male, n (%)	Female, n (%)
Group 1	48	20 (41.7)	28 (58.3)
Group 2	40	11 (27.5)	29 (72.5)
Group 3	30	15 (50.0)	15 (50.0)

Note. Data are presented as n (%).

The distribution showed that in Group 1, men comprised 41.7% (20 individuals), and women 58.3% (28 individuals). In Group 2, men accounted for 27.5% (11 individuals), while women comprised 72.5% (29 indi-

viduals). In Group 3, males and females were equally distributed (50%–50%). Although gender distribution was relatively unbalanced across groups, it was sufficient for analysis.

Table 2.

Demographic and clinical characteristics of participants

Indicator	Group 1 (n=48)	Group 2 (n=40)	Group 3 (n=30)
Age (years)	61.52 ± 8.15	55.40 ± 10.08	52.53 ± 4.88
COMPASS-31 (pts)	33.56 ± 9.73	19.55 ± 8.14	14.80 ± 5.16
PSS-10 (pts)	23.29 ± 8.55	14.62 ± 7.03	8.63 ± 1.90
HADS-A (pts)	9.90 ± 2.90	8.00 ± 2.33	6.47 ± 1.74

Note. Data are presented as mean ± SD.

The average age was significantly higher in Group 1 (61.52 ± 8.15 years) compared to Group 2 (55.40 ± 10.08) and Group 3 (52.53 ± 4.88) ($p<0.001$). COMPASS-31 scores were highest in Group 1 (33.56 ± 9.73) compared with Group 2 (19.55 ± 8.14) and Group 3 (14.80 ± 5.16). PSS-10 scores were also higher in Group 1 (23.29 ± 8.55) than in Group 2 (14.62 ± 7.03) and Group 3 (8.63 ± 1.90). HADS-A scores were elevated in Group 1 (9.90 ± 2.90) compared with Group 2 (8.00 ± 2.33) and Group 3 (6.47 ± 1.74), with the Group 1–Group 3 difference being highly significant ($p<0.001$).

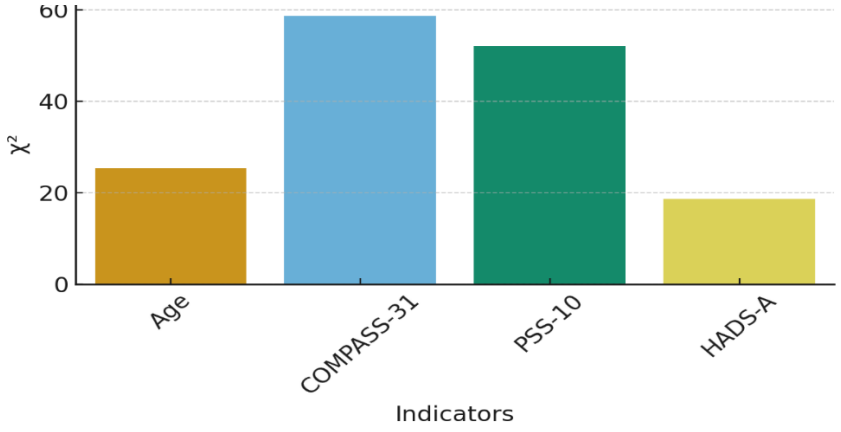


Figure 1. Intergroup differences (χ^2 values) for age, COMPASS-31, PSS-10, and HADS-A

All χ^2 values were significant at $p<0.001$, indicating meaningful intergroup differences. The highest χ^2 values were noted for COMPASS-31 and PSS-10, reflecting pronounced group variation in autonomic symptoms and stress levels.

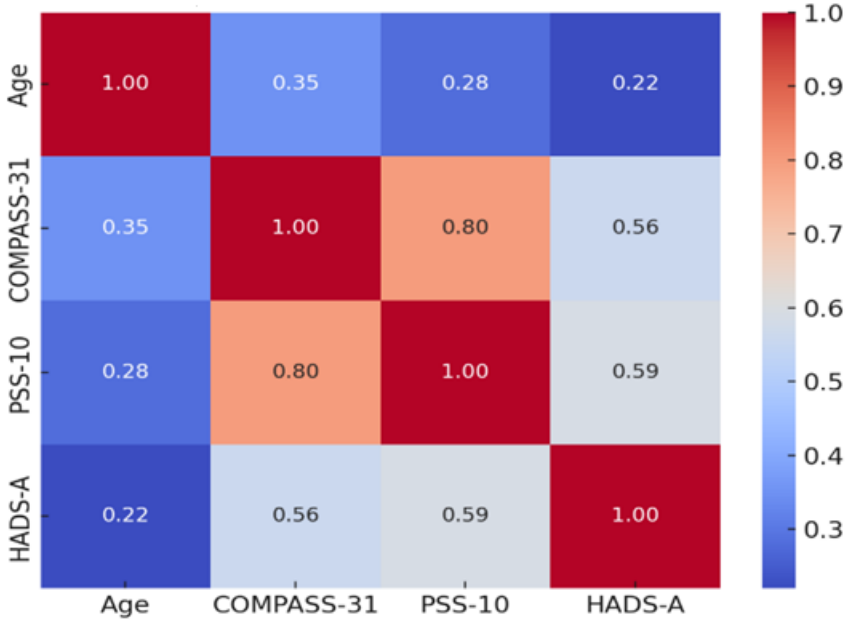


Figure 2. Correlation matrix of age, COMPASS-31, PSS-10, and HADS-A

Red denotes positive, and blue denotes negative correlations. Strong positive correlations were observed between COMPASS-31 and PSS-10 (ρ=0.80; p<0.001), and between COMPASS-31 and HADS-A (ρ=0.56; p<0.001). A moderate positive correlation was also identified between PSS-10 and HADS-A (ρ=0.59; p<0.001). Age demonstrated weaker but significant positive associations with psychometric indicators. These results highlight the interdependence of psychometric scales and the necessity of their combined use in assessing SAD.

Conclusion

This study demonstrated that somatoform autonomic dysfunction (SAD) in patients with chronic cerebral ischemia (CCI) is closely associated with psychometric indicators. The use of standardized tools such as COMPASS-31, PSS-10, and HADS-A not only revealed statistically significant intergroup differences but also highlighted strong intercorrelations between stress perception, anxiety symptoms, and autonomic imbalance. These findings underscore the integral role of psychometric evaluation in the multidimensional assessment of patients with CCI.

Importantly, psychometric scales can serve as reliable, non-invasive, and cost-effective instruments for the early detection, monitoring, and longitudinal follow-up of SAD. Their application contributes to identifying high-risk subgroups, optimizing diagnostic accuracy, and tailoring individualized therapeutic strategies. Moreover, the integration of psychometric profiling into clinical practice bridges the gap between subjective symptomatology and objective biochemical markers, thereby strengthening the overall diagnostic framework.

Our results are in line with previous evidence. Zhang et al. [7] demonstrated that alterations in heart rate variability are closely related to SAD, further supporting the link between autonomic regulation and clinical outcomes. At the same time, Röhricht et al. [12] emphasized the therapeutic importance of psychotherapeutic approaches in managing somatoform disorders, suggesting that a combined use of psychometric assessment and targeted therapy may provide a more comprehensive management strategy.

Taken together, these data indicate that systematic use of psychometric scales represents a practical and clinically valuable approach in the diagnosis and monitoring of SAD among CCI patients. Incorporating such tools into routine neurological practice may enhance early recognition, facilitate timely interventions, and ultimately improve patient outcomes.

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