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Clinical Reasoning Case

When "Normal Enough" is Not Normal: Detecting Mild Neurocognitive Disorder Through Integrated Primary Care and Neuropsychology

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CASE PRESENTATION

81-year-old woman presented to the clinic with family concern regarding her declining cognitive function over a 12-month period. Her primary care physician noted emerging forgetfulness including repetitive content in patient communications which were corroborated by the patient's spouse. He described a gradual decline in managing her financial affairs as well. Our patient consistently denied cognitive changes and attributed lapses to normal aging. She did note independence in basic activities of daily living (ADL) and most instrumental ADLs. The patient denied any prior neurologic or psychiatric conditions. Her previous medical history was significant for well-controlled hypertension and hyperlipidemia with a low-density lipoprotein (LDL) at goal. She had a history of premature ventricular (PVCs) and premature atrial contractions (PACs).

The discrepancy between the patient's self-reported and observations from both the physician and spouse raise concern for anosognosia, which is defined as a neurological condition where a person is physically unable to recognize or perceive their own medical, psychiatric, or cognitive impairment. It is commonly seen in early Alzheimer's disease patients.[1] In an older adult with a gradual, insidious course of change, this pattern increases the pre-test probability of an underlying neurodegenerative process. The differential diagnosis includes mild neurocognitive disorder due to Alzheimer's disease, vascular cognitive impairment given existing vascular risk factors, and less likely, normal age-related cognitive change or psychiatric contributors. At this stage, collateral information is essential, as patients may underestimate or lack awareness of early deficits. Early cognitive decline in primary care is often first detected through longitudinal observation and collateral report rather than patient complaint. In this case, repeated observations across encounters, supported by informant history, appropriately increased clinical suspicion despite preserved functional independence, lack of clear symptoms reported by the patient, and likely high cognitive reserve. This is the brain's ability to improvise, find alternate ways to complete tasks, and maintain function despite damage from disease, injury, or aging. For internists, discordance between patient and informant perspectives should prompt further evaluation rather than reassurance.[2]

Our patient's Mini-Cog score was 4/5 with a 1-word recall deficit. Her Montreal Cognitive Assessment (MoCA) score was 24/30, with deficits in cube copy and delayed recall, falling near the commonly used abnormal threshold. She was noted to have high cognitive reserve based on educational and occupational history.

Brief cognitive screening tools are designed for sensitivity but have reduced specificity in highly educated individuals, where cognitive reserve may mask early decline.[3] Scores near established cutoffs, such as a MoCA of 24/30, are inherently ambiguous, especially in a patient who is not reporting cognitive symptoms and can be falsely reassuring if interpreted without clinical context.[4] In this case, the presence of domain-specific deficits increases suspicion for early impairment despite "near-normal" scores. This case illustrates the limitations of relying on screening thresholds alone in high-functioning patients. For internists, the key clinical task is not simply interpreting a score but determining whether that score aligns with the patient's expected baseline and overall clinical picture. Borderline results should prompt reconsideration rather than reassurance when they conflict with longitudinal observations or informant concerns.

Despite somewhat reassuring cognitive screening scores and preserved functional status, concern for cognitive decline remained based on a discrepancy between screening results and clinical impression based on longitudinal observation. Referral was made to an embedded neuropsychologist within the primary care clinic for comprehensive evaluation, with the goal of obtaining timely diagnostic clarification.

The presence of subtle domain-specific deficits on screening lowered the threshold for further evaluation in the context of high clinical suspicion. Access to neuropsychology enabled timely, lower-barrier assessment, allowing the primary care physician to characterize cognitive functioning and distinguish early neurodegenerative disease from normal aging.[5] This approach supports earlier diagnosis and more targeted management. When screening results are equivocal but clinical concern remains high, referral for comprehensive evaluation is appropriate. Embedding neuropsychology within primary care reduces delays, enhances diagnostic accuracy, and facilitates collaborative care plan-

ning. For internists, this model supports more confident decision-making in cases where screening tools provide incomplete or ambiguous information. We found that the patient's intellectual functioning (IQ scores) to be high average to superior. Her attention, processing speed, executive function were intact. Her memory assessment revealed bilateral impairments in verbal and nonverbal learning, retention, and delayed recall, reflecting encoding and storage deficits. Her visuospatial functioning was mildly weak on complex visuospatial construction, and her language revealed subtle confrontation naming decline. Her overall functional assessment was preserved basic ADLs with mixed instrumental ADL concerns.

Can neuropsychological testing differentiate normal aging, vascular cognitive impairment, and early Alzheimer's disease and what specific features on neuropsychological evaluation support a diagnosis of mild neurocognitive disorder due to Alzheimer's pathology?

Neuropsychological testing demonstrating consistent impairments in memory encoding and storage, especially when bilateral and occurring in the context of preserved attention and executive functioning, is characteristic of early Alzheimer's disease. Visuospatial and naming weaknesses further support this etiology. In contrast, vascular cognitive impairment more commonly affects frontal-subcortical areas, leading to prominent executive dysfunction, slowed processing speed, or attention deficits; memory performance often reflects impaired retrieval rather than storage, and relatively preserved recognition.[6] A pattern of broadly intact cognition with only mild inefficiencies would be more consistent with normal age-associated cognitive change.

Neuropsychological profiles are particularly valuable in distinguishing normal aging, vascular cognitive impairment, and early Alzheimer's disease when brief cognitive screening yields equivocal results. Because neuropsychological test interpretation accounts for age and educational background, deviations from expected baseline can be quantified, and patterns of impairment provide important etiologic cues. Specifically, memory testing that shows impaired learning, retention, and recognition across both verbal and non-verbal modalities, is more characteristic of mesial-temporal involvement, a hallmark of Alzheimer's disease pathology. In contrast, memory profiles marked by retrieval difficulty with preserved recognition are more suggestive of frontal-subcortical dysfunction, as may be seen in vascular cognitive impairment. In this case, the circumscribed yet consistent memory deficits helped explain why screening scores appeared only mildly abnormal, while still representing a clinically meaningful decline from the patient's high premorbid baseline.

Our patient underwent brain magnetic resonance imaging (MRI) with contrast which revealed no acute or prior infarcts. No hippocampal atrophy was noted. There were scattered bilaterial white matter FLAIR hyperintensities consistent with mild chronic microvascular ischemic changes and mild age-related cerebral atrophy.

Neuroimaging findings of mild atrophy and chronic microvascular changes are common in aging and should be interpreted cautiously as they often lack specificity.[7] Moreover, these findings do not adequately account for the patient's pronounced memory impairments. The absence of hippocampal atrophy does not exclude Alzheimer's disease, particularly in the earlier stages of illness. When imaging findings are interpreted alongside the neuropsychological profile (marked by bilateral impairments in memory encoding and storage with preserved executive functioning) the overall pattern is more consistent with Alzheimer's pathology than vascular cognitive impairment. The gradual progression of symptoms and the patient's limited insight further support this conclusion. Attribution of etiology in early cognitive decline often requires synthesizing imperfect and sometimes discordant data. In this case, the convergence of a progressive amnestic neuropsychological profile, preserved non-memory domains, and anosognosia outweighed nonspecific imaging findings, supporting a diagnosis of mild neurocognitive disorder likely due to Alzheimer's disease.

For internists, this case highlights the importance of integrating multiple data sources when evaluating early cognitive symptoms, including neuropsychological assessment and neuroimaging findings. The case demonstrates that primary care physicians can arrive at a confident diagnosis of early neurocognitive disorder when integrating clinical, neuropsychological, and imaging data.

The primary care physician and the neuropsychologist developed a joint management plan addressing safety, future planning, and longitudinal monitoring. Interventions included counseling regarding advance care planning, medication options, driving safety, and anticipatory guidance for the patient and spouse. Management strategies accounted for the patient's limited insight alongside preserved functional independence and compensatory skills.

Management of mild neurocognitive disorder in primary care requires addressing medical, functional, and psychosocial domains over time. Integrating neuropsychological recommendations with the primary care physician's longitudinal knowledge of the patient supports early discussions of advance care planning, driving safety, and medication options. This enables patients to meaningfully participate while decision-making capacity is preserved. When insight is limited, communication should be framed around observable changes and should be future oriented rather than focusing on diagnostic labels. Engaging caregivers is critical for safety monitoring, supporting adherence, and addressing psychosocial needs. Routinely assessing caregiver stress and facilitating connection to caregiver support can mitigate downstream burden. Regular follow-up enables timely reassessments of functioning and adjustments of management as the disease evolves. Primary care clinicians play a central role in the ongoing management of mild neurocognitive disorder, particularly when patients remain functionally independent but have limited awareness of cognitive change. In this context, management decisions extend beyond pharmacologic considerations to include counseling,

safety planning, and anticipatory guidance.[8] Framing discussions around maintaining independence and preparing for future needs can help preserve patient autonomy while supporting caregiver involvement. This case illustrates how early, coordinated management in primary care can align patient values, caregiver concerns, and clinical priorities in the setting of early neurodegenerative disease.

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