Vascular Cognitive Impairment and Dementia (VCID): Diagnosis and Disease Mechanisms

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Historical Development & Terminology

Diagnostic Criteria

(From Roman, 2009)

Year	Source	Characteristics
1952	APA: DSM-I	Chronic brain syndrome associated with cerebral arteriosclerosis, embolism, hemorrhage, hypertension, and other chronic cardiovascular disease
1974-75	Hachinski	Large-vessel MID (vol loss> 50-100 ml): Ischemic score
1980	DSM-III	MID
1983	Loeb & Gandolfo	Large-vessel MID
1985	Román	Small-vessel: Lacunes & Binswanger
1986	Erkinjuntti	Cortical v Subcortical
1987	DSM-IIIR	VaD, MID, Lacunar state
1987	Román	Senile dementia of the Binswanger type
1991	NINDS/AIREN	International Workshop on Vascular Dementia
1992	Chui ADDTC	Ischemic VaD
1993	Román NINDS/AIREN	Comprehensive vascular etiologies; imaging required
1993	WHO: ICD-10	Acute onset (FO1.0), MID (FO1.1), Sub-cortical (FO1.2), Mixed C-SC (FO1.3), Other (FO1.8), Unspecified (FO1.9)
1994	DSM-IV	Executive dysfunction

Diagnostic Criteria

Year	*Source*	*Characteristics*
1995	Hachinski	**Vascular cognitive impairment** (VCI) refers to the entire spectrum of vascular brain pathologies that contribute to any degree of cognitive impairment, ranging from subjective cognitive decline to dementia1
2000	Erkinjuntti	Subcortical VaD (NINDS/AIREN-derived)
2004	Sachdev	Vascular Cognitive Disorder
2011	AHA/ASA	Consensus statement on VCI
2013	DSM-5	mild and major Vascular Neurocognitive Disorder
2014	Sachdev et al	VASCOG criteria
2018	Skobrot et al	Vascular Impairment of Cognition Classification Consensus Study (VICCCS)
2018	ICD-11	Dementia due to Cerebrovascular Disease

Which term should we use?

- Vascular Dementia (VaD)
- Vascular Cognitive Impairment (VCI)
- Vascular Cognitive Disorders (VCD)
    - Mild VCD
    - Vascular Dementia (or Major VCD)
- Vascular Cognitive Impairment and Dementia (VCID)

Four commonly used criteria for VaD

1. National Institute of Neurological Disorders and Stroke-Association Internationale pour la Recherché et l'Enseignement en Neurosciences (NINDS-AIREN) criteria (Roman et al 1993)
2. State of California Alzheimer's Disease Diagnostic and Treatment Centers (ADDTC) criteria (Chui et al, 1992)
3. DSM-IV criteria
4. ICD-10 criteria

Hachinski Ischaemic Score

VaD =>7

AD=<4

• Abrupt onset	2	+
• Stepwise deterioration	1	
• Fluctuating course	2	
• Nocturnal confusion	1	
• Relative preservation of personality	1	
• Depression	1	
• Somatic complaints	1	
• Emotional incontinence	1	
• History of hypertension	1	
• History of strokes	2	+
• Evidence of associated arteriosclerosis	1	
• Focal neurological symptoms	2	+
• Focal neurological signs	2	+

Sensitivity & specificity 70-80%

HOW WELL DO THEY PERFORM?

Applied Criteria	Concordance	κ
NINDS-AIREN/DSM-IV	35.7%	0.08
NINDS-AIREN/ICD-10 and DSM-III	85.0%	0.87
NINDS-AIREN/ADDTC	33.3%	0.05
DSM-IV/ICD-10 and DSM-III	39.8%	0.09
DSM-IV/ADDTC	87.3%	0.37
ICD-10 and DSM-III/ADDTC	33.3%	−0.03

Only 31/ 107 patients fulfilled all the criteria for VaD applied.

Pohjasvaara et al. Stroke. 2000;31:2952-2957

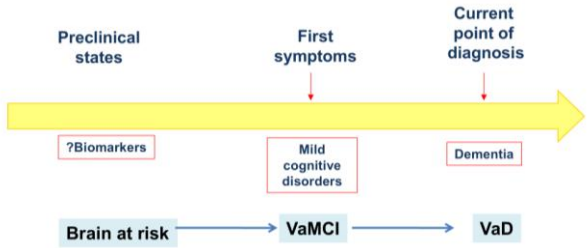
Level of agreement (Cohen's kappa) between pairs of vascular dementia classifications

	DSM-5	VICCCS	NINDS	ADDTC	DSM-IV
VASCOG	1.00	1.00	0.63	0.55	0.47
DSM-5		1.00	0.62	0.54	0.49
VICCCS			0.65	0.54	0.49
NINDS				0.74	0.88
ADDTC					0.60

Sachdev PS et al. Eur J Neurol 2019

The Challenges

1. The problem with dementia
   - Alzheimerisation
   - Late in course
2. The diversity of pathology
3. Are biomarkers essential?
   - Neuroimaging
   - Fluid
   - Retinal
4. What about risk factors?
5. How does one operationalize the criteria?



VASCOG-2-WSO criteria

Revised Diagnostic Criteria for Vascular Cognitive Impairment and Dementia—The VasCog-2-WSO Criteria

Sachdev PS et al. The VasCog-2-WSO Criteria Consortium

JAMA Neurol. doi:10.1001/jamaneurol.2025.3242

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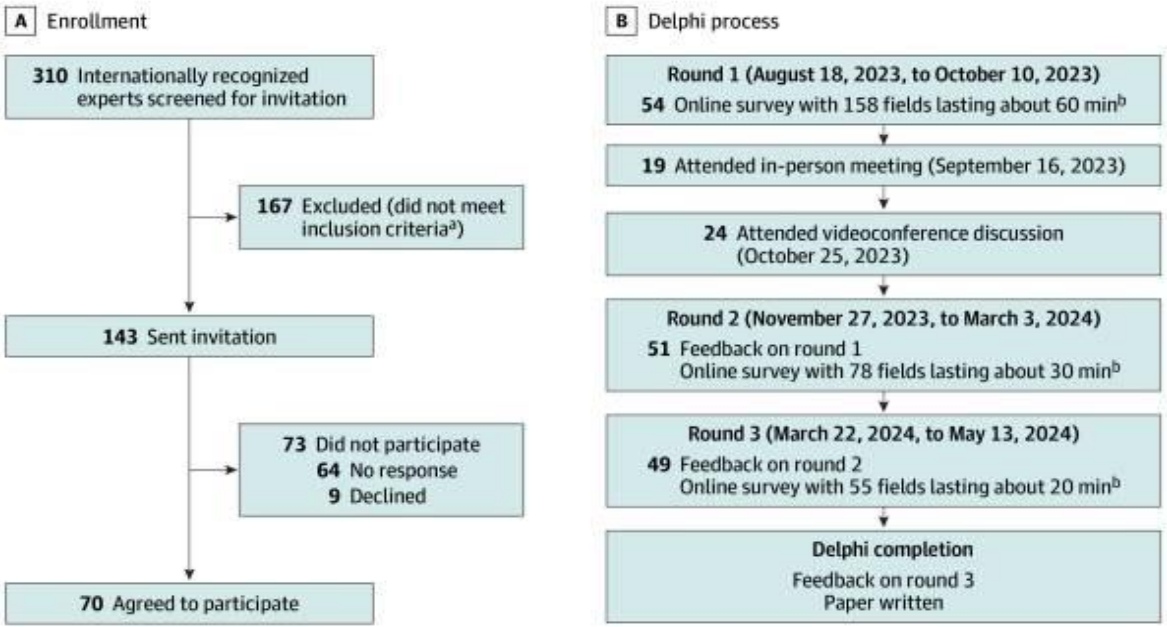
VasCog-1 Criteria; Sachdev PS et al, 2014

Methodology



Consensus on any statement was defined as agreement or disagreement of greater than or equal to 75% and non-consensus as less than 75% through multiple iterative rounds.

VASCOG-2-WSO criteria

Steps in the diagnostic process:

1. Establish a syndromal diagnosis
2. Evidence for a predominantly vascular aetiology
3. Level of certainty
4. Presence of alternative/co-morbid pathologies
5. Associated neuropsychiatric symptoms

Terminology

Vascular Cognitive Impairment and Dementia (VCID)



Vascular Mild Cognitive Impairment (VaMCI)

Vascular Dementia (VaD)

DEMENTIA (MAJOR NEUROCOGNITIVE DISORDER)

(BOTH A AND B ARE NECESSARY)

- **A. Evidence of significant cognitive decline from a previous level of performance in one or more of the domains outlined above based on:**
  1. **Concerns of the patient, a knowledgeable informant or the clinician that there has been a significant decline in cognitive function; AND**
  2. **A clear impairment in cognitive performance preferably documented by standardized neuropsychological testing or, in its absence, another quantified clinical assessment.**
- **B. The cognitive deficits are sufficient to interfere with independence (i.e., requiring assistance at a minimum with instrumental activities of daily living [i.e., more complex tasks such as paying bills or managing medications]).**

***Guidance*:** *Test performance typically falls ≥2 SDs below appropriate norms (or below the third percentile) and/or below an estimate of the person's premorbid cognitive functioning when a formal neuropsychological assessment is available, or an equivalent level as judged by the clinician including from appropriate score cutoffs and/or decline measured from cognitive screening tests.*

Mild Cognitive Impairment (MCI)

(Mild Neurocognitive Disorder)

A. Evidence of modest cognitive decline from a previous level of performance in one or more of the domains outlined above based on:

1. **Concerns of the individual, a knowledgeable informant, or the clinician that there has been a mild decline in cognitive function; and**
2. **A modest impairment in cognitive performance preferably documented by standardized neuropsychological testing or, in its absence, another quantified clinical assessment.**

B. The cognitive deficits are insufficient to interfere with capacity for independence in everyday activities, i.e., complex instrumental activities of daily living such as paying bills or managing medications are preserved. However, greater effort, compensatory strategies, or accommodation may be required to maintain independence.

Guidance: *The test performance is typically in the range between 1 and 2 SDs below appropriate norms (or between the 3rd and 16th percentiles) and/or below an estimate of the person's premorbid cognitive functioning when a formal neuropsychological assessment is available, or an equivalent level as judged by the clinician including from appropriate score cutoffs and/or decline measured from cognitive screening tests.*

Consensus Cognitive Domains Assessed in Vascular Cognitive Impairment and Dementia

Cognitive domains	Subdomains/description
1. Attention and processing speed	Sustained attention, divided attention, selective attention, processing speed
2. Executive function	Planning, decision-making, working memory, responding to feedback, inhibition, flexibility
3. Learning and memory	Free recall, cued recall, recognition memory, semantic and autobiographical long-term memory, implicit learning
4. Language	Object naming, word finding, fluency, grammar and syntax, receptive language
5. Perceptual-motor function	Visual perception, visuoconstructional reasoning, perceptual-motor coordination
6. Social cognition	Recognition of emotions, theory of mind, insight

Part B. Evidence for predominantly vascular etiology of cognitive impairment.

	(A) One of the following clinical features (A.1. or A.2.)
1	**The onset of the cognitive deficits is temporally related to ≥ 1 clinical strokes. The evidence of stroke is one of the following**
	a) Documented history of a stroke, with cognitive decline temporally associated with the event
	b) Physical signs consistent with stroke *Guidance: Physical signs may include but are not limited to hemiparesis, lower facial weakness, Babinski sign, pronator drift, sensory deficit, visual field defect, pseudobulbar syndrome—supranuclear weakness of muscles of face, tongue and pharynx, spastic dysarthria, swallowing difficulties—and cerebellar signs eg, limb ataxia. Covert neuroimaging evidence (eg, brain infarct or hemorrhage) of cerebrovascular disease may be present in the absence of clinical history of stroke and should be taken into consideration, as for physical signs of stroke.*
2	**If no history or signs of stroke, cognitive decline has more gradual onset and course, typically predominant in some combination of attention and processing speed, and/or executive functioning.** *Guidance: Memory impairment, where present, typically involves inefficient encoding and/or retrieval (vs consolidation and storage in Alzheimer disease).* *Guidance (supportive features): Personality and mood changes, particularly apathy, depressive symptoms and emotional lability, and gait-balance disorders are common in vascular cognitive impairment and dementia.*

(B) Presence of significant neuroimaging MRI (preferable) or CT evidence of cerebrovascular disease (at least one of the following)

1) Multiple infarcts or a single extensive or strategically placed infarct

2) Multiple lacunes (≥2) outside the brainstem; one lacune may be sufficient if strategically placed or in combination with extensive white matter hyperintensities.

3) White matter hyperintensities, particularly if they are extensive and confluent.

Guidance: Extensive and confluent may correspond to a Fazekas score of 2 or 3; or by region: periventricular Fazekas = 3 and/or deep Fazekas = 2 or 3. (Fazekas F et al, 1987)

4) Multiple intracerebral hemorrhages; one may be sufficient if large and/or in a lobar location or otherwise strategically placed

Guidance (supportive features for cerebrovascular burden, not sufficient on their own without meeting neuroimaging criteria)

a. *Cerebral microbleeds and cortical superficial siderosis*
b. *Abnormalities that meet the criteria of cerebral amyloid angiopathy (eg, Boston criteria version 2.023)*

Guidance (all imaging evidence): The Standards for Reporting Vascular Changes on Neuroimaging 2 (STRIVE-2)definitions and guidelines of measurement of cerebrovascular lesions and changes should be adopted.

Strategic areas for post-stroke CI
- Left angular gyrus
- Left basal ganglia
- Thalamus
- Hippocampus

(C) Features that May Suggest an Alternative or Additional Etiology

Clinical features
a) Insidious early onset of cognitive, perceptual and motor symptoms in the absence of corresponding focal vascular lesions (infarct or small vessel disease) on brain imaging or history of vascular events.
b) Early and prominent movement disorder suggestive of Lewy body disease or other alpha-synucleinopathy or other non-vascular movement disorder
c) Features strongly suggestive of another primary neurological disorder sufficient to explain the cognitive impairment.
Neuroimaging
a) Absent or minimal cerebrovascular lesions on CT or MRI
Biomarkers
a) The presence of biomarkers of Alzheimer's disease, dementia with Lewy bodies, or other disease related to cognitive decline.

VasCog-2-WSO criteria (contd)

Level of Certainty

1. Probable (a or b are required)

a. Clinical criteria for VCID are supported by neuroimaging

b. Both clinical and genetic evidence of cerebrovascular diseases.

Guidance: Genetic disorders include but are not limited to cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL); cerebral autosomal recessive arteriopathy with subcortical autosomal recessive leukoencephalopathy (CARASIL); hereditary endotheliopathy retinopathy nephropathy and stroke (HERNS); pontine autosomal dominant microangiopathy and leukoencephalopathy (PADMAL); retinal vasculopathy with cerebral leukodystrophy (RVCL); and collagen type IV, α-1 (COL4A1)–related disorders.

2. Possible. Clinical criteria for VCID are met, but neuroimaging and/or genetic evidence is not available

Subtypes of VCID

Hemorrhagic, ischemic, or mixed hemorrhagic-ischemic.

VasCog-2-WSO criteria (contd)

Multiple or Mixed Causation (Multifactorial or Mixed Cognitive Impairment or Dementia)

State which etiology is clinically more salient: vascular or other.

1. **VCID with 1 or more concomitant neurodegenerative disease(s) (eg, Alzheimer disease [AD], dementia with Lewy bodies) (a and b are required)**
    a. **Meets criteria for VCID (except for features that suggest an alternative etiology/etiologies)**
    b. **Meets criteria for other neurodegenerative disease(s) (eg, VCID [primary] with contribution from AD; or AD [primary] with contribution from VCID)**
2. **VCID with additional non-neurodegenerative pathology (eg, VCID [primary] with contribution from traumatic brain injury, brain tumor, etc)**
3. **VCID with contribution from depression**

Associated behavioral or psychiatric symptoms: with apathy, depressive symptoms, emotional lability, psychotic symptoms, agitation, etc.

VasCog-2-WSO criteria (contd)

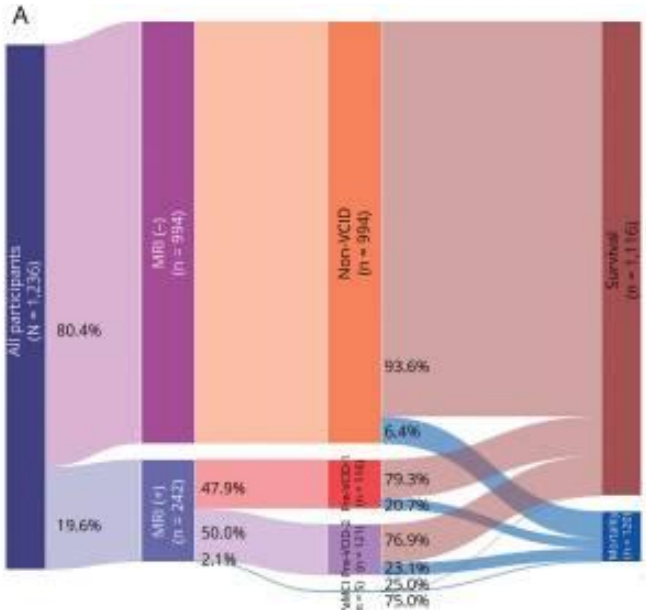
Preclinical/At-Risk VCID *(covert cerebrovascular disease cCVD*)
The purpose of this group would be to signal the importance of further study, primarily in a research setting, and for risk reduction and preventive medicine.
The category would require the person to present with (a and b):

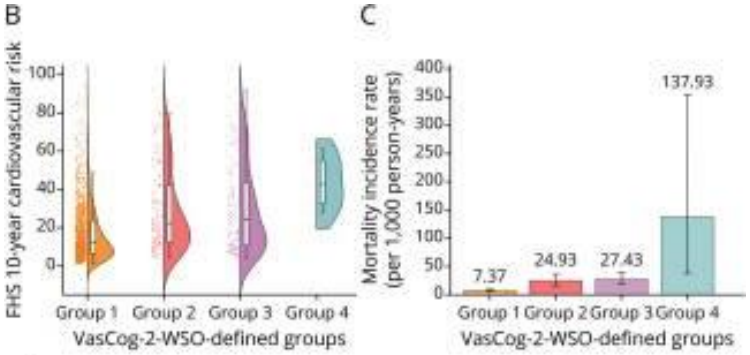
a. **Significant incidental neuroimaging evidence (MRI [preferred] or CT) of cerebrovascular disease that satisfies the VCID criteria for vascular aetiology criterion B; and**

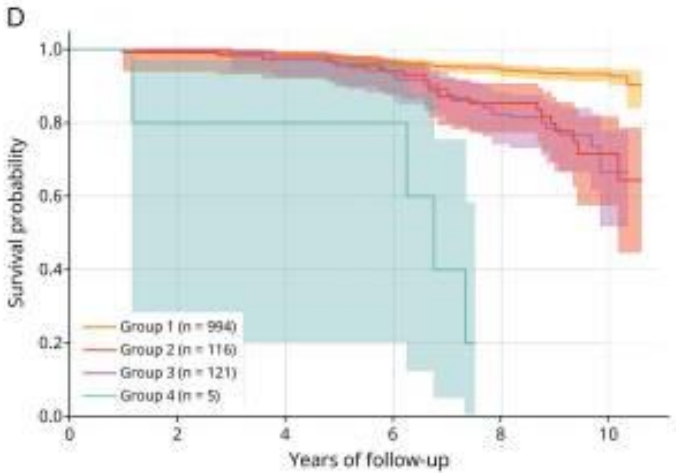
b. **Does not satisfy criteria for mild or major cognitive impairment/ dementia and the person, their informant or relevant clinician do not have concerns of cognitive change**

Guidance: this diagnosis is supported by the presence of substantial vascular risk factors for VCID.

Validation of the VasCog-2-WSO VCID Criteria in a Dementia-Free and Stroke-Free Community Cohort







1. Group 1 (neuroimaging-negative, non-VCID)
2. Group 2 (preclinical VCID without objective cognitive impairment)
3. Group 3 (preclinical VCID with objective cognitive impairment)
4. Group 4 (overt vaMCI).

Lin Y-T et al, Neurology 2026

Kalaria R & Sachdev P, Neurology 2026

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Harmonised Neuropsychological Battery

Table 3. International Society for Vascular Behavioural and Cognitive Disorders–Neuropsychological Consensus Core Harmonized Neuropsychological Test Battery

Domain and preferred tests	Time, mins[a]	H1	Alternative/supplemental tests	H2
Attention and processing speed				
TMT A[b,c]	2	NINDS-CSN, UDS3, C	SDMT (written and/or oral)[c]	NA
Digit Symbol Coding[c,d]	3	NINDS-CSN	Stroop-Non-interference[d]	
Digit Span Forward[c]	3	C	Number Span Forward[c]	UDS3
			Number Span Backward[c]	UDS3
			Color Trails A[c]	NA
			Oral SDMT[c,e]	NA
Executive function				
TMT B[b]	3	NINDS-CSN, UDS3, CUDS	Stroop-Interference[d]	NA
Digit Span Backward[c,e]	3	CUDS	Category Fluency Switching	NA
Letter fluency[d]	4	NINDS-CSN, UDS3, CUDS	Hayling Sentence Completion	NA
			Color Trails B[c]	NA
Learning and memory (verbal)				
CVLT[d]	10	NINDS-CSN	HVLT-R	NINDS-CSN
			RAVLT	NINDS-CSN
Learning and memory (visual)				
RCFT - Recall[b,c]	3	NINDS-CSN	VR (any variant)[c]	NINDS-CSN
			Doors Test[c,e]	NA
			Computerized test[c,e,f]	NA
Language				
Category fluency[b,d]	2	NINDS-CSN, UDS3, CUDS	MINT	UDS3
BNT[b]	5	NINDS-CSN, CUDS	SYDBAT subtests	NA
Perceptual-motor function				
RCFT- Copy[b,c,g]	3	NINDS-CSN, CUDS	Block Design (any variant)[c]	NA
			CLOX[c]	NA
			Matrix Reasoning[c]	NA
Mood				
GDS	3	NA	PHQ-9	NA
			CES-D	NINDS-CSN

Abbreviations: BNT, Boston Naming Test; CES-D, Center for Epidemiological Studies-Depression; CLOX, Clock Drawing Task; CUDS, Clinician's Uniform Dataset; CVLT, California Verbal Learning Test; GDS, Geriatric Depression Scale; H1, harmonization for preferred tests; H2, harmonization for alternative/supplemental tests; HVLT-R, Hopkins Verbal Learning Test, Revised; MINT, Multilingual Naming Test; NA, not applicable; NINDS-CSN, National Institute for Neurological Disorders and Stroke and the Canadian Stroke Network; PHQ-9, Patient Health Questionnaire Depression Module; RAVLT, Rey Auditory-Verbal Learning Test; RCFT, Rey Complex Figure Test; SDMT, Symbol Digit Modalities Test; SYDBAT, Sydney Language Battery; TMT A, Trail Making Test Part A; TMT B, Trail Making Test Part B; UDS3, Version 3 of the Alzheimer Disease Centers' Neuropsychological Test Battery in the Uniform Data Set; VR: visual reproduction.

[a] Administration time approximately 45 minutes or less. Times for each test are estimates based on publications where this is reported and/or estimates from the test publishers.

[b] Clear first preference from round 1 or 2.

[c] Tests with lower language demands, lower education influence, or lesser sociocultural content that may facilitate the assessment of persons from a diverse cultural and linguistic background. Users should still exercise caution in interpreting results from these groups.

[d] Any variant.

[e] Tests in a visual or spatial context without a drawing requirement, to better accommodate persons with motoric limitations.

[f] For example, Cambridge Neuropsychological Test Automated Battery Paired Associate Learning.

[g] Preferably also with a quantitative or qualitative measure of visuospatial function to help distinguish this from impairment that is predominantly mediated by executive function.

Bentvelzen AC,…., Sachdev PS.
JAMA Netw Open 2025;8;(12):e2547948.

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Further reading

1. VasCog-2-WSO Criteria Consortium; Sachdev PS, et al. Revised Diagnostic Criteria for Vascular Cognitive Impairment and Dementia-The VasCog-2-WSO Criteria. JAMA Neurol. 2025 Nov 1;82(11):1103-1112. doi: 10.1001/jamaneurol.2025.3242. PMID: 40955506
2. Sachdev PS, et al. Vascular Cognitive Impairment and Dementia: Clinical Features, Neuropathology, and Biomarkers. J Am Coll Cardiol. 2026 Jan 6;87(1):52-76. doi: 10.1016/j.jacc.2025.11.008. PMID: 41498479.
3. Goodall LS, Lennon MJ, Sachdev PS, Gorelick PB, Kovacic JC, Samaras K. Current and Emerging Therapeutic Approaches for Vascular Cognitive Impairment and Dementia. J Am Coll Cardiol. 2026 Jan 6;87(1):77-100. doi: 10.1016/j.jacc.2025.09.1502. PMID: 41498480.
4. Tardo DT, Cortes-Canteli M, Fuster V, Sachdev PS, Kovacic JC. The Heart-Brain-Metabolism Axis in Cardiovascular and Neurologic Disease. J Am Coll Cardiol. 2025 Dec 23;86(25):2663-2686. doi: 10.1016/j.jacc.2025.09.1602. PMID: 41432331.
5. Lennon MJ, Sachdev PS. Vascular cognitive impairment and dementia: Prevention, treatments, mechanisms and management options for the future. Neuropsychopharmacology. 2026 Jan 19. doi: 10.1038/s41386-026-02331-3. Epub ahead of print. PMID: 41554956.
6. Xu Y, Sachdev PS, Lennon MJ. Vascular cognitive impairment and dementia drug-development pipeline 2025. Alzheimers Dement (N Y). 2026 May 11;12:e70263. doi: 10.1002/trc2.70263. PMID: 42130608.
7. Hosoki S, Hansra GK, Jayasena T, …, Sachdev PS. Molecular biomarkers for vascular cognitive impairment and dementia. Nat Rev Neurol. 2023 Dec;19(12):737-753. doi: 10.1038/s41582-023-00884-1. Epub 2023 Nov 13. PMID: 37957261.

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