

Cognitive assessments for the early diagnosis of dementia after stroke

Noor Kamal Al-Qazzaz^{1,2}
Sawal Hamid Ali¹
Siti Anom Ahmad³
Shabiul Islam⁴

¹Department of Electrical, Electronic and Systems Engineering, Faculty of Engineering and Built Environment, Universiti Kebangsaan Malaysia (UKM), Bangi, Selangor, Malaysia; ²Department of Biomedical Engineering, Al-Khwarizmi College of Engineering, Baghdad University, Baghdad, Iraq; ³Department of Electrical and Electronic Engineering, Faculty of Engineering, Universiti Putra Malaysia (UPM), Serdang, Selangor, Malaysia; ⁴Institute of Microengineering and Nanoelectronics (IMEN), UKM, Bangi, Selangor, Malaysia

Abstract: The early detection of poststroke dementia (PSD) is important for medical practitioners to customize patient treatment programs based on cognitive consequences and disease severity progression. The aim is to diagnose and detect brain degenerative disorders as early as possible to help stroke survivors obtain early treatment benefits before significant mental impairment occurs. Neuropsychological assessments are widely used to assess cognitive decline following a stroke diagnosis. This study reviews the function of the available neuropsychological assessments in the early detection of PSD, particularly vascular dementia (VaD). The review starts from cognitive impairment and dementia prevalence, followed by PSD types and the cognitive spectrum. Finally, the most usable neuropsychological assessments to detect VaD were identified. This study was performed through a PubMed and ScienceDirect database search spanning the last 10 years with the following keywords: “post-stroke”; “dementia”; “neuropsychological”; and “assessments”. This study focuses on assessing VaD patients on the basis of their stroke risk factors and cognitive function within the first 3 months after stroke onset. The search strategy yielded 535 articles. After application of inclusion and exclusion criteria, only five articles were considered. A manual search was performed and yielded 14 articles. Twelve articles were included in the study design and seven articles were associated with early dementia detection. This review may provide a means to identify the role of neuropsychological assessments as early PSD detection tests.

Keywords: poststroke dementia, vascular dementia, neuropsychological assessments, early dementia detection

Introduction

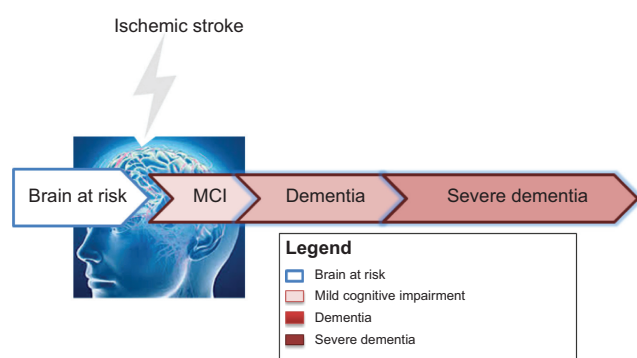
Stroke, a cerebrovascular disease (CVD), is one of the major causes of quality of life reduction through physical disabilities; the second most important risk factor for cognitive impairment and dementia, with a frequency ranging from 16%–32%; and the third cause of mortality after heart diseases and cancer.^{1,2} However, aging and possession of a specific form of gene that links mild cognitive impairment (MCI) to Alzheimer’s disease (AD) are the most common causes of significant cognitive impairment.³ Poststroke dementia (PSD) includes all dementia types that may occur after stroke. Stroke survivors more prone to develop dementia, of which 20%–25% are diagnosed with vascular dementia (VaD), approximately 30% with degenerative dementia (particularly AD), and 10%–15% with a combination of AD and VaD (mixed dementia).^{4–6} Approximately 1%–4% of elderly people aged 65 years suffer from VaD, and the prevalence doubles every 5–10 years after this age.^{5,7} However, cognitive impairment and dementia following a stroke diagnosis may involve multiple functions, and the most affected domains are attention, executive function, memory, visuospatial ability, and language, as shown in Table 1.^{1,8,9}

Correspondence: Noor Kamal Al-Qazzaz
Department of Electrical, Electronic and Systems Engineering, Faculty of Engineering and Built Environment, Universiti Kebangsaan Malaysia, UKM, Bangi, Selangor 43600, Malaysia
Email noorbmemsc@siswa.ukm.edu.my

Table 1 Cognition domain and function

Domain	Function related to domain
Attention	Focusing, sustain, selective, alternative, divided, shifting
Executive function	Planning, organizing thoughts, inhibition, control
Memory	Recall and recognition of visual and verbal information
Visuospatial ability	Visual search, drawing, concentration
Language	Expressive (Broca's aphasia), receptive (Wernicke's aphasia)

VaD is the second most common type of dementia after AD and is considered to be the primary cause of clinical deficits with burden on cognition in vascular cognitive impairment (VCI) resulting from CVD and ischemic or hemorrhagic brain injury.^{10,11} VCI describes the VaD cognitive spectrum in the cognitive domain starting from MCI and ending with severe dementia. The VCI spectrum is defined after the point where the brain's cognitive function remains intact; at risk, this period is called cognitive impairment no dementia.^{12–15} MCI in cognitive function is greater than expected with respect to the age and education level of patients, but it is unrelated to daily life activities.^{8,14} Clinically, MCI is the transitional stage between early normal cognition and late severe dementia, and patients with MCI exhibit a high potential to develop dementia.^{5,16} The most frequently observed symptoms of MCI are limited to memory retrieval; however, the daily life activities are unaffected.¹⁷ The stage after MCI, which is termed the dementia stage, will reduce long-term memory (particularly episodic memory) and will eventually result in executive function impairment.^{18–20} VaD or severe dementia is the endpoint of the VCI spectrum that affects 10% of single-attack stroke patients in the subsequent months after ischemic stroke onset and 30% after recurrent ischemic stroke.²¹ Figure 1 shows the cognitive consequences that predispose stroke individuals in the VCI spectrum.

**Figure 1** VCI spectrum and dementia.

Abbreviations: VCI, vascular cognitive impairment; MCI, mild cognitive impairment.

Poststroke cognitive impairment patterns change considerably during the whole VCI spectrum. Thus, the cognitive defects following a stroke may be mapped by assessing the cognitive domain. However, poststroke prominent impairments are associated with one or more cognitive functions and thus require the evaluation of the decline in cognitive function, including attention, memory, language, and orientation.¹ The neuropsychological assessments that are currently available for cognitive impairment and dementia have been developed by the National Institute of Neurological Disorders and Stroke and Association Internationale pour la Recherche et l'Enseignement en Neurosciences (NINDS–AIREN) for VaD.²² In clinical practice, the most usable test to evaluate the severity of dementia (but not limited to this condition) is to run the diagnosis based on the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV),²³ Mini-Mental State Examination (MMSE),²⁴ Montreal Cognitive Assessment (MoCA),²⁵ and Addenbrooke's Cognitive Examination Revised (ACE-R).^{26–29} The severity of cognitive symptoms could be assessed using the Clinical Dementia Rating (CDR)³⁰ and Geriatric Depression Scale (GDS).³¹ These neuropsychological assessments have been used to assess different cognitive functions to promote neuropsychological diagnosis and the understanding of cognitive impairment following a stroke diagnosis.

This review will focus on VaD as a common cause of PSD. This work aims to identify the risk factors for cognitive impairment and the effect of such factors on cognitive function. A secondary aim is to assess various neuropsychological assessments used for the early detection of dementia in stroke patients.

Methods

Studies have been performed for PubMed and ScienceDirect. Articles were dated from 2003–2014. The search was based on the following keywords: “post-stroke”; “dementia”; “neuropsychological”; and “assessments”. Figure 2 shows the three steps that were used to select eligible articles. In step 1, the search was limited to English-language and case-control or cohort studies with patients aged 50 years and above and excluded the articles found to be related to brain injury, kidney or pulmonary diseases, heart bypass surgery, aortic valve implementation, and left ventricle assist devices. In addition to the articles related to AD and psychiatric diseases or other dementia types, systematic review, pilot studies, and meta-analysis papers were the primary articles found in this search. Step 2 was based on the following three diagnostic criteria: 1) patient risk factors associated with PSD; 2) description and information

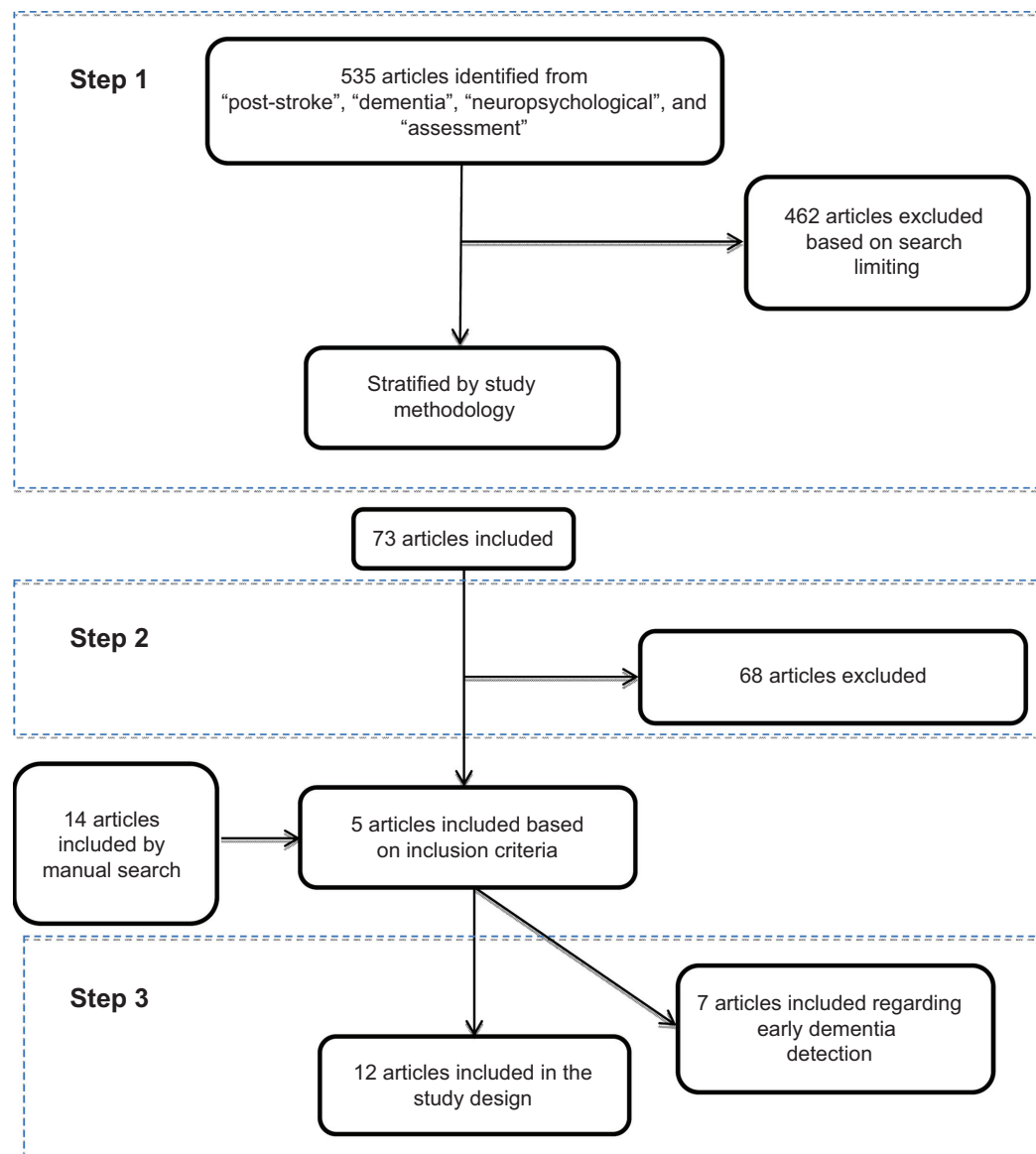


Figure 2 Flowchart of the literature search and selection process.

about the poststroke cognitive impairment/dysfunction in cognitive function among survivors that had been tested by neuropsychological assessments and evaluated using DSM or other dementia evaluation scales; and 3) PSD evaluation that started from a baseline of first stroke up to 3 months poststroke using neuropsychological assessments. In step 3, due to the limitations imposed by the previous search that focused on the first 3 months after stroke, a manual search of the cited references for related articles identified additional related articles for this research.

Results

Initially, the literature search yielded 535 articles. After applying the search criteria (as in step 1), the number of

relevant articles decreased to 73. Step 2 reduced the number of relevant articles to five, and with the manual search, an additional 14 articles were included to yield 19 articles for review.

Association between risk factors and cognitive impairment

Table 2 shows an overview of the demographic and clinical characteristics of patients on the basis of dementia severity. The relationship between individual risk factors and post-stroke VaD development are shown. Several risk factors such as CVD, hypertension, heart disease, and hyperlipidemia, are associated with stroke and VaD development.³² Diabetes mellitus was another risk factor that was highly associated

Table 2 Studies investigating the associations between stroke, cognitive impairments, and neuropsychological assessments within 3 months of stroke onset

Author (year)	Stroke characteristics (patients' demography)	Risk factors	Cognitive impairment associated with PSD	Neuropsychological tests and cognitive decline evaluation
Tham et al ³² (2002)	N=252 TIA (N=140 intact; age: 56.7 years; N=102 CIND, age: 65.1 years; N=10, D, age: 60.4 years) M/F =66/34	Hypertension, previous stroke, hyperlipidemia, diabetes, ischemic heart disease	Attention, language, verbal memory, visual memory, visuoconstruction, visuomotor speed	Tests: MMSE; WMS-R; CDT; WAIS; digital cancellation task; digital symbol modalities test; maze task; picture recall; mBNT; auditory detection test. Evaluation: subject classified by DSM-IV; MMSE score: 27.4 for intact, 23.5 for CI, 17.0 for D
Auchus et al ³⁷ (2002)	N=125 VaD; N=12 SSID, age: 69 years; M/F =58/41	Hypertension, myocardial infarction, diabetes mellitus	Attention, language (naming and verbal fluency), verbal memory (recall and recognition), visual memory (recall and recognition), and visuoconstruction	Tests: attention, language (naming and verbal fluency); verbal memory (recall and recognition); visual memory (recall and recognition); and visuoconstruction; together with the MMSE and screening for depression. Evaluation: VaD identified by NINDS-AIREN. Subject classified by DSM-III-R. MMSE score: 17.5 (range: 9–24)
Garrett et al ⁴¹ (2004)	N=26 VaD, age: 77.1 years, M/F =66/34; N=18, VCIND, age: 78.4 years, M/F =56/29. Individual risk factors of CVD: age: 67.4 years, M/F =62/63; N=25, controls, age: 76.5 years, M/F =44/43	CVD	Visual memory, verbal fluency, verbal list-learning and memory abilities	Tests: TMT part A and B; CVLT; BNT; COWAT; MMSE; and CDR. Evaluation: subject classified by DSM-III; VaD identified by NINDS-AIREN. MMSE score: 28.2 for early control, 28.9 for individual at risk for CVD, 26.3 for VCIND, 26 for VaD
Mok ⁴² (2004)	N=75 patients, age: 71 years, M/F =41.5/58.5; N=42 control, age: 69.6 years, M/F =47/48	Diabetes, previous stroke, hyperlipidemia, heart disease, smoking, alcohol use	Memory, thinking (decision making or answering questions), orientation (in time, place, or person), aphasia, and speech comprehension	Tests: CDR; MMSE; MDRS; IQCODE. Evaluation: daily living activity assessed by BI, IADL. MMSE score: 27.7 for control; 24.8 for patients
Graham et al ⁴⁰ (2004)	N=19 subcortical VaD, age: 71.2 years, M/F =73.6/26; N=19 AD, age: 68.9 years, M/F =47.3/52.6; N=19 controls, age: 68.1 years, M/F =47.3/52.6	CVD	Episodic and semantic memory, executive/attentional functioning, and visuospatial and perceptual skills	Tests: MMSE, ACE-R; CDR; CBI. Evaluation: VaD identified by NINDS-AIREN. MMSE score: 25.3 for VaD patients, 24.2 for AD patients
Sachdev et al ³⁹ (2004)	N=123 patients, age: 72 years, M/F =39.7/40.7; N=78 control, age: 70.6 years, M/F =43.9/44.9	Hypertension, diabetes, AF, CAD hyperlipidemia, smoking, alcohol use	Attention, global memory, verbal memory, visual memory, executive, abstraction, working memory, language, visuoconstruction	Tests: MMSE; WMS-R; BNT, WAIS-R; TMT part A and B; HAM-D; IQCODE. Evaluation: daily living activity assessed by ADL. MMSE score: 28.4 for control, 27.6 for patients
Firbank et al ⁶ (2007)	N=79 patients (N=65 ND, age: 80.1 years, M/F =50/50; N=14 D, age: 80 years, M/F =64/35)	CVD	Medial temporal lobe atrophy associated with cognitive decline and brain atrophy, increasing the suggested role for AD	Tests: CAMCOG-R; MMSE; Sheffield language screening test. Evaluation: subject classified by DSM-IV. Daily living activity assessed by Bristol activity; stroke assessed by OCSF. MMSE score: 26 for ND, 25.1 for D

Mok et al ³³ (2008)	N=61 patients, age: 68.7 years, M/F =53/47; N=35 control, age: 68.9 years, M/F =37.1/62.9	Hypertension, diabetes mellitus, hyperlipidemia, heart disease, smoking, alcohol use	Verbal learning and memory, orientation, language, praxis and visuospatial functions, verbal fluency, and verbal, motor, and graphomotor programming	Test: MMSE. Evaluation: subject classified by CDR; stroke assessed by NIHSS. Daily living activity assessed by BI, MDRS, Lawton's IADL index, AD assessed by ADAS-cog. MMSE score: 27.6 for control, 25.7 for patients Tests: IQCODE; MMSE; digital forward; WMS-R; digital backward; BNT; BDAE; animal naming; figure recognition test; Raven's matrices; grooved pegs domain; symbol digital oral score; CLTR. Evaluation: subject classified by DSM-IV. MMSE score: 28.7 for NCI, 25.8 for CI Tests: MMSE; WAIS. Evaluation: stroke assessed by NIHSS. MMSE score: 28.5 for patients, 29.15 controls
Stebbins et al ³⁸ (2008)	N=91 patients (N=51 NCI, age: 63.1 years, M/F =52/47; N=40 cognitive impairment, age: 67.4 years, M/F =47/52)	CVD	Orientation, attention, working memory, language, visuospatial, psychomotor, memory	
Jaillard et al ³⁶ (2009)	N=177 patients, age: 50.6, M/F =62.3/36.7; N=81 control, age: 51.9 years, M/F =58/42	Hypertension, diabetes mellitus, admission glycemia, low-density lipoprotein cholesterol, smoking, alcohol use, homocysteine	Short-term memory, episodic memory, executive function, working memory	
Khedr et al ³⁴ (2009)	N=81 patients (N=17 D, age: 65.5±9.2 years, M/F =64/35; N=64 ND, age: 56.9±5.3 years, M/F =67/32)	Hypertension, homocysteine level, smoking, CVD	Orientation, repetition of words, attention, calculation, recall of words, language, visual construction	Tests: IQCODE; MMSE; WMS-R; CASI. Evaluation: subject classified by DSM-IV. MMSE score at baseline: 25.58 for patients; 25.72 for control. MMSE score after 3 months: 22.77 for patients, 26.04 for control
Kandiah et al ³⁵ (2011)	N=97 NCI, age: 53 years, M/F =71/29; N=48 CI, age: 61 years, M/F =40/60	Hypertension, diabetes, hypercholesterolemia	Executive function, memory, visuospatial	Tests: MMSE, MoCA; FAB; IQCODE. Evaluation: subject classified by DSM-IV-TR, CDR. Stroke assessed by Rankin score. MMSE score: 29.1 for NCI, 26.4 for CI

Notes: M/F values were represented as percentages. Age is given as the average age in years.

Abbreviations: PSD, poststroke dementia; N, number of patients; TIA, transient ischemic attack; CIND, cognitive impairment no dementia; D, dementia; M, male; F, female; MMSE, Mini-Mental State Examination; WMS-R, Wechsler Memory Scale-Revised; CDT, Clock Drawing Test; WAIS, Wechsler Adult Intelligence Scale; mBNT, modified Boston Naming Test; DSM-IV, *Diagnostic and Statistical Manual of Mental Disorders Revised, fourth edition*; CI, cognitively impaired; VaD, vascular dementia; SSID, strategic single infarct dementia; NINDS-AIREN, National Institute of Neurologic Disorders and Stroke and the Association Internationale pour la Recherche et l'Enseignement en Neurosciences; DSM-III-R, *Diagnostic and Statistical Manual of Mental Disorders, Revised, third edition*; VCIND, vascular cognitive impairment no dementia; CVD, cerebrovascular disease; TMT, Trail Making Test; CVLT, California Verbal Learning Test; BNT, Boston Naming Test; COWAT, Control Oral Word Association Test; CDR, Clinical Dementia Rating Scale; DSM-III, *Diagnostic and Statistical Manual of Mental Disorders, third edition*; MDRS, Mattis Dementia Rating Scale; IQCODE, Informant Questionnaire on Cognitive Decline of Elderly; NIHSS, National Institutes of Health Stroke Scale; BI, Barthel Index; IADL, instrumental activity of daily living; ACE-R, Addenbrooke's Cognitive Examination; CBI, Cambridge Behavioral Inventory; AD, Alzheimer's disease; AF, atrial fibrillation; CAD, coronary artery disease; WAIS-R, Wechsler Adult Intelligence Scale-Revised; Ham-D, Hamilton Depression Rating Scale; ADL, activity of daily living; ND, no dementia; CAMCOG-R, Cambridge Assessment of Mental Disorders in Elderly, Revised; OOSP, Oxfordshire Community Stroke Project; ADAS-cog, Alzheimer's Disease Assessment Scale-Cognitive Subtest; NCI, noncognitive impairment; BDAE, Boston Diagnostic Aphasia Examination; CLTR, Control Learning and Enhancing Recall; CASI, Cognitive Abilities Screening Instrument; MoCA, Montreal Cognitive Assessment; FAB, Frontal Assessment Battery; DSM-IV-TR, *Diagnostic and Statistical Manual of Mental Disorders, fourth edition, text revision*.

with VaD development. Other factors were cigarette smoking and alcohol intake.

Demographic determinants, such as age and sex (shown in Table 2), are nonmodifiable risk factors for stroke patients. Compared to the control subjects, the risk of dementia was higher with age and may have resulted in VaD. Age was also correlated with a diagnosis of vascular diseases. However, no significant relationship was observed between dementia development and patient sex.

Table 2 also shows that CVD was the major risk factor for cognitive impairment and VaD following stroke onset. Modifiable risk factors such as hypertension, heart disease, and diabetes mellitus were the highest risk factors for stroke and dementia, followed by other factors that might predispose subjects to stroke and even VaD.^{33–38} Moreover, an association between a risk factor and the decline or even loss of one or more cognitive functions (for instance, attention and executive function) were significantly impaired in the studies referenced.^{33,35–41} This was followed by short-term memory, working memory, and verbal and visual memories, which were significantly impaired in all studies. The decline in cognitive functions was assessed using neuropsychological assessments such as the MMSE, MoCA, ACE-R, and other assessments within the third month of stroke onset.

Focusing on the MMSE score to differentiate dementia severity among patients provided a clear picture of the

decreasing trend of the score among stroke patients. Patients were evaluated for VaD using NINDS–AIREN, and the subjects were classified using the DSM-IV or daily living activity assessments.

Function of neuropsychological assessments in the early diagnosis of poststroke VaD

Within the VCI spectrum that ranges from MCI to VaD, efforts have been made to assess patients in the early dementia stage to predict optimal medical treatment for patients. Table 2 shows that at the early stage (MCI), attention and executive function were affected, which was later followed by the loss of other functions in the memory domain.

Table 3 shows the poststroke VaD that occurred within the first 3 months of the first stroke onset. All studies used one or more dementia evaluation assessments, such as the DSM-III or DSM-IV, CDR and Mattis Dementia Rating Scale. All studies used the MMSE, in addition to other neuropsychological assessments, to assess patients after stroke diagnosis and dementia evaluation. The evaluation score of MMSE for normal individuals was above 24, but Table 2 shows that the MMSE score for stroke patients ranged from 17–28. This result indicates that the MMSE cannot accurately measure the cognitive impairment of poststroke patients. MMSE is

Table 3 Studies presenting the limitations in poststroke memory assessment

Study/year	Stroke characteristics of patients	Neuropsychological tests
Cao et al ⁴⁴ (2007)	N=40 patients, age: 37.8 years, M/F =37/62; N=40 control, age: 38.8 years, M/F =40/60	MMSE, AVLT, DST, Token test, linguistic tasks, SVF, BNT, Cori's block-tapping board, similarities, RPM, SDS
Yoshida et al ⁴⁵ (2011)	N=126 patients (N=62 control, age: 66.7±10.1 years, M/F =48/51.6; N=13 MCI, age: 62.7±12.3 years, M/F =46/53.8; N=65 AD, age: 74.1±7.8 years, M/F =26/73.8; N=24 FTD, age: 61.8±9.1 years, M/F =54/45; N=11 DLB, age: 75.5±5 years, M/F =27/72; N=28 VaD, age: 73.4±9.8 years, M/F =69/30)	ACE, MMSE, CDR
Bour et al ⁴³ (2010)	N=194 patients, age: 68.3 years, M/F =55.2/44.8	MMSE, MAAS, CAMCOG, GIT, SCWT, CST, AVLT
Dong et al ⁴⁷ (2010)	N=100 patients, age: 61.2±11.3 years, M/F =62/38	MMSE, MoCA, IQCODE
Pendlebury ⁴⁹ (2012)	N=91 ND patients, age: 73.4 years, M/F =56/44; N=39 MCI patients	MoCA, ACE-R, MMSE, TMT parts A and B, SDMT, BNT, Rey–Osterrieth complex figure, HVLT, GDS
Sikaroodi et al ⁴⁸ (2012)	N=50 patients, age: 51.8±13.18 years, M/F =32/68	MMSE, MoCA
Raimondi et al ⁴⁶ (2012)	N=83 patients (N=26 control, age: 73.23 years, M/F =50/50; N=25 AD, age: 77.64 years, M/F =40/60; N=32 VaD, age: 75.59 years, M/F =50/50)	CDR, BDI-II, MMSE, ACE-R

Notes: M/F values were represented as percentages. Age is given as the average age in years.

Abbreviations: N, number of patients; M, male; F, female; MMSE, Mini-Mental State Examination; AVLT, Auditory–Verbal Learning Test; DST, Digital Span Test; SVF, Semantic Verbal Fluency; BNT, Boston Naming Test; RPM, Raven's Progressive Matrices; SDS, Self-Rating Depression Scale; MCI, mild cognitive impairment; AD, Alzheimer's disease; FTD, frontotemporal dementia; DLB, dementia Lewy body; VaD, vascular dementia; ACE, Addenbrooke's Cognitive Examination; CDR, Clinical Dementia Rating Scale; MAAS, Maastricht aging study; CAMCOG, Cambridge Assessment of Mental Disorders in Elderly; GIT, Groninger Intelligence Test; SCWT, Stroop Color Word Test; CST, Concept Shifting Test; MoCA, Montreal Cognitive Assessment; IQCODE, Informant Questionnaire on Cognitive Decline of Elderly; ND, no dementia; ACE-R, Addenbrooke's Cognitive Examination, Revised; TMT, Trail Making Test; SDMT, Symbol Digit Modalities Test; HVLT, Hopkins Verbal Learning Test; GDS, Geriatric Depression Scale; BDI-II, Beck Depression Inventory II.

insensitive to complex cognitive deficits. All patients in the selected articles were evaluated using the MMSE and other neuropsychological assessments to detect cognitive abnormalities after ischemic stroke.

Bour et al⁴⁴ and Cao et al⁴⁵ reported that MMSE alone is inaccurate for the detection of neuropsychological deficits in young people. The authors recommended an MMSE cutoff of 27 for multiple sclerosis. Yoshida et al⁴⁶ and Raimondi et al⁴⁷ used Addenbrooke's Cognitive Examination (ACE) with the MMSE to detect early dementia, and the authors showed that ACE was more accurate than MMSE. Dong et al⁴⁸ and Sikaroodi et al⁴⁹ found that MoCA was superior to MMSE in detecting early dementia. Pendlebury⁵⁰ found that MoCA and ACE-R exhibited good sensitivity and specificity for MCI, and both were feasible as short tests useful for routine clinical practice.

Discussion

Stroke is a CVD that can increase the risk for cognitive impairment and lead to dementia. PSD, particularly VaD, may affect up to 21% of stroke survivors after the third month of stroke onset. This study shows that stroke patient demographics, including age and sex, were associated with cognitive impairment and dementia.

Cognitive impairment increased with age because of the decrease in vessel and cerebral artery inflow. This decrease in cerebral blood flow to the brain may damage the brain and cause cognitive decline. Moreover, the risk factors associated with stroke, including CVD, hypertension, and diabetes mellitus, can be linked to VaD. For example, hypertension can reduce blood flow to the brain, which can lead to VaD and heart diseases, such as atrial fibrillation. This can also reduce cardiac output leading to cerebral hypoperfusion and myocardial infarction and is associated with cognitive decline.

A heavy smoking habit is also a stroke risk factor that can cause decline in memory function. For instance, Atluri et al⁵¹ analyzed the molecular mechanisms behind the increase the risk of human immunodeficiency virus (HIV)-associated neurocognitive disorder in HIV-infected heavy cigarette smokers. Impairment in cognitive function, such as attention, executive function, language, short-term memory, working memory, and visual and verbal memories, could also be associated with VCI spectrum stages.

Several neuropsychological assessments are used to test the mental ability of patients following a stroke diagnosis. The MMSE test is widely used for dementia. However, this test only emphasizes language and constructional items.

The MMSE cannot predict long-term cognitive decline. Researchers have used the MoCA and ACE-R assessments to detect dementia because both exhibit good sensitivity, particularly at the MCI stage.

This study has some limitations. First, only 19 articles were reviewed because studies on VaD patients, stroke risk factors, and cognitive function within the first 3 months after stroke onset are limited. Moreover, the sample sizes were small and there is a need for additional studies for the early detection and prediction of PSD. Besides, the file drawer problems for all studies in this area of research, many studies may be reviewed but never published, and these studies may have different results from the other studies that have significant findings and are typically published. Despite these drawbacks, this review may provide a means to identify the role of neuropsychological assessments for the early detection of PSD. Finally, cognitive impairments are widely assessed by neuropsychological assessments, but until recently, no specific neuropsychological assessments to evaluate and predict PSD that included memory loss existed.

Future research on the roles of neuropsychological assessments is required to predict and evaluate poststroke VaD, including the evaluation of memory impairment in the early stages. This will reveal subtle changes that might define indicators for the early detection of dementia that will help medical doctors and clinicians in planning and providing a more reliable prediction of the course of the disease. In addition, this will aid in the development of the optimal therapeutic program to provide dementia patients with additional years of a higher quality of life.

Conclusion

This article reviewed the roles of the available neuropsychological assessments in detecting poststroke cognitive impairment and dementia, as well as the affected domain, within the first 3 months of a stroke diagnosis. No specific assessment was suitable for the whole spectrum of the VCI, but several assessment candidates can be suggested for the early detection of the disease. MMSE, MoCA, and ACE-R were among the most useful assessments that would help in the clinical evaluation of cognitive impairment following a stroke. Given their differences in sensitivity and specificity, these tests can be used together to assess different types of patient demographics and risk factors. Combining several assessment methods for neuropsychological testing will be useful and help health care practitioners to provide a suitable treatment program for and to help avoid the cognitive decline of stroke survivors.

Disclosure

The authors report no conflicts of interest in this work.

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