NEUROCOGNITIVE DISORDERS

Major or Mild Neurocognitive Disorder Due to Vascular Disease

VaD

DSM-5-TR 290.40 MAJOR, 331.83 MILD | ICD-10-CM F01.5X, G31.84

Cognitive decline caused by cerebrovascular disease, classically stepwise, with executive dysfunction and slowed processing leading the picture.

SHARE OF DEMENTIA	TYPICAL ONSET	SEX RATIO	COURSE
~15-20% of dementia cases	After 65; earlier post-stroke	Slight male predominance	Stepwise; ~5 yr median survival

CLINICAL PICTURE

- Processing speed, complex attention, and executive function fail before memory, so patients appear slowed and disorganized rather than forgetful.
- Decline is classically stepwise after clinical strokes, but small vessel disease produces a gradual course that mimics Alzheimer's on history alone.
- Focal neurological findings are common: gait disturbance, urinary urgency, brisk reflexes, pseudobulbar affect, and early falls.
- Memory failure is retrieval-based and improves with cueing and recognition, unlike the rapid forgetting and failed encoding of Alzheimer's disease.
- Apathy, emotional lability, and post-stroke depression are prominent and often burden families more than the cognitive deficits themselves.
- Insight is relatively preserved early, so patients report their own decline more often than families do at first presentation.

DIAGNOSTIC CRITERIA AT A GLANCE

- Criteria for major or mild neurocognitive disorder are met, with decline documented by informant report plus objective cognitive testing.
- Clinical features suggest a vascular etiology: onset tied to a cerebrovascular event, or prominent decline in complex attention and frontal executive function.
- Evidence of cerebrovascular disease from history, physical examination, or neuroimaging is sufficient to account for the observed deficits.
- Probable vascular etiology requires neuroimaging support, a clear temporal link to stroke, or genetic evidence; otherwise the etiology is called possible.
- Symptoms are not better explained by another brain disease or systemic disorder; specify major or mild, with or without behavioral disturbance.

NEUROBIOLOGY & PHYSIOLOGY

- Mechanisms span large-vessel infarcts, strategic single infarcts in thalamus or angular gyrus, lacunes, hemorrhage, and diffuse white matter ischemia.
- Subcortical small vessel disease disconnects frontal-subcortical circuits, which explains the executive and processing-speed signature of the syndrome.
- MRI shows white matter hyperintensities, lacunes, microbleeds, and enlarged perivascular spaces, graded with the Fazekas scale and STRIVE criteria.
- Hypertension, diabetes, atrial fibrillation, smoking, dyslipidemia, and obstructive sleep apnea are the dominant modifiable risk factors.
- CADASIL, caused by NOTCH3 mutations, produces early-onset subcortical disease with migraine with aura and merits testing before age 60.
- Mixed Alzheimer's and vascular pathology is the most common substrate found at autopsy in dementia beginning after age 80.

PSYCHOLOGICAL MECHANISMS

- Frontal-subcortical disconnection produces apathy that families read as laziness or depression, a distinction that changes the entire management plan.
- Insight preserved early in the course drives real demoralization, and post-stroke depression affects roughly one-third of stroke survivors.
- Pseudobulbar affect, with laughing or crying detached from felt emotion, distresses families, is commonly misread as lability, and is treatable.
- Slowed processing means patients answer correctly when given time, so rushed interviews and hurried caregiving manufacture apparent impairment.
- Behavioral activation and structured routine counter apathy far more reliably than encouragement or insight-oriented approaches.

DIFFERENTIAL & COMORBIDITY

- Alzheimer's disease shows insidious amnestic onset with little benefit from cueing, while vascular disease shows executive slowing with focal signs.
- Lewy body disease brings fluctuating attention, visual hallucinations, parkinsonism, and REM sleep behavior disorder, with gait overlap adding confusion.
- Normal pressure hydrocephalus presents with gait apraxia, urinary incontinence, and cognitive slowing, and is potentially reversible with shunting.
- Depression, delirium, obstructive sleep apnea, and medication effects mimic or amplify vascular cognitive impairment and should be treated first.
- Stroke, cardiac disease, diabetes, and depression coexist as the rule, so care is inherently multidisciplinary and medically driven.

Treatment

EVIDENCE-BASED MANAGEMENT

PHARMACOLOGIC

- Secondary stroke prevention is the core treatment: antihypertensives, **statins**, antiplatelet therapy, and anticoagulation for atrial fibrillation.
- Target blood pressure below 130/80 mm Hg where tolerated, since intensive control reduced incident cognitive impairment in randomized trials.
- Cholinesterase inhibitors and memantine offer minimal benefit in pure vascular disease, though they are reasonable when Alzheimer's pathology coexists.
- Treat post-stroke depression with an **SSRI** such as **sertraline** or **escitalopram**, weighing bleeding risk alongside antithrombotic therapy.
- **Dextromethorphan-quinidine** is approved for pseudobulbar affect and rapidly reduces involuntary episodes of crying and laughing.

PSYCHOTHERAPY

- **Cognitive rehabilitation** aimed at attention, planning, and error monitoring fits the executive profile far better than memory drills.
- **Behavioral activation** and problem-solving therapy treat post-stroke depression and apathy, with good evidence in rehabilitation settings.
- **Caregiver psychoeducation** reframes apathy as a symptom of brain injury rather than a choice, which reduces conflict and caregiver strain.
- **CBT** adapted for slowed processing, using shorter sessions and written summaries, treats comorbid anxiety and depression effectively.
- Goal-oriented rehabilitation with occupational and speech therapy after stroke yields real functional gains over the following months.

ADJUNCT & SUPPORTIVE

- Cardiovascular risk management is dementia treatment here: blood pressure, glucose, lipids, smoking cessation, and atrial fibrillation control.
- Aerobic exercise improves executive function and gait, and structured physical therapy reduces the fall risk that drives hospitalization.
- Treat obstructive sleep apnea with CPAP, since untreated apnea worsens vascular risk and cognitive performance simultaneously.
- Assess with the **MoCA**, which detects the executive deficits the MMSE misses, adding clock draw and trail making for frontal function.
- Address driving safety, falls, home modification, and advance care planning early, given the stepwise and unpredictable trajectory.

CLINICAL PEARLS

- Executive slowing with focal neurological signs, not amnesia, is the vascular signature.
- Cueing rescues recall in vascular disease; in Alzheimer's disease it does not.
- Treating blood pressure is the most effective drug therapy available here.

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NOTE

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