Oral Communication

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Is There any Neural Substrates of Post-Stroke Depression? A Cross-Sectional Study

Topic: Neuroscience

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BACKGROUND

Post-stroke depression (PSD) affects 27% of individuals after stroke, hindering recovery and quality of life. The underlying pathophysiology remains unclear, with ongoing debate between neurobiological mechanisms driven by stroke-related brain alterations and psychological reactions to disability. This study hypothesizes that PSD does not have a specific neural basis linked to any particular brain structure and is not related to lesion volume but may be driven by the integrity of the cerebral parenchyma.

METHODS

We analyzed 126 first-ever unilateral hemispheric stroke patients from the DOBRAS cohort (NCT: 03203109) to assess the relationship between PSD and stroke characteristics, including lesion location, volume, and white matter hyperintensities. Inclusion criteria were age 18-80, confirmed ischemic or hemorrhagic stroke, Aphasic Depression Rating Scale (ADRS) evaluation within 60 days, and magnetic resonance imaging (MRI) acquisition approximately two months post-stroke. Exclusion criteria included prior stroke, neurological or psychiatric comorbidities, unstable medical conditions, early or late MRI, bilateral or multiple strokes, severe subarachnoid hemorrhage, or brain hernia. PSD was defined as ADRS ≥ 11. MRI scans (1.5T) included T1, T2, and fluid-attenuated inversion recovery (FLAIR) sequences for lesion segmentation and white matter hyperintensities (WMH), graded with the Fazekas scale: scores 0-1 vs. ≥ 2. Voxel-based lesion-symptom mapping (VLSM) was performed to examine correlation between lesion characteristics and ADRS scores. A p value < 0.05 was considered as statistically significant.

RESULTS

Among 126 participants (median age 65.9 years, 67% male), 53 (42%) had a PSD with a mean ADRS score 16 (4). Strokes encroached many structures. Lesion side and volume did not differ in individuals with or without PSD, and no correlation was found between stroke volume and ADRS scores (r = 0.077; p = 0.389). Higher ADRS scores were associated with moderate-to-severe WMH (p = 0.011). VLSM analysis did not reveal any association between lesion location and PSD (p = 0.429).

CONCLUSION

Post-stroke depression is associated with confluent WMH but not with damage to a specific brain structure. These findings suggest that diffuse cerebrovascular pathology, rather than focal structural damage, plays a key role in PSD pathophysiology. PSD should be systematically detected and treated when necessary in individuals with confluent WMH.

Statement of Disclosures

None

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