Detection: If a patient or caregiver presents with concerns about a change in cognition, the healthcare provider (HCP) should review cognitive history and focus on evidence of impairment in two or more cognitive domains. Patients with mild AD dementia will have symptoms that interfere with their ability to function.1,2

Examples of Cognitive Concerns2,3

- Problems Planning
- Decreased Judgment
- Confusion with Time or Place
- Difficulty with Visual and Spatial Relationships
- Memory Loss
- Mood Changes
- Social Withdrawal
- Difficulty with Familiar Tasks
- Language Problems

LINDA, 75 YEARS OLD
Declining functionality

“I used to be the mother hen. I knew where everything was. I knew what everyone was doing. Now, I feel more lost every day.”
American Academy of Neurology (AAN) guidelines recommend: If a patient or caregiver presents with a concern about memory or impaired cognition, HCPs should assess for Alzheimer’s disease and not assume the concerns are related to normal aging.4

Brief cognitive assessments can provide evidence of impairment

AAN guidelines recommend: HCPs assessing cognitive impairment should use brief, validated cognitive assessment tools, in addition to eliciting a history of cognitive concerns, to build an accurate diagnosis and run tests to rule out other causes.4,5

Evidence of progressive cognitive decline is essential for accurate AD diagnosis and treatment. While no test represents a “gold standard,” use of brief cognitive assessment tools with appropriate patients can aid in the early identification of mild cognitive impairment (MCI) due to AD and mild AD dementia. Screening assessments will vary based on clinical practice settings and patient response.6,7

Examples of Common Screening Tools8-10

<table>
<thead>
<tr>
<th>MMSE</th>
<th>MoCA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cognitive areas assessed</td>
<td>Orientation • Attention and concentration • Language • Visual construction • Memory</td>
</tr>
<tr>
<td>Sensitivity for MCI detection</td>
<td>Less sensitive; patients with MCI may score as “normal”</td>
</tr>
<tr>
<td>Sensitivity for moderate or severe impairment</td>
<td>Appropriate</td>
</tr>
<tr>
<td>Administration time</td>
<td>10 min</td>
</tr>
<tr>
<td>Scoring</td>
<td>Maximum of 30 points† 25 or above = normal cognitive function 20 to 24 = mild dementia 13 to 20 = moderate dementia 12 or lower = severe dementia</td>
</tr>
</tbody>
</table>

†Cutoff varies with age and education.11
‡The raw score is adjusted by educational attainment (1 extra point for 10 to 12 years of formal education; 2 points added for 4 to 9 years of formal education).9

Other assessment tools include: GP-COG (General Practitioner Assessment of Cognition), Mini-Cog, ACE-R (Addenbrooke’s Cognitive Examination – Revised), Clock Drawing Test, 6-CIT (6-Item Cognitive Impairment Test), MIS (Memory Impairment Screen).7,8

MMSE=Mini-Mental State Examination; MoCA=Montreal Cognitive Assessment.

A Patient With Declining Functional Independence Is Worried About Her Future

Linda*
75-year-old female
Worsening cognitive problems; memory failure.

OCCUPATION: Widowed/living alone
EDUCATION: College degree
FAMILY HISTORY OF AD/DEMENTIA: No
COMORBIDITIES: Coronary artery disease and diabetes
MEDICATION: Lisinopril, metformin
NEUROLOGICAL TESTS: Difficulty with ideomotor apraxia testing, like mimicking the combing of her hair, and some diminished balance
LABORATORY TESTS: Within normal limits

*Not an actual patient. Patient profile provided for illustrative purposes only.
Evidence for the diagnosis of mild Alzheimer’s disease dementia

**COGNITIVE ASSESSMENT:**
Elevated subjective memory complaints, MMSE=24; conceptual and spatial/planning errors in the Clock Drawing Test without hands and with gaps in number spacing, respectively

**FUNCTIONAL DEPENDENCE:**
Difficulty with daily functioning, including housework, shopping, remembering her medications; dependent for managing finances (Stage 4 on FAST [Functional Assessment Staging Test]—mild AD dementia)

**MRI:**
Ventricular dilatation, hippocampal atrophy, mild frontal and anterior temporal cortical atrophy

To help differentiate the cause of cognitive decline, structural brain MRI may be used to rule out other systemic brain diseases (vascular, traumatic, medical).6,12

MRI=magnetic resonance imaging.

Confirming disease pathology with biomarkers

Biomarkers of amyloid beta (Aβ) deposition can be used to help establish the underlying etiology of the clinical syndrome.13,14

Once it is determined that the clinical syndrome is consistent with AD, the clinician can determine the primary cause.9 Biomarker tests that can detect Aβ help support the early detection of mild AD dementia by confirming abnormal pathophysiological changes.13,14

The diagnostic value of AD as the cause of cognitive impairment provides the clinician an opportunity to intervene before greater neuronal damage occurs, and more cognition and function are lost.15

Aβ biomarker tests include15:
• Positron emission tomography (PET) imaging
• CSF test

Measures of Aβ by CSF test and PET imaging are strongly and inversely correlated, reflecting Aβ deposition in the brain.18,19

**BIOMARKER CONFIRMATION (CSF):**
CSF Aβ_{42/40} detected below LLN (lower limit of normal) cutoff

**DIAGNOSIS:** Mild AD dementia
The earliest signs of Alzheimer’s disease present your earliest opportunity to intervene.

Early AD dementia still presents an opportunity to diagnose and manage the disease.

Alerting your patient or a caregiver about a mild AD dementia diagnosis can provide many benefits, including the potential to reduce patient anxiety by addressing their concerns about symptoms and allowing them to make informed decisions and plan for the future.

**Start a dialogue with your patient about mild AD dementia**

Early diagnosis allows for individual management plans, including multi-domain non-pharmaceutical interventions that may potentially improve cognition.

Listen for cognitive complaints and look for the core clinical criteria of AD.

Use cognitive assessment tools to build an accurate diagnosis and run tests to rule out reversible, non-AD causes.

After clinical diagnosis, Aβ testing can be used to confirm AD pathology.

**Biomarkers can be used to help support your diagnosis.**

**References:**

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