STRUCTURAL AND FUNCTIONAL CORRELATES OF POST-STROKE DEPRESSION DEPENDING ON STROKE SEVERITY

Muradimova A.R.
PhD, Head of the Department of Neurology and Psychiatry, Fergana Medical Institute of Public Health, Fergana, Republic of Uzbekistan
https://doi.org/10.5281/zenodo.20501848

ARTICLE INFO

Received: 26th May 2026
Accepted: 28th May 2026
Online: 30th May 2026

KEYWORDS

post-stroke depression, ischemic stroke, stroke severity, magnetic resonance imaging, structural correlates, neurological deficit, cognitive impairment, anxiety, apathy, neurorehabilitation

ABSTRACT

This thesis investigates the structural and functional correlates of post-stroke depression in relation to ischemic stroke severity. Particular attention is given to the association between lesion localization and volume, neurological deficit, cognitive impairment, anxiety, apathy, and depressive symptoms. The findings demonstrate that cortical strokes are more commonly associated with anxiety-depressive manifestations, whereas subcortical lesions are characterized by apathetic-aboulic symptoms. The study emphasizes the importance of integrating neuroimaging and psychometric assessment for early prediction and individualized neurorehabilitation of post-stroke depression.

Relevance. Post-stroke depression (PSD) is one of the most common complications of ischemic stroke, occurring, according to various data, in 30-60% of patients [4-6, 8, 10]. It substantially affects the pace of recovery, motivation for rehabilitation, and the overall prognosis of the disease [1-3, 7, 9]. Establishing the relationship between structural brain damage and the severity of emotional and cognitive impairments opens new opportunities for early diagnosis and personalized therapy.

Aim of the study. To investigate the structural and functional correlates of post-stroke depression and anxiety in patients with ischemic stroke of varying severity using magnetic resonance imaging (MRI) and clinical psychometric scales.

Materials and methods. The study included 64 patients (mean age 59.4 +/- 8.7 years) who had experienced ischemic stroke in the territory of the middle cerebral artery and were in the early recovery period (7-90 days). According to MRI findings, patients were divided into two groups: Group I (n = 34), cortical strokes involving the frontotemporal, parietal, and insular regions; and Group II (n = 30), subcortical strokes involving the basal ganglia, thalamus, and internal capsule. The severity of neurological deficit was assessed using the NIHSS; cognitive functions were assessed using MoCA; depression using the Beck Depression Inventory (BDI); anxiety using the Spielberger-Hanin State-Trait Anxiety Inventory (STAI); and apathy using the Starkstein Apathy Scale. Lesion volume was determined by semi-automatic analysis of MRI slices. Correlation analysis was performed using Pearson's r, with statistical significance set at $p < 0.05$.

Results. The mean NIHSS score for stroke severity was 8.7 +/- 3.2 points; in the subcortical stroke group, the values were significantly higher than in the cortical stroke group (9.8 +/- 2.7 vs. 7.6 +/- 3.1; $p < 0.05$). MRI data showed that ischemic lesions in patients with subcortical involvement were more often localized in the caudate nucleus, thalamus, and anterior limb of the internal capsule, which was accompanied by marked dysregulation of subcortical-frontal connections. In patients with cortical strokes, lesions of the dorsolateral prefrontal cortex and cingulate gyrus, structures responsible for emotional regulation and cognitive control, predominated.

Analysis of psychoemotional scales showed the following: BDI (depression) scores were 21.3 +/- 4.8 points in patients with cortical strokes and 19.5 +/- 5.2 points in those with subcortical strokes ($p > 0.05$), indicating a tendency toward greater severity in cortical lesions; STAI (anxiety) scores were 46.7 +/- 6.2 and 42.8 +/- 5.4 points, respectively ($p < 0.05$); and Starkstein Apathy Scale scores were 17.4 +/- 2.9 in cortical strokes and 22.6 +/- 3.1 in subcortical strokes ($p < 0.01$). Thus, cortical strokes were characterized by a predominance of anxiety-depressive disorders, whereas subcortical strokes were associated mainly with an apathy-abulic syndrome.

Correlation analysis revealed significant relationships between neurological deficit and the severity of emotional disturbances: NIHSS and BDI, $r = 0.61$ ($p < 0.01$); NIHSS and apathy scale, $r = 0.58$ ($p < 0.01$); and MoCA and BDI, a negative correlation of $r = -0.49$ ($p < 0.05$), indicating a relationship between cognitive deficit and emotional status.

Structural analysis showed that an ischemic lesion volume greater than 25 cm3 was associated with increased severity of depression and anxiety, as well as a significant decline in cognitive functions (MoCA < 20 points). Lesions of the basal ganglia and thalamus were accompanied by disturbances in dopaminergic and serotonergic regulation, which was supported by biochemical data showing decreased BDNF levels and increased IL-6 in the subgroup of patients with severe depression. MRI morphometry revealed reduced gray matter volume in the anterior cingulate gyrus, orbitofrontal cortex, and caudate nucleus, which correlated with the severity of apathy and reduced initiative. At the same time, involvement of hippocampal and temporal regions was accompanied by increased anxiety and feelings of hopelessness.

Dynamic follow-up at three months showed that patients with smaller lesion volumes and moderate depression had more favorable recovery indicators according to the Stroke-Specific Quality of Life Scale (SS-QOL) and a 25-30% reduction in BDI scores. In subcortical strokes, recovery of the emotional state occurred more slowly, indicating the need for prolonged psychocorrective support.

Conclusion. The severity of ischemic stroke and the pattern of parenchymal injury according to MRI have a direct influence on the severity of post-stroke depression, anxiety, and apathy. Cortical strokes are associated with a predominance of anxiety-depressive symptoms, whereas subcortical strokes are associated with apathy-abulic manifestations. Lesion volume and involvement of regulatory structures of the limbic-frontal network directly correlate with the severity of emotional disturbances. Comprehensive assessment using MRI, BDI, STAI, MoCA, and the Starkstein Apathy Scale makes it possible to predict the

development of PSD and individualize neurorehabilitation programs in the early recovery period..

References:

1. Muradimova A.R. Nursing care for patients with hemorrhagic stroke. In: Innovations in Medicine. Proceedings of the 1st International Scientific and Practical Conference. Makhachkala, 2019; Vol. II: 188.
2. Usmanova D.D., Muradimova A.R., Ashuraliev I.M. Blood neurotrophic proteins and their correlations with vascular risk factors in patients with dyscirculatory encephalopathy complicated by vascular dementia. Territory of Science. 2019;5:12-16.
3. Muradimova A.R., Usmanova D.D., Sadikov U.T. Intersecting parallels: vascular dementia and ischemic heart disease. Current Problems of Pathophysiology. 2020:85-88.
4. Muradimova A.R. Neurophysiological aspects of metabolic therapy for chronic cerebral ischemia. Innovations in Medicine. 2019:192-197.
5. Gafurov B.G., Rakhmatullaev M.K. Epidemiology of Stroke in Uzbekistan: Current Trends and Ways to Optimize Neurorehabilitation. Tashkent: Ilm-Ziyo; 2021. 156 p.
6. Muradimova A.R., Madzhidova Yo.N. Clinical and psychological characteristics of patients with post-stroke depression and methods of their correction. Neurology and Psychiatry Journal. 2024;2:45-53.
7. Rakhimov Sh.J., Nurmatov A.Sh. Improvement of early rehabilitation methods in ischemic stroke. Tashkent: Medical Publishing House; 2023. 112 p.
8. Levin O.S., Damulin I.V. Post-stroke cognitive and affective disorders: diagnosis, prognosis, therapy. Neurological Journal. 2020;25(6):15-23.
9. Suslina Z.A., Gulevskaya T.S. Current concepts of the pathogenesis of post-stroke depression. S.S. Korsakov Journal of Neurology and Psychiatry. 2019;10:72-80.
10. Robinson R.G., Jorge R.E. Post-Stroke Depression: Mechanisms and Treatment. Cambridge: Cambridge University Press; 2016. 320 p.