

Early Symptomatic Alzheimer's Disease: *A Patient Care Pathway*

A practice guide for primary care providers and dementia specialists^a, offering guidance on detecting, assessing, diagnosing, and treating mild cognitive impairment and mild dementia due to Alzheimer's disease.

^aDementia specialists also encompass neurologists who specialize in Alzheimer's disease care.

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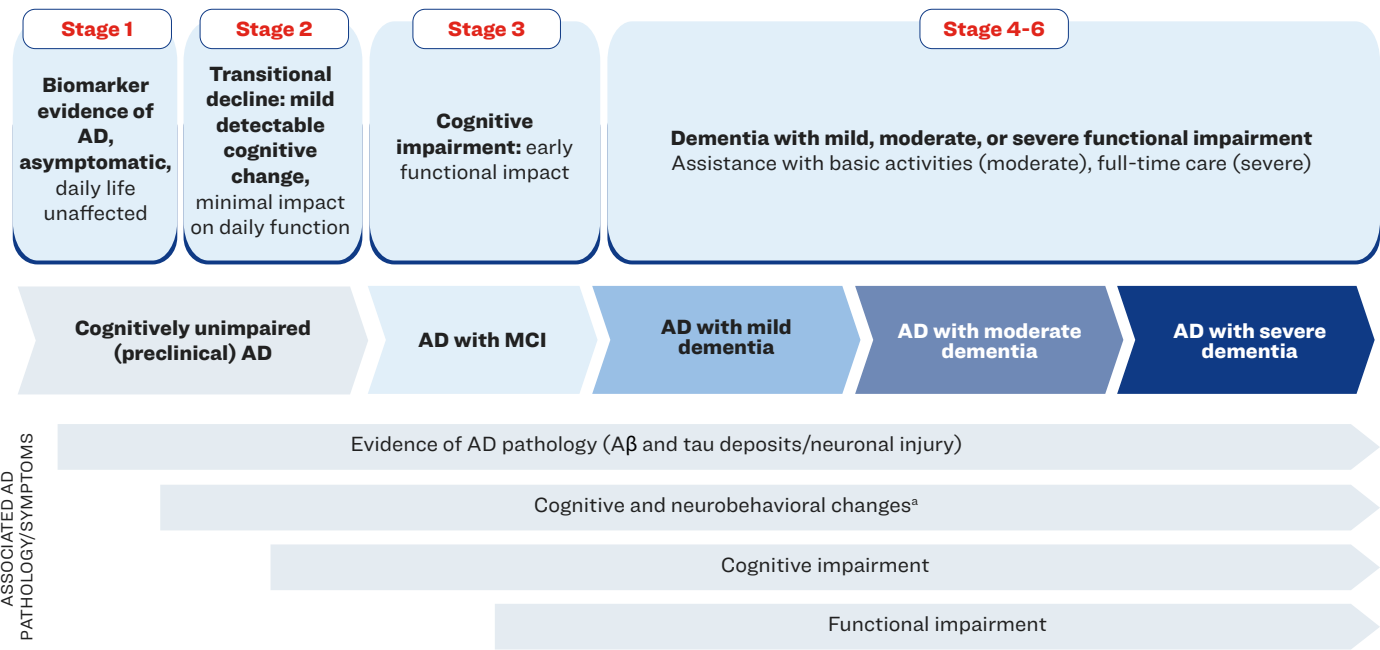
This guide offers a step-by-step framework for dementia specialists and primary care providers to detect, assess, diagnose, and treat early symptomatic Alzheimer's disease. The goal is to adapt the communication to the various steps of the care pathway and optimize patient outcomes.

Early Symptomatic Alzheimer's Disease: What, Why, and How

Alzheimer's disease (AD) is the most common cause of dementia, becoming more prevalent as the global population of people 65 years and older is increasing.¹

It is a progressive, neurodegenerative disease in which patients experience increasing cognitive, functional, and behavioral impairments.¹

Stages within the Alzheimer's disease continuum¹⁻⁵



Note: Time spent in different stages of the disease varies by patient, and the size of the arrows is not equivalent to the duration of each stage of the AD continuum. The biomarker staging by clinical stage presented herein is believed to represent the typical progression in individuals with AD, though considerable variability exists.

^aOne important cognitive and neurobehavioral change to consider is mild behavioral impairment, a clinical construct that describes the emergence of sustained and impactful neuropsychiatric symptoms that may occur in patients ≥ 50 years old prior to cognitive decline and dementia.

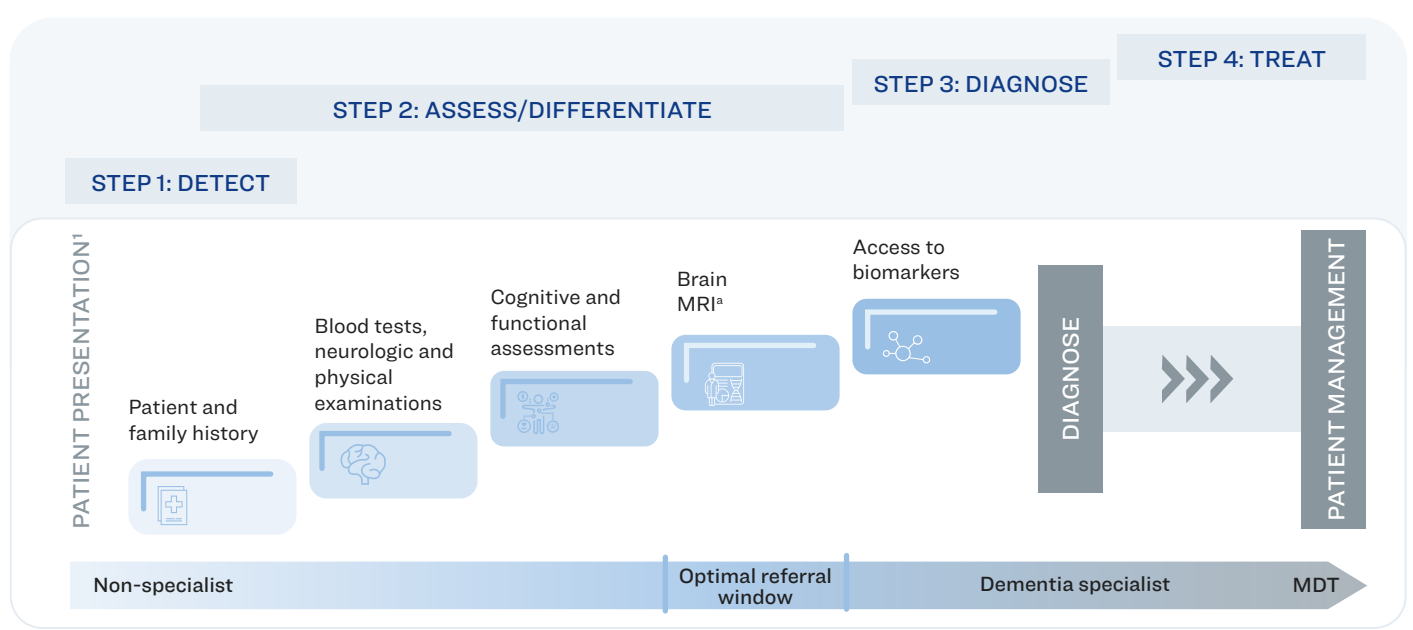
Given the complex nature of AD, a multidisciplinary team approach is necessary to ensure timely and accurate diagnoses for patients suspected of having or living with AD.¹

This approach, which begins with primary care and extends through various specialists, can be seen as a **patient care pathway**.

Why is early diagnosis important?^{1,6}

- Provides clarity for the patient
- Gives patients and families time to plan ahead and access support systems
- Enables early treatment, which can slow disease progression and help maintain independence for longer

Patient Care Pathway



Note: Exact practice for individuals in a collaborative care team may vary based on many factors, including expertise and resources.
^aCT if MRI is not available or contraindicated, used mainly to look for non-AD causes of cognitive impairment.⁷

Goals of the 4 steps^{1,7,8}

- Detect:**
Learn to recognize the most common signs and symptoms of AD.
- Assess and Differentiate:**
Gain insights into the most common assessments. Learn to rule out other conditions, possibly reversible, such as depression or vitamin deficiencies.
- Diagnose:**
Learn how to detect the clinical evidence and biomarker confirmation for a diagnosis of early symptomatic AD.
- Treat:**
Develop an individualized management plan for the patient living with early symptomatic AD.

Recommended^a roles and responsibilities in the patient care pathway¹

● = Primary Care Providers ▲ = Dementia Specialists

Primary care providers	Dementia specialists
• Detect early signs and symptoms of AD¹ • Perform an initial assessment, including risk factors and medical history¹ • Evaluate the differential diagnosis, as other conditions may have similar presentations¹ • May use P-tau217 tests with high diagnostic accuracy to support triage and referral decisions⁸⁻¹⁰	• Further evaluation with clinical and biomarker assessments to determine underlying etiology¹ • Confirm the diagnosis¹ • Establish a treatment plan and engage in shared decision-making with patient and care partners^{1,11,12} • Develop a patient-focused management and follow-up plan¹ • Provide the patient with additional support resources¹

^aThis is based on the review by Porsteinsson et al (2021), which reflects a US practice context; practice may vary regionally.

Step 1: Detecting Alzheimer's Disease

Have you covered everything?¹

Symptoms affecting cognition?

Changes in behavior?

Any psychological changes in state of mind?

Symptoms with a physical trait, eg, affecting gait?

Sleep disorder?

- Detecting early signs and symptoms of AD is the first step of the care pathway.¹
- Early signs of AD are often subtle and vary from person to person. They typically develop gradually as the disease progresses
 - It is important that you pay attention to subtle details and listen closely to inputs from family members or care partners, as they can provide valuable insights for recognizing early signs
 - Being aware of the most common symptoms of early symptomatic AD is important
 - Have you reviewed the patient's medication list as a potential cause of cognitive impairment?

Key symptoms linked to the early stages of Alzheimer's disease include¹:

Category	Symptoms	Practical examples to be aware of
Cognition	• Short-term memory loss • Word-finding difficulties (anomia)	• Forgetting appointments, names, recent events • Frequently misplacing items • Trouble finding exact words
Behavior	• Withdrawal from social activities • Disinhibition and impulsivity	• Inability to participate in meaningful social situations • Poor or decreased judgment
Psychological	• Depression • Mood disturbances • Apathy	• Changes in mood or personality • Loss of initiation • A sense of helplessness and loss of meaning in life
Physical	• Visuospatial problems • Gait impairment	• Frequent falls
Other	• Sleep disorder	• Rapid eye movement disorder (eg, acting out dreams)



Step 2: Assessing and Differentiating Alzheimer's Disease

The second step of the care pathway consists of conducting several assessments to rule out potentially reversible causes, such as depression or vitamin deficiencies, and other causes of mild cognitive impairment.¹ AD can cause a wide range of symptoms, and assessments can include history and physical assessment, cognitive, functional, and behavioral assessments, and laboratory and imaging testing.^{1,13}



Engage care partners: Care partners, such as next of kin, can provide critical insights on cognitive and functional changes over time.¹



History and assessment^{1,7,14}

Risk factors:

- Family history of AD or related dementias in first-degree relatives, age, female sex, *APOE* ε4 carriers, physical inactivity, low educational status, chronic conditions, obesity, and psychiatric conditions

Medical conditions that can affect cognition:

- Eg, hypertension, diabetes, stroke, Parkinson's disease, thyroid dysfunction, speech or hearing impairment, or head trauma

Prescribed medications that can impair cognition:

- Eg, sleep aids, anxiolytics, analgesics, or anticholinergics

Social history:

- Illicit drug use, exposure to heavy metals, or excessive alcohol use



Laboratory analysis¹

Recommended:

- Complete blood cell count
- Blood glucose
- Thyroid-stimulating hormone
- Serum B12 and folate
- Serum electrolytes
- Liver function
- Renal function

Considerations:

- Urinalysis⁷
- Urine culture⁷

Note: This list of tests is not exhaustive and is intended to provide a general overview only.



Physical assessment¹

Mental status

- Assess for depression

Neurologic assessment

- Signs that could indicate a stroke

Diet and nutrition

Review medications

- Assess whether any medications mentioned during the initial assessment could impair cognition

Physical examination

- Blood pressure
- Temperature
- Pulse
- Listen to heart and lungs



Structural imaging¹

Recommended:

- Magnetic resonance imaging (MRI)

Consideration:

- For patients with more established AD symptoms, fluorodeoxyglucose-PET (FDG-PET) may be used

Have you^{1,13}



Confirmed medical and family history?



Reviewed medications for cognitive impairment risks?



Performed laboratory tests to rule out reversible causes?



Conducted a brief clinical assessment to identify cognitive and functional impairment?

Cognitive, functional, and behavioral assessments

Digital Cognitive Assessments (DCAs) are now available alongside traditional paper-based tools. DCAs may offer automated scoring, reduced rater bias, and electronic health record integration.^{15,16}

Type of assessment	Assessment	Items	Time (min)	Scoring system	Sensitivity and specificity	Considerations
Traditional paper-based cognitive assessment	AD8 ¹	8	2-3	Scores >2 signify impairment	Sens: 90% Spec: 68%	Brief assessment for cognitive impairment
	SLUMS ¹⁷⁻¹⁹	11	~7	≥27: Normal cognition ^a 21-26: Mild neurocognitive disorder ^a <21: Dementia ^a	Sens/Spec MCI ^b : 74%/65% Dementia ^b : 93%/96%	Detection of symptoms in early stages of AD; designed to minimize bias due to education
	MMSE ¹	30	5-10	23-24	Sens: 85-100% Spec: 66-100%	Requires payment to use ²⁰
	MoCA ¹	12	10	<26 for MCI or dementia	Sens: 78-100% Spec: 65-94%	Requires certification and payment ²¹
	Mini-Cog ¹	3-item recall + clock	2-3	Recall 2/3 items; clock drawing used to determine presence of cognitive deficits	Sens/Spec comparable to MMSE	Brief assessment; easy to interpret; no training requirements
	IQCODE ¹	16 or 26	10	Scores >3.44 signify impairment	Sens: 76-100% Spec: 65-86%	Measures decline from premorbid level
Digital cognitive assessment	TabCAT-BHA (UCSF; iPad-based) ²²	Multi-domain	Not reported	Composite score; demographically adjusted z-score (< -1.5z = high likelihood of impairment)	Sens: 84% Spec: 85% for MCI (outperforms MoCA at equivalent specificity)	iPad-administered; EHR-integrated; can be administered by medical assistants; studied in a diverse primary care clinic; validated across diverse populations (including English, Spanish, Mandarin, and Cantonese speakers)

^aFor individuals with less than high school education, 25-30: normal cognition; 20-24: mild neurocognitive disorder; <20: dementia.

^bWith at least a high school education.



Type of assessment	Assessment	Items	Time (min)	Scoring system	Sensitivity and specificity	Considerations
Digital cognitive assessment	Linus Health CCE/DCR™ + DCTclock™ (Linus Health; tablet-based) ^{23,24}	Memory & executive function	DCR™: ~3	Automated; machine learning-based scoring of clock drawing process and word recall	Strong psychometric properties for identifying possible MCI and early dementia vs. MMSE	Tablet-based; includes Digital Clock Drawing Test (DCTclock™) + 3-word recall; studied across 7 racially and socioeconomically diverse primary care clinics (suburban and urban); captures process measures (drawing efficiency, information processing speed, motor performance)
	CogState Brief Battery (CBB) (CogState; computer/tablet) ²⁵	4 domains: processing speed, attention, working memory, learning	~10	Composite scores for psychomotor/attention and learning/working memory; -1 SD cut for MCI; -1.7 SD cut for AD	High test-retest reliability (r=0.95); large effect sizes in MCI (g=2.2) and AD (g=3.3) for learning composite	Extensively validated; used in AD clinical trials; best suited for specialty care or clinical trial settings; sensitive to preclinical and prodromal AD
	MyCog (Mobile app) ^{26,27}	Multi-domain	~10	Automated scoring; likelihood of impairment output	Designed for primary care older adults; iterative co-design with patients and PCPs Sens: 79% Spec: 82%	Smartphone/tablet app; designed specifically for primary care workflow integration; co-designed with older adult patients; pragmatic RCT underway in underserved US communities
Traditional paper-based functional assessment	FAQ ¹	10 categories	5 ^c	0-3 scale (0=normal; 3=dependent)	Sens: 90% Spec: 83%	Highly reliable assessment
Traditional paper-based behavioral assessment	GDS ¹	15 or 30	5-10	≥5 suggestive of depression; ≥10 significant of depression ^c	No data available	Reliable assessment for depression (often associated with dementia; symptoms may overlap)
	NPI-Q ¹	12	5	0-3 scale (0=none; 3=severe)	Sens: 86% Spec: 76%	Brief and reliable assessment

^cPersonal communication.
Note: This is not a comprehensive list of available cognitive assessment tools. Selection should be based on clinical context, patient population, and available resources. Cognitive assessment results should always be interpreted in conjunction with a comprehensive clinical workup. Products/company names mentioned herein are the trademarks of their respective owners.

Step 3: Diagnosing Alzheimer’s Disease Pathology

Once the assessment is completed and other conditions have been ruled out, the third step of the care pathway is to diagnose AD pathology by confirming the presence of neuropathological hallmarks such as amyloid and tau with or without neurodegeneration.^{2,9,28}

Note:
Access to these tools and their reimbursement status can differ depending on where you live. It is important to verify the availability and local practice environment.



P-tau217 blood tests: triaging and confirmatory test modality^{a,9,10,28,29}

P-tau217 blood tests can confirm AD pathology and are now positioned as the triaging and substitute test modality^a for confirming AD biology in primary and specialty care settings.^{9,10,28,29}

- High-performing, validated P-tau217 assays can confirm AD pathology and serve as a triaging test or, at ≥90% sensitivity/specificity, a substitute for amyloid PET or CSF testing^{9,28}
- P-tau217 testing may be initiated by a PCP or specialist before or after referral^{8,28}
- Not all blood tests are equivalent — emphasize use of high-performing, validated P-tau217 tests²⁹

Note: CSF and PET biomarkers may still be used in instances of indeterminate results or when further pathological characterization is needed.^{2,9,28} ^aFor use as a triaging test before confirmatory amyloid PET or CSF testing, a BBM test should have sensitivity of ≥90% with a specificity of ≥85% in primary care and ≥75% in specialized care for memory disorders. For use as a substitute for amyloid PET imaging or CSF AD biomarker testing, a BBM test should have both sensitivity and specificity ≥90%.^{28,29}



Imaging biomarkers^{1,2,9,28}

Amyloid PET scanning

- Visualizes β-amyloid plaques in the brain using specific tracers
- **Considerations:** May be reserved for cases of indeterminate blood biomarker results or to identify select patients where further confirmation is required
- Should be used alongside clinical assessments; PET scans alone cannot definitively diagnose AD
- **Limitations:** Accuracy depends on observer experience; amyloid PET lacks a clear cutoff value for normal vs. pathologic findings



CSF biomarkers^{1,2,9,28}

Cerebrospinal fluid biomarker analysis

- Can detect changes in AD biomarkers, such as amyloid (Aβ42 and Aβ42/40) and tau levels (P-tau and T-tau), indicative of amyloid accumulation and neurodegeneration
- **Consideration:** Elevated T-tau is not specific to AD
- There are appropriate use criteria available to help identify suitable patients for CSF biomarker analysis
- May be used at the discretion of the ordering provider for indeterminate blood biomarker results

AD diagnosis integrates clinical, cognitive, and functional evidence, with concordant biomarker findings across P-tau217 tests, amyloid PET, and CSF testing, interpreted alongside clinical presentation.^{1,2,9,28}



Step 4: Treating Alzheimer’s Disease

The final and fourth step of the care pathway is to determine the best management plan, including possible treatment options, for the patient. This should be personalized and based on earlier steps to address specific risk factors and symptoms.

Diet and lifestyle changes^{30,31}

- A plant-based diet rich in fruits, vegetables, whole grains, legumes, soy, seeds, spices (such as curcumin), and nuts; and low in refined carbohydrates, sweeteners, and harmful fats
- It is recommended to stop smoking and to keep alcohol consumption to a minimum

Cognitive training^{32,33}

- Simple memory exercises (eg, remembering past events or naming familiar objects and people), word games and puzzles, and practice with planning daily activities
- The patient can be reminded of details such as upcoming events, using conversational cues, a calendar, or other external memory aids

Exercise^{14,30}

- Physical activities like walking are cognitively engaging
- A randomized clinical trial found that exercise and relevant lifestyle changes (eg, diet and stress management) may improve cognition in patients with MCI or early dementia due to Alzheimer’s disease

Care partner training³⁴

Educational programs are available for care partners. They help improve patients’:

- Cognitive outcomes
- Functional outcomes
- Health-related quality of life

Pharmacologic treatment^{5,11,12,14}

Based on the AD staging and eligibility factors, clinicians can prescribe:

- Symptomatic treatments** that may help lessen or stabilize symptoms for a limited time by affecting certain chemicals involved in carrying messages among and between the brain’s nerve cells^{5,14}
- Disease-modifying therapies — amyloid-targeting therapies (ATTs):** ATTs can slow the decline in memory, thinking, and function in patients living with early AD by influencing the underlying biological processes of the disease^{5,11,12}
- Currently approved ATTs target amyloid plaques and are indicated for patients with MCI or mild dementia due to AD who have confirmed amyloid pathology^{5,11,12}
- Shared decision-making** with the patient and care partners is essential when discussing ATT initiation, including a thorough discussion of benefits, risks, and monitoring requirements^{5,11,12}

APOE ε4 genotype testing and ARIA risk^{11,12}

- APOE ε4 genotype testing is recommended in the context of ATT treatment decision-making, not as a routine diagnostic tool
- APOE ε4 carriers (especially homozygotes) are at increased risk of amyloid-related imaging abnormalities (ARIA-E and ARIA-H) when on ATT treatment
- Discuss ARIA risk and monitoring protocols with patients and care partners before initiating ATT
- APOE ε4 status should inform the benefit-risk discussion and MRI safety monitoring frequency during ATT treatment

Keep in mind¹:

The goal of the management plan is to preserve the patient’s cognitive abilities and for the patient to maintain independence in their daily activities for as long as possible. Regular, ongoing monitoring is important. In early symptomatic AD, non-pharmacologic therapies are commonly used, alongside medications when appropriate.

Abbreviations & References

Abbreviations:
Aβ=amyloid beta; AD=Alzheimer’s disease; AD8=Ascertain Dementia 8-item; APOE=apolipoprotein E; ARIA-E=amyloid-related imaging abnormalities-edema/effusion; ARIA-H=amyloid-related imaging abnormalities-hemosiderin deposition; ATT=amyloid-targeting therapy; BBM=blood-based biomarker; CCE=The Linus Health Core Cognitive Evaluation; CSF=cerebrospinal fluid; CT=computed tomography; DCA=Digital Cognitive Assessment; DCR=Digital Clock and Recall; EHR=electronic health record; FAQ=Functional Activities Questionnaire; FDG-PET=fluorodeoxyglucose-positron emission tomography; GDS=Geriatric Depression Scale; IQCODE=Informant Questionnaire on Cognitive Decline in the Elderly; MCI=mild cognitive impairment; MDT=multidisciplinary team; Mini-Cog=Mini Cognitive Assessment Instrument; MMSE=Mini-Mental State Examination; MoCA=Montreal Cognitive Assessment; MRI=magnetic resonance imaging; NPI-Q=Neuropsychiatric Inventory Questionnaire; PCP=primary care physician; PET=positron emission tomography; P-tau=phosphorylated tau; RCT=randomized clinical trial; SD=standard deviation; Sens=sensitivity; SLUMS=Saint Louis University Mental Status Examination; Spec=specificity; TabCAT-BHA=Tablet-Based Cognitive Assessment Tool-Brain Health Assessment; T-tau=total-tau; UCSF=University of California, San Francisco.

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Notes