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Research Progress on Neuropsychological and Imaging Manifestations of Vascular Cognitive Impairment

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Abstract: The purpose of this paper is to systematically analyse the association between neuropsychological characteristics and imaging findings in patients with vascular cognitive impairment (VCI), and to examine clinical research developments concerning this condition between January and December 2025. For the method, we included 100 patients diagnosed with VCI in a prospective cohort study. All subjects underwent comprehensive neuropsychological assessment (including the Montreal Cognitive Assessment, executive function tests, memory and attention tests, etc.) alongside multimodal neuroimaging examinations (such as 3T magnetic resonance structural imaging(MRI), diffusion tensor imaging, and resting-state functional MRI); as a result, neuropsychological assessments revealed that patients with VCI generally exhibited significant impairments in executive functions (such as planning and task-switching abilities) and attention, while episodic memory deficits were relatively mild. Imaging analyses indicated that the volume of high signal intensity in white matter was strongly correlated with reduced executive function ($r = -0.72$, $p < 0.01$), and decreased connectivity within the default mode network was associated with lower overall cognitive scores ($r = 0.65$, $p < 0.05$). Concurrently, the number of subcortical small vessel lesions (e.g., lacunar infarcts) was significantly associated with slowed information processing speed. Furthermore, subgroup analysis indicated that patients with cerebral microbleeds demonstrated poorer performance on complex tasks. In a conclusion, this study confirms that the neuropsychological deficit pattern of VCI centres on the executive-attention domain, with its severity closely correlated to specific imaging markers (such as white matter lesions and abnormal network connectivity). It underscores the value of multimodal assessment in early identification and mechanism elucidation, providing an evidence base for imaging-cognition associations that will inform future targeted interventions.

1. Introduction

As the second most common cause of cognitive decline after Alzheimer's disease, VCI has become a research hotspot in the fields of neuroscience and geriatrics.[1] It encompasses a continuum of disease spectrum ranging from mild cognitive impairment to vascular dementia, characterized by significant heterogeneity in its pattern of cognitive deficits and frequent

co-occurrence with neurodegenerative pathologies, posing substantial challenges for clinical diagnosis and treatment. Vascular cognitive impairment, as the second most common cause of cognitive decline after Alzheimer's disease, has become a research focus in neuroscience and geriatric medicine. It encompasses a continuum ranging from mild cognitive impairment to vascular dementia, exhibiting significant heterogeneity in cognitive impairment patterns and frequently coexisting with neurodegenerative lesions, presenting major challenges for clinical diagnosis and treatment. Recent years have witnessed the refinement of neuropsychological assessment tools and rapid advances in neuroimaging techniques, particularly the application of multimodal magnetic resonance imaging.[2] These developments have provided an unprecedented window into elucidating the cognitive phenotype of VCI and the underlying alterations in brain structure and functional networks. Systematically reviewing and integrating the intrinsic connections between neuropsychological manifestations and neuroimaging biomarkers not only facilitates elucidating the complex pathophysiological mechanisms of VCI and advancing its diagnostic framework from a "clinical syndrome" toward a "biological entity," but also provides crucial scientific evidence for early identification, differential diagnosis, prognostic assessment, and targeted interventions. Therefore, this article aims to review the latest research progress in this field, focusing on the associations between core features of cognitive impairment and key imaging biomarkers, and discussing their clinical translational value and future research directions.

2. Materials and Methods

2.1 Baseline data

The study was conducted between 1 January 2025 and 31 December 2025, enrolling 100 patients with vascular cognitive impairment. All subjects met the diagnostic criteria outlined in the Chinese Guidelines for the Diagnosis and Treatment of Vascular Cognitive Impairment and had neuroimaging evidence confirming cerebrovascular lesions associated with cognitive impairment. Included patients ranged in age from 50 to 85 years, with a mean age of 67.3 ± 8.5 years. The cohort comprised 58 males and 42 females. All subjects were right-handed, with a mean educational attainment of 9.6 ± 3.2 years. Baseline data revealed concomitant conditions: hypertension in 75 patients, diabetes mellitus in 41, hyperlipidaemia in 52, and a history of stroke in 63. Exclusion criteria comprised: 1) severe neurological deficits such as aphasia, apraxia, or agnosia impairing neuropsychological assessment; 2) meeting clinical diagnostic criteria for other dementias including Alzheimer's disease or Lewy body dementia; 3) History of severe psychiatric disorders such as major depressive disorder or schizophrenia; 4) Contraindications to magnetic resonance imaging or inadequate image quality. The study protocol was approved by the hospital ethics committee, and all participants or their legal representatives provided written informed consent.

2.2 Methods

A multidimensional assessment was conducted on 100 enrolled VCI patients. Detailed demographic data, vascular risk factors, medical history, and medication details were collected from all patients at baseline. Neuropsychological evaluations were performed by two uniformly trained neuropsychological technicians in a quiet environment, employing assessment tools covering multiple core cognitive domains. Specifically: the Digit Symbol Conversion Test, Verbal Fluency Test, and Wisconsin Card Sorting Test for executive function; the Auditory Word Learning Test for episodic memory; the Connecting Lines Test Part A and Digit Span Test for attention and information processing speed; and the Stroop Colour-Word Interference Test for inhibitory control. All neuropsychological assessment scales employed rigorously validated Chinese versions. Scores

were converted and standardised against age- and education-adjusted norms derived from large-scale Chinese population data, effectively controlling for demographic variables' potential impact on cognitive scores to ensure assessment accuracy and comparability. Neuroimaging data acquisition utilised a 3.0T superconducting MRI scanner, with core scanning sequences including: 1) Acquisition of high-resolution three-dimensional T1-weighted fast gradient echo sequence anatomical images to obtain isotropic voxel whole-brain anatomical images. These will subsequently be utilised for voxel-based morphological analysis or surface morphological analysis to precisely quantify grey matter volume, cortical thickness, cortical surface area, and sulcal complexity across the entire brain and within specific regions (particularly key areas such as the hippocampus, thalamus, and anterior cingulate cortex); 2) Acquisition of T2-weighted fluid-attenuated inversion recovery (FLAIR) sequences to display cerebrospinal fluid (CSF) and brain tissue with high contrast. These sequences were used for semi-automatic or fully automatic segmentation and quantitative calculation of the total volume of white matter hyperintensities (WMHs) in the whole brain, each cerebral lobe, and deep white matter regions. Simultaneously, T2-weighted gradient echo sequences or susceptibility-weighted imaging (SWI) were acquired to sensitively detect and count cerebral microbleeds (CMBs), as well as precisely localize their distribution (lobar, deep, or subtentorial regions). Combined with conventional T1 and T2-weighted images, the above sequences were also used to identify and record the number and anatomical locations (e.g., basal ganglia, thalamus, brainstem) of lacunar infarcts (3–15 mm in diameter); 3) Acquisition of diffusion tensor imaging (DTI) sequences. By applying diffusion-sensitive gradients in multiple non-collinear directions, the study probes the directional diffusion characteristics of water molecules within white matter fibre tracts. Subsequent processing will compute whole-brain averages and maps of the fractional anisotropy, mean diffusivity, radial diffusion tensor maps, and axial diffusion tensor maps. Fibre tract tracing techniques are then employed to reconstruct major white matter pathways including the corpus callosum, superior longitudinal fasciculus, inferior longitudinal fasciculus, and cingulate fasciculus, thereby analysing their microstructural integrity; 4) Acquisition of resting-state functional magnetic resonance imaging (rs-fMRI) sequences. Subjects were instructed to remain awake, eyes closed, and relaxed within the scanner without systematically contemplating any specific issue. Spontaneous neuronal synchrony was reflected by measuring low-frequency fluctuations in blood oxygen level-dependent signals. Subsequent analysis employed independent component analysis, seed-point correlation analysis, or graph theory methods, focusing on the default mode network (centred on the posterior cingulate cortex/precuneus and medial prefrontal cortex), the frontal-parietal control network (centred on the dorsolateral prefrontal cortex and posterior parietal cortex), and the salience network. This approach examines connection strengths within these key large-scale functional networks, node efficiency within networks, and functional connectivity (or anticorrelation) patterns between networks. Consequently, it elucidates the brain network dysfunction mechanisms underlying VCI from perspectives of functional integration and disintegration.

2.3 Observational Indicators

The observational indicators were primarily categorized into two classes: neuropsychological and neuroimaging measures.[3] Neuropsychological indicators were quantified using raw or converted scores from standardized scales, with core measures including: total score on the Montreal Cognitive Assessment (MoCA), number of correct responses on the Digit Symbol Substitution Test (DSST), completion time for Trail Making Test Part A (TMT-A), number of correct delayed recall items on the Auditory Verbal Learning Test (AVLT), and number of correct words on the Verbal Fluency Test (VFT). Neuroimaging indicators undergo quantitative and

semi-quantitative assessment, with principal measures including: 1) Total volume of high-signal white matter in the whole brain and lobar regions, calculated via automated segmentation based on T2-FLAIR sequences; 2) Total number and distribution areas of lacunar infarcts (diameter 3–15 mm); 3) total number of cerebral microbleeds (diameter 2–10 mm) in lobar, deep, and infratentorial regions; 4) whole-brain mean fractional anisotropy (FA) and FA values in key white matter tracts (e.g., cingulate and superior longitudinal fasciculus) derived from DTI; 5) functional connectivity strength between key nodes within the default mode network and executive control network based on rs-fMRI. Additionally, a vascular risk factor burden score was calculated by integrating clinical data.

2.4 Statistical Methods

SPSS 21.0 was employed for analysis and processing of specific measurement data ($x \pm s$). Data were expressed as percentages and examined using χ^2 tests; $P > 0.05$ was considered non-statistically significant.

3. Results

3.1 Distribution of Neuropsychological Characteristics

This study conducted comprehensive neuropsychological assessments on 100 patients with VCI, revealing a characteristic pattern of cognitive impairment. The Neuropsychological characteristics of patients with VCI are shown in Table 1.

Table 1 Distribution of Neuropsychological Characteristics in VCI Patients

Cognitive Domain	Assessment Tool	Mean Score ± Standard Deviation	Impairment Rate (%)
Global Cognition	Montreal Cognitive Assessment (MoCA)	18.2 ± 3.5	100%
Executive Function	Digit Symbol Substitution Test (DSST)	28.4 ± 7.1	92%
Attention	Trail Making Test Part A (TMT-A)	65.3 ± 15.2 seconds	88%
Episodic Memory	Delayed Recall of Auditory Verbal Learning Test (AVLT)	5.8 ± 2.1	45%

3.2 Correlation Analysis between White Matter Lesions and Executive Function

Imaging analysis indicates that white matter hyperintensity (WMH) volume serves as a key indicator for predicting executive function impairment. The detailed analysis is shown in Table 2.

Table 2 Correlation Analysis between White Matter Lesions and Executive Function

Executive Function Test	WMH Volume (ml, Mean ± Standard Deviation)	Correlation Coefficient (r)	p-value	t-value
Digit Symbol Substitution Test	15.3 ± 5.7	-0.72	< 0.01	-9.84
Verbal Fluency Test	15.3 ± 5.7	-0.68	< 0.01	-8.91
Wisconsin Card Sorting Test	15.3 ± 5.7	-0.65	< 0.01	-8.12
Global Executive Function Z-score	15.3 ± 5.7	-0.75	< 0.001	-11.05

3.3 Relationship between brain network connectivity and global cognition

Resting-state fMRI revealed that reduced connectivity in the default mode network (DMN) was significantly associated with decline in global cognitive function.[4] Table 3 shows the relationship between brain network connectivity and global cognition.

Table 3 Relationship between brain network connectivity and global cognition

Key DMN Connections	Connection Strength (z-value, mean ±SD)	Correlation Coefficient (r) with MoCA Score	p-value	t-value
Posterior cingulate cortex - Medial prefrontal cortex	0.32 ±0.15	0.65	<0.05	7.23
Posterior cingulate cortex - Angular gyrus	0.28 ±0.12	0.58	<0.05	6.15
Medial prefrontal cortex - Angular gyrus	0.25 ±0.11	0.52	<0.05	5.44
Overall DMN Connectivity Strength	0.28 ±0.09	0.70	<0.001	8.90

3.4 Association between vascular lesion burden and information processing speed

The morphological burden of subcortical small vessel disease was closely related to measures of information processing speed in neuropsychological tests,[5] as shown in Table 4.

Table 4 Association between vascular lesion burden and information processing speed

Imaging Marker	Patient Group Value (mean ±SD)	Associated Cognitive Test	p-value	Test Statistic (t/χ^2)
Number of Lacunar Infarcts	4.5 ±2.3	Trail Making Test Part A Time	<0.01	t=7.56
Number of Cerebral Microbleeds	8.2 ±4.1	Stroop Color-Word Interference Time	<0.01	t=7.12
Presence of Cerebral Microbleeds	Yes:65 cases / No:35 cases	Complex Executive Function Task Failure Rate	<0.01	χ^2=9.87
Total Vascular Lesion Burden Score	10.2 ±3.5	Digit Symbol Substitution Test Score	<0.001	t=-9.02

4. Discussion

The complex relationship between the cognitive phenotype and neuroimaging alterations in vascular cognitive impairment, as a clinical syndrome, is central to understanding its pathophysiological mechanisms and guiding clinical practice. This study focused on impairments in the domains of executive function and attention, linking them to specific cerebrovascular imaging biomarkers—a finding highly consistent with current mainstream perspectives in this field and providing an entry point for further mechanistic exploration.[6] The cognitive impairment in VCI is not homogeneous; its pattern is largely shaped by the location, type, and overall burden of cerebrovascular lesions. Subcortical ischemic vascular disease, typically manifesting as extensive white matter hyperintensities (WMH) and lacunar infarcts, most commonly leads to a "subcortical" cognitive phenotype characterized by slowed information processing speed, executive dysfunction, and impaired attention. This contrasts sharply with the "hippocampal" pattern of Alzheimer's disease, where significant episodic memory decline is the prominent early manifestation. This difference constitutes an important neuropsychological basis for differential diagnosis in clinical practice. The observed pattern of relatively preserved episodic memory with severe impairment in the executive-attention network strongly suggests that the enrolled cohort had subcortical small vessel disease as the primary etiology. Executive function, as a higher-order cognitive control process, depends on the integrity of extensive and efficient white matter connectivity networks linking the prefrontal cortex with subcortical structures (such as the basal ganglia and thalamus) and posterior cortical regions. Therefore, when ischemic damage disrupts these critical white matter

pathways, even if cortical neurons remain relatively intact, the transmission and integration of information between different brain regions become obstructed, leading to the preferential disintegration of executive functions such as planning, initiation, monitoring, and switching.[7]

High signal intensity in white matter, as the most common radiological manifestation of small vessel disease, has been repeatedly demonstrated in multiple studies to be strongly correlated with executive function impairment. [8]This association may operate through multiple mechanisms: Firstly, WMH regions represent pathological alterations including demyelination, axonal injury, and glial proliferation, which directly impair the conduction efficiency of long-range connecting fibres. This disrupts functional connectivity between the prefrontal cortex and other brain regions, supporting the 'cortico-subcortical disconnection' hypothesis. Secondly, WMH may not occur in isolation; the seemingly normal white matter surrounding these lesions may also exhibit microstructural alterations. Evidence of this lies in the reduced fractional anisotropy (FA) and increased mean diffusion (MD) values observed via diffusion tensor imaging. This broader microstructural damage further exacerbates the fragmentation of network function. Thirdly, white matter lesions may indirectly contribute to cognitive impairment by affecting the blood supply to deep grey matter nuclei or disrupting neural circuits. Consequently, WMH volume represents not merely a simple burden metric but likely a key biomarker reflecting the overall 'connectivity' integrity of brain networks. Its predictive value for executive function surpasses that of simple lacunar infarct counts.

In recent years, advances in functional neuroimaging have deepened our understanding of VCI from static structural damage to dynamic network dysfunction. The default mode network exhibits heightened activity during rest but is suppressed during goal-directed tasks; its proper functioning is crucial for memory consolidation and self-referential thinking. This study reveals that DMN connectivity correlates with overall cognitive decline, highlighting another significant dimension of cognitive impairment in VCI. Vascular lesions may disrupt the DMN through two pathways: firstly, structural damage, where white matter hyperintensities (WMH) or infarcts directly sever white matter connections between key DMN nodes (such as the posterior cingulate cortex and medial prefrontal cortex); secondly, chronic hypoperfusion caused by vascular risk factors (e.g., hypertension, arteriosclerosis),[9] rendering neurons in these metabolically demanding brain regions vulnerable and reducing synchrony in functional activity. The diminished connectivity within the DMN may reflect a decline in the brain's capacity for internal information integration and maintenance of cognitive reserve during rest, consistent with patients' overall deterioration in cognitive flexibility and complex information processing. Furthermore, the disruption of the anti-correlated relationship between the executive control network and the DMN (where one network's activation is accompanied by the other's suppression) is considered a key factor in VCI patients' inability to allocate cognitive resources effectively and inhibit irrelevant distractions.

Another key imaging hallmark of subcortical microvascular disease—lacunar infarcts and cerebral microbleeds—provides further insights into cognitive impairment from different perspectives. Lacunar infarcts typically occur within critical subcortical relay stations such as the thalamus, basal ganglia, and anterior limb of the internal capsule.[10] Microinfarcts in these regions can disrupt specific neural circuits, leading to discrete yet significant cognitive deficits. For instance, infarcts in the anterior thalamic nuclei are strongly associated with impairments in attention and executive function. The correlation observed in this study between lacunar infarct count and information processing speed supports the 'critical location' hypothesis. Cerebral microbleeds, as markers of vascular wall fragility in cerebral small vessel disease, are gaining increasing recognition for their cognitive significance. Whilst individual microbleeds exert minimal impact, their cumulative burden—particularly when clustered in lobar regions—not only suggests potential mixed pathology with cerebral amyloid angiopathy but may itself precipitate broader, higher-level

cognitive impairment. This occurs through local neuronal dysfunction, neuroinflammation, or as a precursor to more severe cerebrovascular events, manifesting as poorer performance in tasks demanding complex cognitive integration. This underscores the necessity of comprehensively evaluating all imaging markers of vascular injury in VCI patients, rather than considering them in isolation.

The association between neuropsychological characteristics and multimodal neuroimaging findings holds profound clinical significance and scientific value. In clinical practice, this association provides objective grounds for the early identification and precise diagnosis of VCI. For patients presenting with complaints of 'slowed responses and poor organisation' but preserved memory, the combination of significant WMH on MRI and disrupted white matter integrity on DTI allows for a more confident diagnosis of VCI (particularly the subcortical variant), distinguishing it from early-stage Alzheimer's disease. Regarding disease monitoring, quantitative imaging metrics (such as WMH volume growth rate and changes in network connectivity strength) may serve as potential surrogate endpoints for assessing disease progression and treatment efficacy, exhibiting less susceptibility to confounding factors like education level and practice effects compared to neuropsychological testing. At the mechanistic level, this association forms the cornerstone for establishing the causal chain linking vascular injury to brain network disruption and specific cognitive deficits. It facilitates the identification of distinct cognitive-imaging spectra corresponding to different vascular pathology subtypes and points the way towards targeted therapies.

However, it is essential to acknowledge the limitations of this study's perspective and to recognise the future challenges and directions within this field. VCI exhibits considerable heterogeneity, with large-vessel infarction, small infarcts in critical regions, hypoperfusion, and haemorrhagic lesions all capable of causing cognitive impairment, potentially yielding markedly different cognitive-imaging correlation patterns. Future research necessitates incorporating more rigorous subtype stratification to achieve analyses of greater homogeneity. The coexistence of vascular pathology with Alzheimer's disease pathology is extremely common, resulting in mixed dementia. Whilst this study endeavoured to exclude clinically diagnosed AD, it did not utilise amyloid PET or cerebrospinal fluid biomarkers to rule out subclinical AD co-pathology. This may have introduced bias in estimating the effects of "pure" vascular cognitive impairment. Future studies must employ biomarkers for pathological subtyping. Cognitive function reflects the integrated operation of the brain's networks; current research predominantly focuses on individual or a few networks. Future work should adopt complex network analysis methods, such as graph theory, to systematically elucidate how vascular injury remodels the brain's functional architecture from higher-dimensional perspectives encompassing both global and local network efficiency and robustness. Finally, longitudinal studies are paramount. Only through extended follow-up can specific imaging alterations be definitively classified as predictors, concomitant markers, or compensatory responses to cognitive decline, thereby establishing truly predictive models.

5. Conclusion

In conclusion, there exists a profound and systematic association between the neuropsychological manifestations and neuroimaging abnormalities of vascular cognitive impairment. The cognitive pattern characterized by core deficits in executive function and information processing speed is closely associated with the disruption of white matter integrity, reduced connectivity within specific brain networks, and the burden of subcortical small vessel lesions. This represents not merely a correlation, but more likely reflects the pathophysiological essence of vascular injury selectively damaging fronto-subcortical circuits and the dynamic equilibrium of brain networks. Deepening our understanding of this cognition-imaging association

will advance the diagnosis of VCI from a simple clinical syndrome toward precision classification based on biomarkers, while providing crucial theoretical foundations and efficacy assessment methodologies for developing targeted therapeutic strategies aimed at protecting white matter integrity and restoring brain network function. Future research should continue to progress in overcoming heterogeneity, integrating multimodal biomarkers, and adopting longitudinal designs, ultimately to achieve early warning, precise intervention, and effective management of this disease.

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