Vascular Cognitive Impairment

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Robert Paul and Lauren Salminen

Introduction to Vascular Cognitive Impairment Nomenclature

Approximately 800,000 individuals experience a cerebrovascular event on an annual basis in the USA. Worldwide, the number is close to 15 million. Unfortunately, the vast majority of individuals who experience a stroke will develop cognitive symptoms secondary to neuronal injury. There is no cure for vascular cognitive impairment (VCI) or intervention capable of fully arresting the underlying disease process. As such, VCI represents a major global health concern.

VCI refers to the full spectrum of cognitive dysfunction associated with cerebrovascular disease (CVD). The recently updated Diagnostic and Statistical Manual (DSM)-5 [1] criteria for major neurocognitive disorder does not require a primary memory disorder (see Table 30.1). The evolution away from an Alzheimer's disease (AD)-centric diagnostic system is still relatively new, and the nomenclature has yet to be fully adopted in the scientific literature. Rather, the majority of research refers to VCI as vascular dementia (VaD), vascular cognitive impairment-dementia (VCID), subcortical ischemic disease (SID), and subcortical ischemic vascular dementia (SIVD). VCI and VaD are often used to describe cognitive impairment regardless of whether the vascular injury involved cortical or subcortical brain regions. By contrast, SID and SIVD refer specifically to cognitive impairment secondary to ischemia in the white matter or subcortical gray matter. To simplify the terminology for this chapter, we refer herein to the full spectrum of cognitive impairment as VCI and vascular injury limited to the subcortical regions as SIVD. Mild VCI is used to describe cognitive impairment without disruption in activities of daily living (ADLs), and VaD is used to refer to cognitive impairment with disruption to ADLs. When appropriate, reference to DSM 5 nomenclature is noted.

Risk Factors for Vascular Cognitive Impairment

The risk of occlusive or hemorrhagic stroke exists across the lifespan, but the incidence is inherently tied to advanced age. Samples of community-dwelling elders reveal CVD on structural neuroimaging in the majority of individuals [2].

R. Paul (✉)
Missouri Institute of Mental Health,
St. Louis, MO, USA

Department of Psychological Sciences, Division of Behavioral Neurosciences, 1 University Blvd,
St. Louis, MO, USA
e-mail: paulro@umsl.edu

L. Salminen
Imaging Genetics Center, Stevens Neuroimaging and Informatics Institute, University of Southern California, Marina del Rey, CA, USA

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Table 30.1 DSM-5 Criteria for Major Neurocognitive Disorder [1]

A.	Significant cognitive decline[a] from a previous level of performance in one or more cognitive domains
	Learning and memory
	Language
	Executive function
	Complex attention
	Perceptual motor
	Social cognition
B.	The cognitive deficits interfere with independence in everyday activities. At a minimum, assistance should be required with complex instrumental activities of daily living, such as paying bills or managing medications
C.	The cognitive deficits do not occur exclusively in the context of a delirium
D.	The cognitive deficits are not better explained by another mental disorder (e.g., major depressive disorder, schizophrenia)

[a]Evidence of decline is based on concern of the individual; a knowledgeable informant, or the clinician; and a substantial impairment in cognitive performance, preferably documented by standardized neuropsychological testing or, in its absence, another quantified clinical assessment.

Endothelial dysfunction, reduced vascular plasticity, atherosclerosis, hypertension, diabetes mellitus, and hyperlipidemia are important risk factors for CVD. Heart disease is especially critical, as emboli often emerge from the heart as a consequence of underlying CVD, thus increasing the risk of occlusive stroke.

Lifestyle factors are implicated in the development of age-related vascular pathophysiology. Smoking, obesity, lack of exercise, and poor nutrition separately, and collectively, correlate with cardiovascular risk factors. Lifestyle variables do not fully account for the development of CVD, but as modifiable risks, they represent prime therapeutic targets. Medications (e.g., antihypertensives) and behavioral interventions (e.g., smoking cessation, weight loss) reduce the risk of recurrent stroke, but no intervention completely halts the progression of cardiovascular or CVD with advanced age. Further, recent work suggests that the optimal therapeutic window may be many years before the expression of clinical symptoms. For example, blood pressure, atrial fibrillation, and prior myocardial infarction predicted the incidence of stroke in 47–55 year-old males followed for an average of 11 years [3]. These findings highlight the importance of managing risk factors long before the onset of clinical symptoms.

Neuroimaging Markers of Vascular Cognitive Impairment

Common neuroimaging markers of SIVD include lacunes and subcortical hyperintensities (SH). Neuroimaging scans readily demarcate lacunes as small cerebrospinal filled cavities in the white matter and SH as bright white areas. These markers are best visualized on T2-weighted fluid-attenuated inversion recovery (FLAIR) scans because this sequence suppresses the signal generated by cerebrospinal fluid in the ventricles. The enhanced visual contrast is akin to turning off the glow of city lights in order to visualize stellar constellations in the night sky (Fig. 30.1).

The presence of SH or lacunes on neuroimaging does not equate to VCI. Nearly all individuals display these vascular markers at some point in older adulthood, and many individuals with clear ischemic changes on imaging perform within the normal range on cognitive testing. However, it is important to note that "normal" performance here is based on the age of the participant, not optimal function. Erroneously described as "silent" infarcts, these ischemic imaging markers of CVD almost certainly represent a substrate of age-related cognitive decline. The total volume of SH and lacunar burden correlates only modestly with cognitive performance, and debate continues on the relative importance of lacunar count, volume, and location of the lesion. The lack of correspondence is at least partially accounted for by the limited sensitivity of available imaging techniques. Studies using diffusion tensor imaging (DTI) reveal significant alternations in the microstructural integrity of "normal appearing white matter," particularly in regions adjacent to ischemic infarcts [6]. Further, vascular-related abnormalities (e.g., hemosiderin deposits) visible using 7 Tesla MRI are not visible at the more common 3 Tesla strength [7]. These studies highlight that vascular lesions

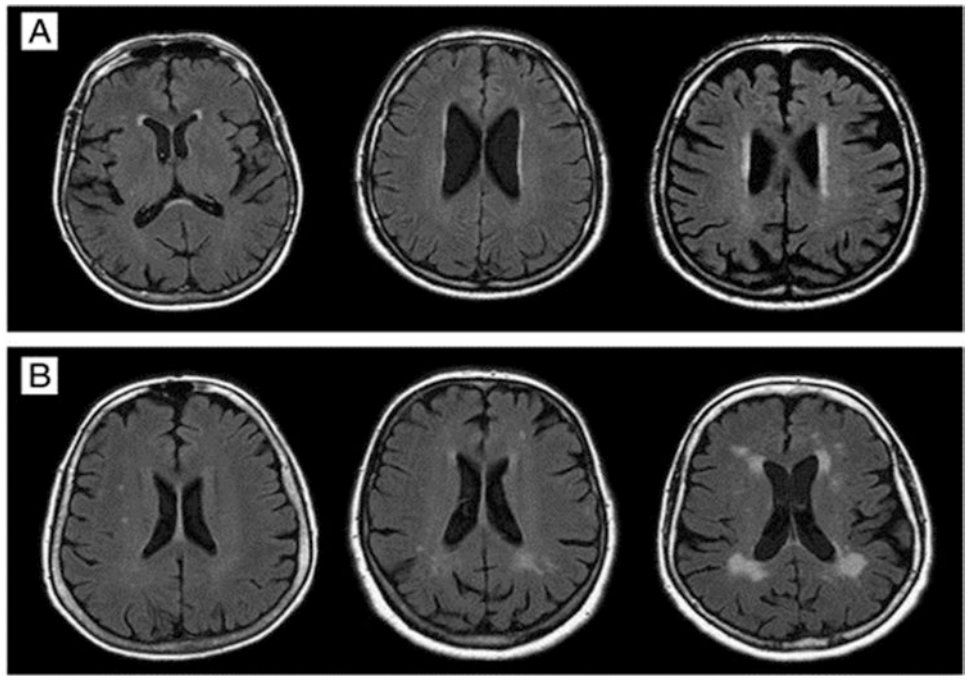

Fig. 30.1 White matter lesions on a T2-weighted FLAIR MRI scan. Images are extracted from work by Freudenberger and colleagues [4] (**a**) Example of typical progression of age-related periventricular white matter lesions in a "healthy" older adult. From left to right: periventricular caps, lining, and halo. (**b**) Typical progression of deep white matter lesions in an adult with cerebrovascular disease (CVD). From left to right: punctate, patchy and early confluent, and confluent [5]

on MRI may contribute to the expression of cognitive impairment, but many vascular-related neuroimaging abnormalities go undetected in routine clinical practice.

The absence of a standardized rating system in radiology further complicates the diagnostic landscape of CVD. Previous efforts to define thresholds based on the volume of infarcted tissue (e.g., 25% of the white matter, or lacunes greater than 10 cm) proved unhelpful. New research methods using data driven models such as machine learning/deep learning have potential to create more accurate predictive algorithms capable of directing personalized patient care. Clinical brain science has moved at a glacial pace in the development and application of these models, but momentum is building in the field of VCI. Recent work reveals high diagnostic accuracy for cardiovascular disease [8], and nearly perfect accuracy in the prediction of acute changes in cerebral blood volume and hemodynamic decompensation [9]. Advances in this space will emerge rapidly as multiple groups, including ours, harness the strength of these algorithms to build predictive models of VCI that integrate neuropsychological, demographic, and neuroimaging input features.

Neuropsychological Phenotype of Vascular Cognitive Impairment

The potential for any branch of the cerebrovascular network to become occluded or to hemorrhage creates a massive range of vulnerable brain regions. Stroke location and stroke volume have long been revered as the primary determinants of symptoms. A stroke in the nondominant association cortex might produce minimal symptoms, whereas a stroke of the same volume in the thalamus or the hippocampus is capable of causing profound impairment.

The neuropsychological phenotype of VCI is directly related to both location and size of the underlying infarct(s). The most common site of vascular damage involves the middle cerebral artery (MCA), which tracts to the lateral surface of the brain after completing a near 90-degree turn. This course correction creates an anatomical vulnerability for embolic occlusion, resulting in temporary (transient ischemic attack; TIA) or permanent (stroke) occlusion of the lumen. The MCA perfuses a large geographic region of the lateral surface as well as deep subcortical brain regions via the lenticulostriate arteries.

When the larger vessels perfusing cortical regions are primarily involved, individuals experience a sudden onset of aphasia, agnosia, paralysis, etc. (see Table 30.2), with "stepwise" decline in function over time. Neuropsychologists working in a rehabilitation setting are most likely to encounter these cases, yet they do not represent the most common subtype of VCI. The most common form of VCI involves subcortical ischemia and strategic subcortical stroke without prominent cortical involvement. These cases have an insidious onset, with a slow and progressive clinical course. Neuropsychologists working in outpatient settings are more likely to see these cases in order to assist the clinical team with differential diagnosis and treatment recommendations. This form of VCI (commonly referred to as SIVD) represents a diagnostic challenge because the history of symptom onset and progression mirrors that of AD, and neuroimaging cannot establish causal links to the cognitive symptoms. Here the potential for misdiagnosis is high. The neuropsychologist is uniquely positioned to guide the clinical process by following a theoretically driven integration of neuropsychological data, patient history, and neuroimaging results to render a highly probable diagnosis of VCI.

The neuropsychological profile of SIVD is typical of a "subcortical profile" [10]. This term does not fully integrate modern perspectives of whole-brain networks and cognition, yet the concept holds value in test interpretation and diagnostic etiologies. Neuropsychological testing of SIVD reveals poor performance in multiple aspects of executive function, verbal retrieval, learning efficiency, and psychomotor speed [10–14]. Lexical fluency is more impaired than semantic fluency (the opposite of AD), but patients frequently perform below average on both due to impaired response fluency. Symptoms of apathy and depression are prevalent, the former possibly due to damage within the subcortical

Table 30.2 Common Stroke Sites and Corresponding Clinical Features

Large vessel stroke site	**Clinical features**
Middle cerebral artery (MCA)	Hemiplegia, aphasia, homonymous hemianopia, contralateral hemianesthesia
Anterior cerebral artery (ACA)	Paraplegia, incontinence, abulia, executive dysfunction, personality changes
Posterior cerebral artery (PCA)	Homonymous hemianopia, alexia with or without agraphia, visual agnosias, color anomia, Balint's syndrome, prosopagnosia
Basilar artery	Ophthalmoplegia, motor deficits, ataxia, dysmetria, vertigo, nausea/vomiting, coma, cranial nerve IV palsy, contralateral decreased sensation to pain and temperature, Horner's syndrome, ipsilateral deafness
Lacunar stroke site	**Syndrome**
Posterior limb of internal capsule (PLIC), basis pontis, cerebral peduncle	Pure motor: Contralateral hemiparesis
PLIC, deep white matter, thalamus	Sensorimotor: Contralateral weakness and numbness
Posterior thalamus	Pure sensory: Loss of contralateral sensory function
PLIC, basis pontis, thalamus	Ataxic hemiparesis: Unilateral weakness disproportionate to contralesional hemiparesis and ataxia
Anterior limb/genu of internal capsule, basis pontis	Dysarthria-clumsy hand: Weakness of contralateral hand and decline in fine motor abilities, slurred speech
Subthalamic nucleus	Hemiballismus/hemichorea: Contralesional limb flailing, dyskinesia

mesolimbic system/tracts and the latter more commonly linked to functional impairments (especially language) following thalamic or MCA infarcts [15, 16].

Significant impairment in functional status due to cognitive impairment equates to a diagnosis of dementia (or major cognitive disorder: DSM 5). VaD most commonly results from extensive white matter ischemia, one or more strategic subcortical strokes, large vessel cortical infarcts, or combination involving the mantle and the subcortex. More rare, VaD can also occur from lesions in the cerebellum. In one memorable case evaluated by our team, VaD developed soon after a cerebellar stroke that damaged the occipitofrontal fasciculus. The patient exhibited profound executive dysfunction in addition to cerebellar-mediated motor abnormalities. The neuropsychological phenotype of VaD is similar to VCI with prominent executive and psychomotor speed deficits, but a global pattern is also possible when patients present with both large cortical infarctions and extensive subcortical ischemia [17]. Physical signs of extrapyramidal and pyramidal disruption are more common in cases of severe vascular disease. Similarly, individuals who meet criteria for VaD may present with imbalance or incontinence that mimics normal pressure hydrocephalus (NPH).

Clinical Course of Vascular Cognitive Impairment

Up to 80% of individuals with first-ever stroke exhibit cognitive impairment [18], and one third of individuals who experience a large vessel stroke meet criteria for VaD within 12 months [19]. Survivors of first-ever stroke are at increased risk for additional vascular events. Prospective studies reveal that nearly 50% of individuals with early stage VCI progress to dementia within a 5-year period [20]. Even more alarming, a recent study revealed that MRI perfusion changes following a TIA predicts subsequent stroke [21]. These studies suggest that the natural history of VCI is characterized by a linear progression from CVD without cognitive complications to dementia.

Diagnosis of Vascular Cognitive Impairment

The most common diagnostic systems for VCI in the research literature include the State of California Alzheimer's Disease Diagnostic and Treatment Centers (ADDTC) criteria and the National Institute of Neurological Disorders and Stroke-Association Internationale pour la Recherche et L'Enseignement en Neurosciences (NINDS-AIREN) criteria [22]. The ADDTC and NINDS-AIREN systems require neuroimaging evidence of CVD for a probable diagnosis. Cognitive impairment is also required, though the NINDS-AIREN criteria mandate evidence of episodic memory dysfunction, whereas the ADDTC criteria are more flexible. As noted previously, the DSM 5 criteria for major neurocognitive disorder no longer require a primary memory disorder, but the criteria have not yet been integrated into the VCI literature.

Differential Diagnosis of Vascular Cognitive Impairment

Both patients and referring parties express specific concern about the diagnostic differentiation between VCI and AD. Some groups have argued this differentiation is not possible given that postmortem data reveal a high frequency of mixed dementia and a low frequency of pure VaD. However, in vivo imaging studies demonstrate that most individuals with SIVD show limited or no cortical binding of ^{11}C-Pittsburgh compound B on positron emission tomography [23]. As such, while autopsy studies suggest that both AD and vascular disease are related substrates to dementia, it is very likely that more pure cases of each condition exist in the early stages of disease, years before the development of comorbid pathology. Described previously in this chapter, the frequency of SIVD means that the clinical course will not aid the diagnostic process. However, impairment in executive function and psychomotor abilities without evidence of amnestic memory loss (percent of information lost from last learning

trial to retention trial or discrimination memory on a recognition trial) argues against AD. Additional diagnostic considerations include frontotemporal dementias (FTDs) and NPH. Patients with FTD often present at clinic with acute changes in personality or isolated aphasia early in the course of the disease and before age 65. By contrast, major personality changes are not common in VCI, and aphasia is more typical of advanced VaD or strategic subcortical infarct. NPH presents a unique challenge because the cognitive phenotype mirrors that seen in VCI. NPH usually includes more prominent gait disturbance and urinary symptoms (urgency, frequency, or incontinence). However, older males with cardiovascular disease, prostate hypertrophy, and arthritis report the same cluster of symptoms, further emphasizing the importance of a carefully conducted patient interview.

Clinical Evaluation: Interview and History

The interview provides a prime opportunity to evaluate receptive and expressive language skills during conversation. Further, interview questions targeting clinically relevant demographic and medical histories allow the clinician an organic opportunity to assess whether memory failures resemble "tip of the tongue" retrieval deficits or amnestic loss of information. Reflexively we query the patient and the family about the clinical course, but this rarely proves valuable. When a history of sudden onset and stepwise decline is present, the diagnosis is nearly already known by the referral source, and the purpose of the evaluation is focused on characterizing strengths and weaknesses.

Neuropsychological Assessment

Proper neuropsychological evaluation of VCI requires a combination of breadth and depth. Screening tools, such as the Mini-Mental State Exam (MMSE), lack sensitivity to mild VCI [24, 25]. The Montreal Cognitive Assessment (MoCA) provides greater coverage of executive processes, but the scope remains inadequate. More comprehensive options are summarized in Table 30.3. It is important that the selected battery includes comprehensive tests of executive function (working memory, response

Table 30.3 Neuropsychological Protocol Recommendations

Domain	Harmonization 30-min protocol[a]	Harmonization 60-min protocol[a]	Additional considerations
Global	MMSE	MMSE	MoCA
Executive function/ activation	Animal fluency Letter fluency Digit Symbol Coding	Animal fluency Letter fluency Digit Symbol Coding Trail Making	Letter-Number Sequencing Stroop test
Psychomotor speed			Grooved Pegboard Test
Language		Boston Naming Test	
Attention/reaction time		Simple and choice reaction time	Digit Span
Visuospatial skill		Rey Complex Figure Test	
Memory	Hopkins Verbal Learning test—Revised	Hopkins Verbal Learning Test—Revised or California Verbal Learning Test-2	Brief Visual Memory Test—Revised
Neuropsychiatric/ Depressive symptoms	Mood questionnaire (BDI II, CES-D)	Neuropsychiatric Inventory Mood questionnaire (BDI II, CES-D)	

[a]Adapted from NINDS and Canadian Stroke Harmonization Network Protocol Recommendations (Hachinski et al.) [24]
[b]Additional considerations
Abbreviations: *MMSE* Mini Mental State Examination, *MoCA* Montreal Cognitive Assessment, *BDI II* Beck Depression Inventory II, *CES-D* Center for Epidemiological Studies Depression

inhibition, cognitive flexibility, planning, and organization), motor, learning, retention, and recognition discrimination, and language. Poor test coverage in these domains will undermine the process to differentiate cortical and subcortical cognitive phenotypes.

Neuroimaging Corroboration

Neuropsychologists are not routinely trained to interpret neuroimaging scans, so the degree of vascular burden must be extracted from the radiology report. This is unfortunate because these reports do not rely on a standardized system to rate severity. A binary approach may be the only option when expert input is unavailable. Within this framework, vascular disease of any severity (even "age-related vascular disease") would be supportive of VCI, whereas the complete absence of vascular disease would argue against VCI. This "winner take all" strategy is admittedly over simplified and only recommended when trained expert input is unavailable.

Clinical Case Example

Here we describe an example of a typical VCI assessment with baseline and 12-month follow-up results. Neuroimaging input is provided verbatim. The case was modified to remove personal identification.

Background and History

Mrs. Smith (fictional name) is a 71-year-old, right-handed, married female who was referred for neuropsychological evaluation by her neurologist secondary to cognitive difficulties identified by the patient. During the interview, Mrs. Smith reported a history of TIAs, "stroke," and memory loss that began approximately 5 years prior to the evaluation. She reported a TIA-like event a year prior followed by a "stroke." She was unable to provide further details regarding the reported stroke, but she did note that she elected to stop driving 2 years ago after developing tingling sensations in both hands.

Mrs. Smith described a 2-year history of short-term memory loss, characterized by repeating herself and difficulty remembering names of people she recently met; she reported no difficulty recalling familiar names. She independently manages her medications (list provided) and all other basic and instrumental ADLs. She denied hallucinations, fluctuating symptoms, urinary incontinence/urgency/frequency, significant visuospatial abnormalities, and changes in personality.

Mrs. Smith's medical history includes high cholesterol, high blood pressure, TIAs, and adult-onset seizures. Psychiatric history is unremarkable. She completed college with a degree in business education, and she worked as a high school business teacher for many years. Mrs. Smith and her husband now live in an independent senior community where they enjoy an active lifestyle. Her husband was unavailable to participate in the interview. She does not smoke or drink alcohol. She reported no family history of dementia. A brain MRI report dated 3 weeks prior was remarkable for "periventricular white matter low attenuation related to chronic small vessel ischemia consistent with generalized age-related cerebral volume loss."

There was no evidence of abnormal gait, posture, physical asymmetries, tremor, or rigidity. During the interview, her conversational speech was fluent and goal-directed. She exhibited appropriate prosody, and there was no evidence of paraphasias. Receptive speech was intact, and there was no clear difficulty comprehending simple or complex material. She was very friendly and cooperative. Her mood was euthymic, and she remained engaged throughout the evaluation.

Baseline Neuropsychological Evaluation

Baseline neuropsychological data are presented in Table 30.4. Mrs. Smith recalled 23 words across 3 learning trials on a verbal learning measure. After a brief delay, she recalled 6 out of 12 target words (low-average performance). Her performance was less strong on a recognition trial, as she tended to endorse semantically related foils. She appeared confused on this aspect of the test compared to the free recall portions. On a test of learning and memory of prose passages, she exhibited intact learning and retention of information, and she performed one standard deviation above average on the delayed trial, suggesting no rapid loss of information. Further, her recognition memory was adequate. Performance on a test of visual learning and memory was significantly impaired in terms of learning and retention, though her recognition memory was intact.

On a measure of semantic fluency, she performed within expectations for her age, naming 17 animals within 1 min. By contrast, on the letter fluency test, she produced only 20 words across all 3 letter cues in 3 min, resulting in below-average performance. She correctly named only 47/60 items on the Boston Naming Test (below-average performance) and incorrect items included both

Table 30.4 Baseline and 12-month follow-up neuropsychological data for case example

Test	Baseline (raw)	12-month (raw)	Outcome
Attention			
WAIS-III Digit Span	14	10	Decline
Executive function			
Trail Making Test, B	Timed out[a]	Unable to complete due to confusion[b]	Decline
Clock drawing	Unable to draw accurately	Concrete setting	Stable
Psychomotor speed			
Trail Making Test, A	77[a]	141[b]	Decline
WAIS-III Symbol Search	12[a]	11[a]	Stable
WAIS-III Digit Symbol	25[a]	27[a]	Stable
Pegs, dominant	145	155[b]	Decline
Pegs, nondominant	123	133[a]	Decline
Activation/language			
Semantic fluency	17	17	Stable
Letter fluency	20[a]	16[b]	Decline
Boston Naming Test	47[a]	48[a]	Stable
Visuospatial			
Rey Figure copy	15.5[a]	18.5[a]	Decline
Memory			
HVLT-R learning	23	17[a]	Decline
HVLT-R delay	6	6	Stable
HVLT-R recognition	6[b]	9	Improve
BVMT-R learning	10[b]	16[a]	Improve
BVMT-R delay	3[b]	7	Improve
BVMT-R recognition	100%	100%	Stable
WMS-III, LM I	40	36	Stable
WMS-III, LM II	26	24	Stable
Mood			
BDI II	4	3	Stable

[a]Mild impairment
[b]Moderate impairment. Abbreviations not previously defined: *WAIS-III* Wechsler Adult Intelligence Scale – 3rd ed., *WMS-III LM* Wechsler Memory Scale 3rd ed. Logical Memory

high- and low-frequency words. Her copy reconstruction of a Rey Complex Figure was impaired. She did not appear to grasp the gestalt of the design, and her placement of details was poorly planned and organized.

Mrs. Smith's ability to repeat a string of digits in forward sequence was intact. Her performance on a test of visual scanning and psychomotor speed was moderately impaired. Though she successfully completed the practice trial of the Trail Making Test, Part B, she became very confused on the test trial, which she was unable to complete. This suggests significant problems with cognitive flexibility. Mild to moderate difficulties were noted on tests of psychomotor speed and visual scanning. When asked to construct a clock and set the hands to a specified time, she drew a clock with the numbers in the reverse order on two separate efforts. When a clock was drawn for her and she was then asked to set the hands of the clock to a specified time, she was unable to complete the task. Psychomotor speed on the Grooved Pegboard Test was moderately impaired for the dominant hand, but performance on the nondominant hand was stronger, with a score in the borderline normal range. Mrs. Smith's total score on the Beck Depression Inventory II was not suggestive of current depressive symptoms.

Based on the neuropsychological test results, clinical history, and neuroimaging data, it appeared that Mrs. Smith meets DSM 5 criteria for minor neurocognitive disorder, vascular origin.

12–Month Follow–Up Evaluation

Mrs. Smith returned for a follow-up examination after 12 months. Performances declined on tests of executive function and psychomotor speed. Her performance also declined on a test of learning efficiency, but she improved on visual learning, and there was no change in retention on either test. Overall, the lack of decline in retention or other major cognitive domain argues against a new diagnostic formulation. Her MRI report dated 11 months after the first MRI revealed "mild to moderate diffuse punctuate T2 and FLAIR hyperintensities within the left and right frontal parietal periventricular subcortical white matter and more confluent increased T2 and FLAIR signal within the left and right parietal periventricular white matter suggesting mild to moderate small vessel ischemic changes. There may be mild diffuse cortical atrophy with prominent sulci bilateral cerebral hemispheres."

Case Summary

The case example includes common issues in the evaluation of VCI. First, the patient's age and medical history raise the probability of a vascular origin. Second, the neuropsychological pattern was typical of a "subcortical" phenotype, with impairment in learning, executive function, and motor skills. The patient performed poorly on the recognition trial of learning test, but this was not accompanied by a loss of information. Finally, her brain MRI reports were congruent with her history and neuropsychological pattern of VCI.

Clinical Pearls

- The clinical course of VCI can be abrupt with stepwise decline or slow, insidious, and progressive.
- The pattern of neuropsychological deficits associated with vascular burden is dependent on the location of damage. Executive impairment is usually dominant but not universal, and is not necessary for diagnosis.
- For differential diagnosis, impairment in executive function and psychomotor abilities without evidence of amnestic memory loss (percent of information lost from last learning trial to retention trial, or discrimination memory on a recognition trial) argues against AD. Additional diagnostic considerations include frontotemporal dementias (FTDs) and NPH.
- Integration of neuroimaging results is mandatory to diagnose VCI.
- The progression of VCI can be influenced by healthy lifestyle interventions.

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