Volume 3, Issue 5, May 2025 ISSN (E): 2938-3765

NEUROIMAGING MARKERS AND CLINICAL SEVERITY CORRELATIONS IN VASCULAR ENCEPHALOPATHY: A RETROSPECTIVE COHORT STUDY

Nozimaxon Abdurasulova 1
Dilshod Tolibov 2
Sharodiya Das 3
1PhD Student of the Department of Neurology, Tashkent State Medical University, Uzbekistan
E-mail: shavkatjonmirzaliyev1989@gmail.com. ORCID ID: 0009-0008-9080-9490
2DSc, Docent of the Department of Neurology, Tashkent State Medical University, Uzbekistan
E-mail: dr.dilshodts1984@gmail.com. ORCID ID: 0000-0002-4283-2628
3Student of International Faculty, Tashkent State Medical University, Uzbekistan
E-mail: sharodiya123@gmail.com. ORCID ID: 0009-0009-7563-5741

Abstract
Vascular encephalopathy (VE) is a critical cause of cognitive impairment but the correlation between neuroimaging abnormalities and clinical severity is unknown. A retrospective study was conducted on 81 patients with MRI-proved VE to investigate correlations of imaging measures with clinical rating scales. These individuals underwent standard 3T MRI with quantitation of atrophy patterns on the Global Cortical Atrophy scale and white matter lesions on the Fazekas scale. Clinical severity was quantified with the Stephen-Porges autonomic dysfunction scale and PSM25 neurologic deficit scale. The study revealed correlation of severity of atrophy in the frontal lobe with respect to PSM25 scores (β=0.76, $p<0.001$), accounting for 58% variance in scores. The burden of white matter lesions was strongly correlated with autonomic dysfunction (r=0.65, $p<0.001$). Male individuals had greater ischemic changes than female individuals (mean Fazekas grade 2.4 vs 1.8, p=0.02). The study confirms the clinical use of visual rating scales in the diagnosis of VE and sets the stage for the primary role of the integrity of the frontal lobe in the concomitant neurologic deficits.

Keywords: Vascular Encephalopathy, Neuroimaging, MRI, Stephen-Porges Scale, PSM25 Scale, Atrophy, White Matter Lesions.

Introduction

Vascular encephalopathy (VE) is now the most common cause of cognitive deterioration, particularly among the elderly [3, 10, 15]. The hallmark of VE is long-standing ischemic brain alteration, and the disease manifests itself as cognitive disturbances and motor deficits substantially affecting the quality of life. It has been documented that 15-20% of the total dementia cases across the globe are due to VE, emphasizing the urgency for timely and accurate diagnosis [1, 7, 11, 14].

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

Neuroimaging with magnetic resonance imaging has been an important diagnostic tool in VE to show cortex atrophy and white matter lesions [4, 6, 8, 13]. Isolated neuroimaging markers' clinical importance is less documented. Although in previous research correlations have been established between brain atrophy and cognition, the majority have been carried out based on volume measurements instead of ordinal visual rating scales [5, 9, 16]. The Global Cortical Atrophy scale and the Fazekas scale are both useful to grade cortex atrophy and white matter lesions, respectively [2, 12].

The aim of this research is to bridge the gap between neuroimaging and clinical practice by examining the correlation of MRI findings with clinical severity in VE patients. We predicted that atrophy of the frontal lobe would correlate more strongly with deficits in the neurologic system, and that white matter lesion burden would correlate more strongly with autonomic function. We also investigated demographic differences based on age and sex in order to shed light on the clinical heterogeneity of presentations of VE.

Methods

Study Design:

It was a retrospective cohort study of 81 consecutive VE patients of age group 50 to 85 years who had been admitted to a tertiary care clinic at Tashkent from 2018 to 2023. Institutional review board approval has been provided, and the data of the patients have been de-identified.

Inclusion criteria:

- VE diagnosis using MRI based on STRIVE version 2 criteria
- Complete clinical evaluation data
- Lack of significant comorbid psychiatric conditions

Participants with mixed dementia or confounding neurological conditions were excluded.

Imaging protocol: An MRI was performed on a 3T Siemens Skyra scanner using the following standard protocols:

- 3D T1-weighted MPRAGE with isotropic resolution of
- Axial FLAIR
- Susceptibility-weighted imaging

Neuroimaging scans included the GCA atrophy scale and the Fazekas scale for periventricular white matter lesions. The scans were read by neuroradiologists who were blinded to the clinical data and who had high inter-rater reliability (ICC > 0.85).

Clinical severity was evaluated by:

- Stephen-Porges autonomic function scale
- PSM25 Neurological deficit scale

Demographic data (age, sex) was also collected.

Statistical Analysis:

Analysis was performed with StataV17. Continuous data underwent Pearson and Spearman correlations. Multivariate regression models were utilized to define predictors of clinical severity.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

Comparisons between groups were carried out with t-tests and ANOVA where the level of significance was $p<0.05$.

Results:

Demographic and baseline features of participants are mentioned in table 1.

Table 1: Demographic and Clinical Characteristics of VE Patients

Parameter	Mean ± SD / n (%)
Age (years)	67.5 ± 8.4
Male	45 (55.6%)
Female	36 (44.4%)
Frontal Lobe Atrophy	75 (92.6%)
Fazekas Grade (Mean)	Male: 2.4, Female: 1.8

Neuroimaging and Clinical Correlations:

Frontal lobe atrophy was present in 92.6% of cases and was the most significant predictor of PSM25 scores ($\beta=0.76$, $p<0.001$), explaining 58% of the variance as shown in table 2. In contrast, white matter lesion burden showed a strong correlation with autonomic dysfunction ($r=0.65$, $p<0.001$).

Table 2: Correlations Between Neuroimaging Markers and Clinical Scales

Variable	Correlation Coefficient (r/β)	p-value	Clinical Scale
Frontal Lobe Atrophy vs PSM25 Score	β=0.76	<0.001	PSM25 Neurological Deficit
White Matter Lesion vs Autonomic Dysfunction	r=0.65	<0.001	Stephen-Porges Scale
Gender (Male vs Female) vs Ischemic Changes	Mean Fazekas: 2.4 vs 1.8	0.02	Fazekas Scale

Gender Differences:

Male patients exhibited significantly higher Fazekas grades compared to females (mean grade 2.4 vs 1.8, $p=0.02$). Additionally, males had higher PSM25 scores, indicating greater neurological impairment (mean 108.3 vs 89.1, $p=0.02$).

Age-Related Findings:

Patients over 70 years demonstrated more parietal lobe involvement, while younger patients had more pronounced frontal atrophy. Combined GCA and Fazekas scores provided superior prediction of clinical outcomes, highlighting the necessity of multimodal imaging assessment.

Discussion:

This study highlights the importance of neuroimaging markers in assessing clinical severity in VE. The significant relationship between frontal atrophy and neurological deficits supports the hypothesis that frontal lobe integrity is crucial for maintaining cognitive and motor functions. White matter lesions primarily impacting autonomic function indicate that cerebrovascular

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

damage in VE may differentially affect brain regions based on underlying pathophysiological mechanisms.

Gender differences in ischemic changes may reflect inherent cerebrovascular vulnerabilities, emphasizing the need for sex-specific diagnostic approaches. Furthermore, the complementary role of GCA and Fazekas scales suggests that visual rating scales can be practically applied to clinical assessments without requiring complex volumetric analysis.

Conclusion:

Our findings demonstrate that visual rating scales, such as the GCA and Fazekas scales, are valuable tools for assessing clinical severity in VE. The strong correlation between frontal lobe atrophy and neurological deficits emphasizes the need for targeted imaging in diagnosing cognitive impairment. Incorporating neuroimaging with comprehensive clinical evaluation can significantly enhance the diagnostic accuracy and management of vascular encephalopathy.

References

1. Botirjonovna M. D., Kizi A. N. A. Assessment of the Cognitive State of Patients with Autonomic Disorders in Chronic Cerebral Ischemia.
2. Brundel, M., et al. (2012). "Cerebral small vessel disease and the risk of dementia: a systematic review and meta-analysis of population-based evidence." Alzheimer's & Dementia, 8(2), 112–121.
3. Chui, H. C., et al. (2006). "Vascular cognitive impairment: a statement for healthcare professionals from the American Heart Association/American Stroke Association." Stroke, 37(6), 1494–1499.
4. Debette, S., & Markus, H. S. (2010). "The clinical importance of white matter hyperintensities on brain magnetic resonance imaging: systematic review and meta-analysis." BMJ, 341, c3666.
5. DeCarli, C., et al. (2005). "Measures of brain morphology and infarction in the framingham heart study: establishing what is normal." Neurobiology of Aging, 26(4), 491–510.
6. DWIVEDI, KALASH. "GENDER DISPARITIES IN BRAIN TUMOR INCIDENCE: INSIGHTS FROM A REPUBLICAN SCIENTIFIC CENTRE OF NEUROSURGERY, UTILIZING RADIOLOGICAL AND STATISTICAL ANALYSIS." Western European Journal of Medicine and Medical Science 2.5 (2024): 106-112.
7. Gorelick, P. B., et al. (2011). "Vascular contributions to cognitive impairment and dementia: a statement for healthcare professionals from the American Heart Association/American Stroke Association." Stroke, 42(9), 2672–2713.
8. Gouw, A. A., et al. (2008). "Heterogeneity of small vessel disease: a systematic review of MRI and histopathology correlations." Journal of Neurology, Neurosurgery & Psychiatry, 79(5), 501–507.
9. Jokinen, H., et al. (2011). "Global burden of small vessel disease-related brain changes on MRI predicts cognitive and functional decline." Stroke, 42(11), 3289–3295.
10. O'Brien, J. T., & Thomas, A. (2015). "Vascular dementia." The Lancet, 386(10004), 1698–1706.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

11. Pantoni, L. (2010). "Cerebral small vessel disease: from pathogenesis and clinical characteristics to therapeutic challenges." The Lancet Neurology, 9(7), 689–701.
12. Prins, N. D., & Scheltens, P. (2015). "White matter hyperintensities, cognitive impairment and dementia: an update." Nature Reviews Neurology, 11(3), 157–165.
13. Smith, E. E., et al. (2017). "Cerebral small vessel disease: a review of clinical, radiological, and pathological perspectives." Journal of Neurology, Neurosurgery & Psychiatry, 88(8), 801–808.
14. Tolibov, D. S., Mirzaeva, D. B., & Abdurasulova, N. A. (2024). ANALYSIS OF THE NEUROPSYCHOLOGICAL STATE OF PATIENTS WITH AUTONOMIC DISORDERS IN CHRONIC CEREBRAL ISCHEMIA (CCI). JOURNAL OF EDUCATION AND SCIENTIFIC MEDICINE, 1(6), 50-55.
15. van Straaten, E. C., et al. (2006). "Impact of white matter hyperintensities scoring method on correlations with clinical data: the LADIS study." Stroke, 37(3), 836–840.
16. Wardlaw, J. M., et al. (2013). "Neuroimaging standards for research into small vessel disease and its contribution to ageing and neurodegeneration." The Lancet Neurology, 12(8), 822–838.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5