NEUROCOGNITIVE DISORDERS

Mild Neurocognitive Disorder

MCI

DSM-5-TR 331.83 | ICD-10-CM G31.84

Modest, objectively measured cognitive decline that does not yet compromise independence, occupying the zone between normal aging and dementia.

PREVALENCE AGE 65+	TYPICAL ONSET	PROGRESSION	REVERSION
~12-18% of adults 65+	Usually after age 60	~10-15% per year to dementia	~15-20% return to normal

CLINICAL PICTURE

- Patient or informant notices decline in memory, word finding, attention, or organization that is genuine but does not stop independent living.
- Complex tasks take longer or need compensation with lists, alarms, and reminders, while the tasks themselves still get completed correctly.
- Amnestic presentations, marked by forgetting recent conversations and appointments, carry the highest rate of progression to Alzheimer's disease.
- Non-amnestic presentations with executive, language, or visuospatial deficits point instead toward vascular, frontotemporal, or Lewy body pathways.
- Testing typically falls 1 to 2 standard deviations below age and education norms, a level routinely missed by brief screens with ceiling effects.
- Anxiety, depression, apathy, and disrupted sleep frequently accompany the decline and can exaggerate the measured degree of impairment.

DIAGNOSTIC CRITERIA AT A GLANCE

- Modest decline from a previous level of performance in one or more cognitive domains, based on concern from the patient, an informant, or a clinician.
- Objective impairment should be documented by standardized neuropsychological testing or another quantified clinical assessment.
- The deficits do not interfere with independence in everyday activities, though greater effort or compensatory strategies may now be needed.
- Deficits do not occur exclusively during delirium and are not better explained by another mental disorder such as major depression.
- Specify the presumed etiology, such as Alzheimer's disease, vascular disease, or Lewy bodies, and whether behavioral disturbance is present.

NEUROBIOLOGY & PHYSIOLOGY

- Amyloid PET or CSF positivity in amnestic cases identifies Alzheimer's pathology and raises annual conversion well above biomarker-negative cases.
- Plasma phosphorylated tau 217 now discriminates Alzheimer's pathology with accuracy approaching CSF, reshaping the workup of mild presentations.
- Hippocampal and entorhinal atrophy on MRI, alongside white matter hyperintensities, indicates the mix of degenerative and vascular contributions.
- APOE e4 carriage accelerates progression, while cognitive reserve built from education and occupational complexity delays clinical expression.
- Vascular risk factors, untreated hearing loss, obstructive sleep apnea, and physical inactivity are the largest modifiable contributors at this stage.
- Medication burden matters: anticholinergics, benzodiazepines, opioids, and sedative-hypnotics each measurably worsen cognitive performance.

PSYCHOLOGICAL MECHANISMS

- Awareness is usually preserved, so fear of becoming demented is common and independently degrades attention and test performance.
- Depression can produce or amplify the deficits, and treating it clarifies how much of the impairment is truly degenerative.
- Compensatory strategies work at this stage because encoding and retrieval remain partly intact and new habits can still be learned.
- Cognitive reserve buffers symptom expression, so highly educated patients harbor greater pathology before they fail any standard screen.
- Catastrophizing about every normal lapse produces hypervigilant self-monitoring and unnecessary disability well ahead of actual decline.

DIFFERENTIAL & COMORBIDITY

- Normal aging shows slowed processing without loss of learned material, whereas mild neurocognitive disorder shows measurable decline from personal baseline.
- Major neurocognitive disorder is distinguished solely by loss of independence in instrumental activities, not by test scores in isolation.
- Delirium must be excluded, and any acute or fluctuating change points toward a medical cause rather than a degenerative process.
- Screen for B12 deficiency, hypothyroidism, sleep apnea, depression, alcohol use, and polypharmacy, each of which can reverse the picture.
- Depression, anxiety, and sleep disorders are frequent comorbidities that worsen both subjective complaints and objective performance.

Treatment EVIDENCE-BASED MANAGEMENT

PHARMACOLOGIC

- Cholinesterase inhibitors are not indicated at this stage, as trials show no delay in conversion alongside meaningful gastrointestinal side effects.
- Anti-amyloid monoclonals **lecanemab** and donanemab are approved for biomarker-confirmed early Alzheimer's disease, with serial MRI monitoring for ARIA.
- Deprescribing anticholinergics, benzodiazepines, sedative-hypnotics, and unnecessary opioids is the most reliable pharmacologic gain available.
- Treat comorbid depression with an **SSRI** such as **sertraline 50-100 mg/day**, avoiding paroxetine and tricyclics for anticholinergic burden.
- Control blood pressure toward a systolic near 120 mm Hg, treat diabetes and dyslipidemia, and manage obstructive sleep apnea with CPAP.

PSYCHOTHERAPY

- **Cognitive rehabilitation** teaches individualized compensatory strategies aimed at real-life goals and outperforms generic computerized brain training.
- **CBT** for anxiety and depression improves mood and measured cognition whenever affective symptoms are inflating the apparent impairment.
- **Behavioral activation** and a structured daily routine counter the withdrawal and inactivity that accelerate functional decline at this stage.
- Psychoeducation for patient and family sets realistic expectations, reduces catastrophizing, and frames risk honestly without fatalism.
- Group memory strategy training using spaced retrieval and errorless learning produces modest but durable gains in everyday tasks.

ADJUNCT & SUPPORTIVE

- Aerobic exercise of 150 minutes weekly plus resistance training carries the strongest behavioral evidence for slowing cognitive decline.
- Multidomain intervention combining diet, exercise, cognitive training, and vascular monitoring improved cognition in the **FINGER** trial.
- Treat hearing loss with amplification and correct vision, both of which reduce apparent impairment and sustain social engagement.
- Establish a baseline with the **MoCA**, more sensitive than the MMSE at this level, and repeat testing every 6 to 12 months.
- Discuss driving, finances, and advance planning while the patient can still direct those decisions, and revisit as function changes.

CLINICAL PEARLS

- Independence, not the test score, separates mild from major neurocognitive disorder.
- Use the MoCA, not the MMSE: the MMSE ceilings out and misses this stage entirely.
- Review the medication list first; anticholinergic burden is reversible impairment.

References

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NOTE

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