Mr. R, age 67, presents with what he describes as uncharacteristic “memory loss” that is affecting his ability to run his accounting business. He and his wife relate that he was doing well until approximately 9 months ago, when he started showing difficulties remembering clients’ names and addresses.

His wife became extremely concerned when he made serious accounting errors in a 1-month period that resulted in the loss of a longtime customer. Mr. R has become easily distracted and absentminded as well, and his wife reports he is misplacing things around the house.

Screening for mild cognitive impairment (MCI) is not recommended for asymptomatic, cognitively healthy older persons, but memory complaints in individuals age ≥50—especially when corroborated by a reliable informant—warrant further assessment. Your challenge is to determine whether subtle cognitive changes in patients such as Mr. R are part of normal aging, caused by medical or mental illnesses, or a harbinger of Alzheimer’s disease (AD) or another dementia.

continued
Amnestic MCI: Proposed diagnostic criteria

| Subjective memory impairment, preferably corroborated by a reliable informant |
| Reduced performance on objective memory tests, compared with persons of similar age and educational background |
| Typical general cognitive function |
| Intact basic activities of daily living and intact or minimally impaired instrumental activities of daily living |
| Absence of dementia |

MCI: mild cognitive impairment
Source: Reference 8

Although no treatments can yet prevent dementia, substantial new research is defining the MCI diagnosis for clinicians. This article describes:

- the evolving understanding of MCI and its subtypes
- risk factors for converting from MCI to AD
- an evidence-based work-up (including functional, cognitive, and neuropsychological testing)
- neuroprotective strategies for patients with an MCI diagnosis, including evidence on cholinesterase inhibitors, vitamin E, and anti-inflammatory agents.

MCI’s evolving definition

MCI is characterized by subjective and objective cognitive decline greater than expected for an individual’s age and education but less than the functional deficit required for a dementia diagnosis. MCI is proposed to identify persons with early but pathologic cognitive impairment that has a high risk to progress to AD and possibly other dementias.

MCI is thought to be a transitional state between normal aging and dementia. Its estimated prevalence in the general population is 19% among individuals age <75 and 29% in those age >85.

MCI subtypes. Some experts view MCI as a single entity, whereas others suggest amnestic (aMCI) and nonamnestic (nMCI) subtypes. Each subtype is further divided into single and multiple cognitive domains. So, for example, the diagnosis would be:

- aMCI-single cognitive domain for memory impairment only
- aMCI-multiple cognitive domains for memory impairment plus nonmemory deficits, such as in language, executive function, or visuospatial function
- nMCI-single or multiple cognitive domains for nonmemory deficits without memory impairment.

MCI subtypes may have different outcomes for progression to dementia, and all progressive dementias may have their own predementia states. Vascular MCI, for instance, is thought to result from cerebrovascular disease and is proposed to describe a prodrome of vascular dementia.

Determining a patient’s MCI subtype is still a research activity and calls for comprehensive neuropsychological testing. MCI patients perform at least 1.5 standard deviations below the average for age- and education-matched healthy individuals on objective measures of memory.

Conversion to dementia

In longitudinal population studies patients with MCI have shown an 11% to 33% risk of developing dementia within 2 years, whereas 44% reverted to normal 1 year later. Reasons for reversibility may include variable definitions of MCI among the longitudinal studies and the possibility that patients who recovered or improved may have had reversible causes of dementia.

When patients with MCI are followed over time, they progress not only to AD but also to non-AD dementias. For example, patients with Parkinson’s disease (PD) and MCI may be at higher risk of progressing to dementia than cognitively intact PD patients. MCI patients with the e4 allele of the apolipoprotein E gene (ApoE e4) are at increased risk to convert from MCI to AD.

Individuals with aMCI (Table 1) progress to AD at a rate of 10% to 15% per year, compared with 1% to 2% per year.
Factors shown to predict conversion from MCI to dementia

<table>
<thead>
<tr>
<th>Category</th>
<th>Predictors of conversion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical</td>
<td>Cognitive: Amnestic MCI</td>
</tr>
<tr>
<td></td>
<td>Neuropsychiatric: Depression, apathy, and possibly nighttime behaviors and anxiety</td>
</tr>
<tr>
<td>Neuropsychological tests</td>
<td>Clock-drawing test, Trail-Making Test B, Symbol Digit Modalities Test, Delayed 10-Word List Recall, New York University Paragraph Recall Test (Delayed), ADAS-Cog total score</td>
</tr>
<tr>
<td>Neuroimaging</td>
<td>MRI: Entorhinal cortex and hippocampal atrophy</td>
</tr>
<tr>
<td></td>
<td>PET: Medial temporal region, parietotemporal association cortex, and posterior cingulate hypometabolism</td>
</tr>
<tr>
<td></td>
<td>fMRI: Abnormal hippocampal, posterior cingulate, and medial temporal region activation on memory tasks</td>
</tr>
<tr>
<td>CSF markers</td>
<td>Increase: t-tau, p-tau</td>
</tr>
<tr>
<td></td>
<td>Decrease: Aβ 42</td>
</tr>
<tr>
<td>Genetic markers</td>
<td>ApoE e4 carriers</td>
</tr>
</tbody>
</table>

ADAS-Cog: Alzheimer's Disease Assessment Scale-Cognitive subscale; ApoE: apolipoprotein E gene; e4 allele; CSF: cerebrospinal fluid; MCI: mild cognitive impairment; MRI: magnetic resonance imaging; fMRI: functional MRI; PET: positron-emission tomography

Source: References 7,9-15

in normal elderly persons. The Mayo AD research center studies reported a conversion rate of up to 80% from aMCI to AD within 6 years.9

Research focuses on identifying preclinical AD states and potential targets for intervention using disease-modifying therapies. Some experts consider MCI to be the earliest clinical manifestation of AD, at least in a subgroup of patients.

Identifying markers to predict which patients are likely to convert from MCI to dementia also is a major research objective. In addition to ApoE status (Table 2),7,9-15 predictors of conversion may include:

- hippocampal atrophy13
- reduced metabolism in the temporoparietal cortex and posterior cingulum14
- elevated CSF tau and the 42 amino acid form of β-amyloid (Aβ 42).15

Research techniques such as structural neuroimaging, positron-emission tomography, functional magnetic resonance imaging (fMRI), and cerebrospinal fluid biomarkers have not been defined for clinical use, however.

Neuropsychiatric symptoms. Individuals with MCI and neuropsychiatric symptoms may be at particular risk for progressing to dementia. Agitation or depression are more prevalent in persons with MCI than in normal elderly but less prevalent than in those with dementia (Table 3, page 40).16,16

The cross-sectional, community-based Cardiovascular Health Study showed one or more neuropsychiatric symptom in:

- 16% of normal healthy elderly
- 43% of MCI patients
- 75% of dementia patients.16

Depression (20%), apathy (15%), and irritability (15%) were the neuropsychiatric symptoms reported most frequently in MCI patients, compared with apathy (36%), depression (32%), and agitation/aggression (30%) in dementia patients.

Sleep disturbances and anxiety in persons with MCI may predict progression to AD.10 A baseline high frequency of apathy in aMCI patients has been associated with progression to AD within 1 year.11

Depression and MCI

Depression and cognitive complaints overlap considerably in older adults. Depressed patients without dementia show persistent cognitive deficits even after depression remits. In some patients, new-onset geriatric
In the differential diagnosis of MCI, significant functional impairment points toward dementia.

depression is considered a prodrome of MCI and AD. Given that AD neuropathologic changes precede clinical symptoms by many years, depression and AD have been proposed as different clinical manifestations of AD pathology.17

Among patients with MCI, 20% meet criteria for major depression and 26% for minor depression. Symptoms often include sadness, poor concentration, inner tension, pessimistic thoughts, lassitude, and insomnia.18

Depressed MCI patients are at higher risk of developing dementia than those without depression, especially if cognitive measures do not improve after depression is treated.19 Similarly, cognitively intact older persons who develop depression are at increased risk for MCI, particularly if they carry the ApoE e4 genotype.19

In the only study in which MCI patients’ neuropsychiatric symptoms have been treated, 39 elderly patients with depression and MCI received open-label sertraline, ≤200 mg/d, for 12 weeks. Among the 26 patients who completed the trial, 17 showed moderate improvement in depressive symptoms, attention, and executive function, and 9 showed no response.20

**Recommendation.** In clinical practice, antidepressant treatment—usually a selective serotonin reuptake inhibitor (SSRI), with or without psychotherapy—is recommended for the MCI patient with comorbid major depression.

**CASE CONTINUED**

**No signs of depression**

Mr. R’s medical, neurologic, and substance use history is unremarkable. Family history includes AD in a paternal aunt diagnosed at age 82. Mr. R reports no history of mood, sleep, or appetite changes and no psychotic symptoms. He shows no deficits in activities of daily living (ADL), although his wife recently took over paying household bills after he forgot to make a payment.

**Evidence-based workup**

**Functional assessment.** In the differential diagnosis of MCI, give special attention...continued on page 46

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**Table 3**

Neuropsychiatric symptoms: Rising prevalence mirrors cognitive deterioration in elderly patients*

<table>
<thead>
<tr>
<th>Neuropsychiatric symptoms</th>
<th>Normal elderly</th>
<th>MCI</th>
<th>Mild AD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressed mood/dysphoria</td>
<td>+</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>Nighttime behaviors/sleep</td>
<td>+</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>Irritability</td>
<td>+</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>Anxiety</td>
<td>+/-</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>Apathy/indifference</td>
<td>+/-</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>Agitation/aggression</td>
<td>+/-</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>Eating/appetite disturbance</td>
<td>+/-</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>Disinhibition</td>
<td>+/-</td>
<td>+/-</td>
<td>++</td>
</tr>
<tr>
<td>Aberrant motor behavior</td>
<td>0</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>Delusions</td>
<td>0</td>
<td>+/-</td>
<td>++</td>
</tr>
<tr>
<td>Euphoria</td>
<td>0</td>
<td>+/-</td>
<td>+/-</td>
</tr>
<tr>
<td>Hallucinations</td>
<td>0</td>
<td>0</td>
<td>+</td>
</tr>
</tbody>
</table>

* 0 = 0%; +/- = 1% to 5%; + = 6% to 10%; ++ = 11% to 20%; +++ = 21% to 40%

MCI: mild cognitive impairment; AD: Alzheimer’s disease

Source: References 10, 16
trials. Body as a Whole—asthma, back pain, accidental injury, chest pain, Cardiovascular—hypertension, Digestive—dry mouth, increased appetite, thirst, constipation, increased salivation; Metabolic and Nutritional—intraduodenal fat intolerance; Skin and Appendages—skin and subcutaneous tissue disorder, Nervous System—confusion, tremor, depression, dizziness, speech disorder, amnesia, paresthesia, apathy, confusion, euphoria, hallucinations; Respiratory—dyspnea; Special Sensory—ampholytic, abnormal vision; Urinary—polyuria.

Adverse Events Listed in Clinical Trials—The following treatment-emergent adverse events were reported at an incidence of <1%, with intramuscular olanzapine for injection (2.5, 10 or 15 mg/d) compared to placebo in short-term, placebo-controlled trials in patients with schizophrenia or bipolar mania: Body as a Whole—asthma, Cardiovascular—hypertension, Digestive—dry mouth, increased appetite, thirst, constipation, increased salivation, Metabolic and Nutritional—hyperglycemia, hypertriglyceridemia; Nervous System—confusion, tremor, depression, dizziness, speech disorder, amnesia, paresthesia, apathy, confusion, euphoria, hallucinations; Respiratory—dyspnea; Special Sensory—ampholytic, abnormal vision; Urinary—polyuria.

Table 4

The following treatment-emergent adverse events were reported with a frequency of <1% in intramuscular olanzapine for injection (2.5, 10 or 15 mg/d) compared to placebo in short-term, placebo-controlled trials in patients with schizophrenia or bipolar mania: Body as a Whole—asthma, Cardiovascular—hypertension, Digestive—dry mouth, increased appetite, thirst, constipation, increased salivation, Metabolic and Nutritional—hyperglycemia, hypertriglyceridemia; Nervous System—confusion, tremor, depression, dizziness, speech disorder, amnesia, paresthesia, apathy, confusion, euphoria, hallucinations; Respiratory—dyspnea; Special Sensory—ampholytic, abnormal vision; Urinary—polyuria.

Gastroparesis

Patients with gastroparesis can be more sensitive to the effects of olanzapine. Considerations for patients with gastroparesis include decreased absorption potential for oral olanzapine and increased risk of orthostatic hypotension and hypotension, particularly in patients with cardiovascular disease, diabetes, or elderly patients. Olanzapine should be started at the lowest possible dose and titrated gradually to the desired dose.

Cognitive assessment. Because most individuals with MCI score in the normal range on the following tests: The Montreal Mini-Mental State Examination (3MS, modifified MMSE 3MS), which is based on 16 ADL and IADL items and can be more sensitive for detecting MCI. The 3MS retains the MMSE’s brevity (41 minutes to administer) but incorporates 4 additional items, has more graded scoring responses, and broadens the score range to 0 to 10. The clock-drawing test is also sensitive for MCI, especially in detecting early visuocognitive dysfunction.

The Montreal Cognitive Assessment (MoCA) is a 10-minute, 30-point scale designed to help clinicians detect MCI (see Related Resources, page 41). The MoCA usually is given with the modified MMSE for a comprehensive cognitive assessment.

Neasreddine et al administered the MoCA to MCI and 94 patients who met clinical criteria for MCI, 93 patients with mild AD, and 90 healthy cognitively normal elderly persons, using a cutoff score of 26. MoCA showed:

- 90% sensitivity for detecting MCI (compared with 18% for the MMSE)
- 87% specificity to exclude normal elderly persons.

The average MoCA score in patients with AD was much lower than in individuals with MCI, but score ranges of these 2 groups overlapped. Therefore, a score <26 could indicate either MCI or AD, and the distinction depends on the patient for functional impairment.

Neuropsychological testing can be more sensitive than office-based screening tools in defining MCI subtypes. In the Alzheimer’s Disease Cooperative Study (ADCS), the neuropsychological measures that most accurately predicted progression of patients with aMCI to AD within 36 months were the:

- Symbol Digit Modalities Test

continued from page 40
• New York University Paragraph Recall Test (Delayed)
• Delayed 10-Word List Recall
• Alzheimer’s Disease Assessment Scale-cognitive subscale (ADAS-Cog) total score.24

**Laboratory tests, imaging.** Use laboratory studies (Table 5, page 48) to rule out reversible causes of MCI symptoms.8 Reserve CSF studies for suspected CNS infection (such as meningitis, human immunodeficiency virus, or neurosyphilis) and brain imaging for suspected cerebral pathology (such as infarct, subdural hematoma, normal pressure hydrocephalus, or tumor).

**CASE CONTINUED**

**Subtle cognitive deficits**

Mr. R scores 27/30 on the MMSE (losing 3 points on recall) and 25/30 on the MoCA (losing points on visuospatial/executive function, fluency, and delayed recall). Thyroid-stimulating hormone, vitamin B12, folic acid, and rapid plasma reagin tests are unremarkable; brain MRI shows no significant abnormalities.

You refer Mr. R for neuropsychological testing, and most cognitive domains are normal. Exceptions include moderate impairment in immediate and delayed verbal and visual memory and mild executive dysfunction.

Based on your clinical evaluation and neuropsychological testing, you diagnose amnestic MCI. Mr. R shows abnormalities in memory and executive functioning without significant decline in basic and instrumental ADLs, is not taking medications, and has no other medical or psychiatric condition that could explain his cognitive deficits.

You discuss the diagnosis with him and his wife, including evidence on his risk for progression to dementia, neuroprotective strategies, and medications.

**After an MCI diagnosis**

**Neuroprotection.** Eliminate medications with anticholinergic effects, including:

- tricyclic antidepressants
- conventional antipsychotics
- antihistamines

**Table 4**

<table>
<thead>
<tr>
<th>Alzheimer’s Disease Functional Assessment and Change Scale (ADFACS)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Basic ADL</strong></td>
</tr>
<tr>
<td>Toileting</td>
</tr>
<tr>
<td>Feeding</td>
</tr>
<tr>
<td>Dressing</td>
</tr>
<tr>
<td>Personal hygiene and grooming</td>
</tr>
<tr>
<td>Bathing</td>
</tr>
<tr>
<td>Walking</td>
</tr>
<tr>
<td>Grasp of situations and explanations</td>
</tr>
<tr>
<td>Handling personal mail</td>
</tr>
</tbody>
</table>

The 16-item ADFACS total score ranges from 0 to 54 (best to worst):

- Rate basic ADLs from 0 (no impairment) to 4 (very severe impairment), for a total score range of 0 to 24.
- Rate IADLs from 0 (no impairment) to 3 (severe impairment), for a total score range of 0 to 30.

Use total scores to assess for functional decline from baseline. A decline from 0 to 1 on individual ADL and IADL items is not considered clinically significant.

**Clinical Point**

Give the 10-minute Montreal Cognitive Assessment (MoCA) with the modified MMSE for a comprehensive cognitive assessment

- drugs used to treat urinary incontinence, such as oxybutynin
- muscle relaxants, such as cyclobenzaprine
- certain antiparkinsonian drugs, such as benztropine.

Encourage patients to avoid alcohol and sedatives. Collaborate with primary care providers to control cerebrovascular risk factors such as hyperlipidemia, diabetes mellitus, hypertension, and obesity. Treat depression, which may be a risk factor for developing dementia.

**Monitoring.** The American Academy of Neurology recommends monitoring patients diagnosed with MCI every 6 to 12 months for cognitive and functional decline. continued
Mild cognitive impairment

Clinical Point
To protect the brain, work with the MCI patient’s physician to stop anticholinergics and control diabetes, blood pressure, and hyperlipidemia

Table 5
Lab studies to rule out reversible causes of MCI

<table>
<thead>
<tr>
<th>Test</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete blood count with differential</td>
<td></td>
</tr>
<tr>
<td>Basic metabolic panel</td>
<td></td>
</tr>
<tr>
<td>Liver function tests</td>
<td></td>
</tr>
<tr>
<td>Serum calcium</td>
<td></td>
</tr>
<tr>
<td>Serum vitamin B12 and folate</td>
<td></td>
</tr>
<tr>
<td>Thyroid function tests</td>
<td></td>
</tr>
<tr>
<td>Rapid plasma reagin</td>
<td></td>
</tr>
<tr>
<td>HIV in high-risk individuals</td>
<td></td>
</tr>
<tr>
<td>CSF studies if CNS infection is suspected</td>
<td></td>
</tr>
</tbody>
</table>

CSF: cerebrospinal fluid; HIV: human immunodeficiency virus; MCI: mild cognitive impairment

Source: Reference 8

In these visits, include:
• repeat office-based cognitive assessment, especially the modified MMSE, clock-drawing test, and MoCA
• careful history-taking from the patient and reliable informant

Compensating for memory loss. Many individuals with MCI have insight into their cognitive deficits and are interested in making lifestyle changes. Experts recommend:
• moderate exercise (at least 30 minutes per session, 3 times a week)
• cognitively stimulating activities that involve language and psychomotor coordination, such as dancing, crossword puzzles, and volunteer work.

Potentially helpful tools include calendars, reminder notes, electronic cuing devices, pill boxes, and “speed-dial” telephones. Encourage patients to participate in local senior organizations and to use community resources.

Medications—yes or no? Cholinesterase inhibitors, rofecoxib, and vitamin E have not been shown to prevent MCI from progressing to AD. Thus, insufficient evidence

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Cleveland Clinic and Lerner
College of Medicine
of Case Western University

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University of Minnesota

Look for the “Meeting of the Minds” icon at CurrentPsychiatry.com. Click on “Download recent Webcasts”
exists to recommend medications for patients with MCI.
Donepezil has shown possible short-term benefits, however, and patients may choose to try this medication. Some find comfort in seeking this “extra time” to make decisions about advanced directives, attend to estate and will issues, and optimize relationships while they have only mild cognitive deficits and possess decision-making capacity.

Donepezil. The Alzheimer’s Disease Cooperative Study—supported by the National Institute on Aging—was designed to determine whether daily doses of donepezil or vitamin E can delay or prevent progression of aMCI to AD. In the double-blind, placebo-controlled, parallel group study, 769 patients with aMCI were randomly assigned to receive donepezil, 10 mg/d; vitamin E, 1,000 IU bid; or placebo for 3 years.

Overall progression to AD was 16% per year, and the 3-year risk of progression was the same in all 3 groups. Donepezil therapy was associated with a reduced rate of progression to AD compared with placebo during the first year of treatment. Donepezil’s benefit was evident among ApoE e4 carriers at 2-year follow-up, but none of the 3 groups showed statistically significant differences after 3 years. Vitamin E showed no effect on AD progression throughout the study.

Rivastigmine. A randomized, placebo-controlled trial in which 1,018 MCI patients received rivastigmine or placebo for 4 years found no statistically significant benefit of rivastigmine on AD progression.

Galantamine. Two international randomized, double-blind, placebo-controlled trials failed to show a statistically significant benefit of galantamine in slowing progression from aMCI to AD. MRI data from one of these studies suggested that galantamine may have reduced the rate of brain atrophy over a 2-year period.

Rofecoxib. Epidemiologic studies indicate that anti-inflammatory drugs may reduce the risk of developing AD, but the COX-2 inhibitor rofecoxib did not delay progression to AD among aMCI patients in a large, placebo-controlled trial.

Related Resources

Drug Brand Names
- Benztropine -Cogentin
- Oxybutynin - Ditropan
- Cyclobenzaprine - Flexeril
- Rivastigmine - Exelon
- Donepezil - Aricept
- Galantamine - Razadyne
- Rofecoxib - Vioxx
- Sertraline - Zoloft

Disclosures
Dr. Goveas and Dr. Dixon-Holbrook report no financial relationship with any company whose products are mentioned in this article or with manufacturers of competing products. Dr. Kerwin is a consultant to Pfizer and a speaker for Pfizer and Novartis. Dr. Antuono receives research support from Eisai, Pfizer, and Elan and is a speaker for Pfizer and Forest Pharmaceuticals.

Educate patients and family members about supportive nonpharmacologic treatments and cholinesterase inhibitors. The Alzheimer’s Association, National Institute on Aging, and local department of aging agencies offer useful resources (see Related Resources).

CASE CONTINUED
Dealing with uncertainty
Mr. R and his wife are unsettled by his MCI diagnosis. They prefer to take a “wait and watch” approach, decline initiation of a cholinesterase inhibitor, and agree to adhere to nonpharmacologic interventions you discussed. You schedule a follow-up visit in 6 months and encourage them to call you with questions.

References

continued
Mild cognitive impairment

Clinical Point
Cholinesterase inhibitors, rofecoxib, and vitamin E have not been shown to prevent MCI from progressing to Alzheimer’s

Bottom Line
Consider mild cognitive impairment (MCI) when patients age >50 present with memory loss. Assess cognition with the MoCA and modified MMSE; noticeable activities of daily living decline points toward dementia. Neuropsychological tests predict risk of MCI progressing to dementia. Treat comorbid depression, and discuss neuroprotective treatments with patient and family. Repeat test battery every 6 to 12 months. Research focuses on identifying preclinical AD states and potential targets for disease-modifying therapies.

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