WYOMING PRIMARY CARE SYMPOSIUM

Dementia in Primary Care

Recognizing the types, reading the biomarkers, choosing the imaging, and treating with intention



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Disclosures

I have no financial disclosures or conflicts of interest.



Learning objectives

Distinguish the major dementia types

Alzheimer's, vascular, Lewy body, frontotemporal, LATE, and mixed pathology, by their clinical signatures.

Use biomarkers wisely

Order and interpret the FDA-cleared blood tests, CSF, and when to escalate to PET.

Choose the right imaging

Know what to look for on MRI, and when FDG-PET, amyloid/tau PET, or DaTscan adds value.

Match treatment to type

Symptomatic drugs, anti-amyloid therapy eligibility and safety, behavioral symptoms, and care support.

Why this matters in primary care

7.4M

Americans 65+ living with Alzheimer's dementia in 2026

~10,300

Wyomingites 65+ with Alzheimer's dementia (~1,300 in Natrona County)

~45%

of dementia linked to 14 modifiable risk factors (Lancet 2024)

In a frontier state, primary care is the dementia specialist. Long drives to neurology and geriatrics, an older rural population, and heavy reliance on family caregivers mean the diagnosis, the conversation, and most of the follow-up happen in our clinics.

References: 1, 2, 3

Syndrome vs. disease: getting the language right

Dementia is a clinical syndrome; Alzheimer's, Lewy body, TDP-43 and vascular injury are the causes

Subjective decline

Patient or family notices change; normal testing

Mild cognitive impairment

Objective decline, independent in daily function

Dementia

Decline significant enough to impair independence in daily activities

Increasing impairment in daily function →

The functional line separates MCI from dementia. Ask about finances, medications, driving, cooking, and technology, and ask an informant.

Biology can precede symptoms by 15 to 20 years. The 2024 Alzheimer's Association revised criteria define AD biologically, but diagnosing a symptomatic patient in clinic still starts with the syndrome.

References: 4, 5, 6

The primary care cognitive evaluation



History

Informant, onset, tempo, first symptom, IADLs



Brief testing

Mini-Cog, MoCA, or SLUMS; depression screen



Medication review

Anticholinergics, benzodiazepines, opioids, sleep aids



Labs

B12, TSH, CBC, CMP; syphilis, HIV if risk



Imaging

MRI brain preferred; CT if MRI not feasible

The first symptom points to the pathology. Memory first suggests AD or LATE. Behavior or language first suggests FTD. Hallucinations, fluctuations, or dream enactment suggest Lewy body disease. Stepwise decline, gait change, and slowed processing suggest vascular disease. Weeks-to-months decline is a rapidly progressive dementia and needs urgent workup.

References: 7, 8, 9, 10, 11

Rule out the contributors and the mimics first

Delirium
Acute, fluctuating attention; look for infection, meds, retention, constipation

Depression
Poor effort, 'I don't know' answers; treat and retest

Medications
Anticholinergic load, sedatives, opioids, polypharmacy

Sleep apnea
Daytime sleepiness, snoring; treatment can improve cognition

Sensory loss
Hearing and vision loss mimic and worsen impairment

Normal pressure hydrocephalus
Gait first, then urinary urgency and cognitive slowing

Metabolic / endocrine
B12 deficiency, hypothyroidism, hypercalcemia, hyponatremia

Alcohol and substances
Alcohol-related brain injury, thiamine deficiency, cannabis

References: 7, 11, 12

PART 1

Types of Dementia

Clinical signatures, pathology, and what makes each one different



The dementia types at a glance

Type	Typical onset	First / hallmark features	Course
Alzheimer's disease	65+ (earlier forms exist)	Episodic memory loss, word-finding, later visuospatial	Gradual, steady
Vascular	Any age with risk factors	Slowed processing, executive dysfunction, gait change	Stepwise or gradual
Lewy body (DLB / PDD)	50s to 80s	Fluctuations, visual hallucinations, RBD, parkinsonism	Gradual, fluctuating
Frontotemporal (FTD)	45 to 65	Behavior or personality change, or language loss	Gradual, often faster
LATE	Usually 80+	Slow amnestic decline, few other domains early	Slower than AD
Mixed	Oldest old	Overlapping features; the rule rather than the exception	Variable

References: 1, 13, 14, 23

Alzheimer's disease: the clinical picture

Typical (amnestic) presentation

- Repeats questions and stories; forgets recent conversations
- Poor delayed recall that does not improve with cues
- Word-finding pauses, getting lost in familiar places later
- Relatively preserved social graces early ('social facade')
- Anosognosia is common: family is more worried than patient

Atypical presentations to recognize

- Posterior cortical atrophy: visual-spatial problems, trouble reading, 'needs new glasses' repeatedly
- Logopenic aphasia: word-finding pauses, impaired repetition of sentences
- Frontal/dysexecutive variant: planning and judgment first
- Early-onset AD (under 65): more often atypical; screen family history

References: 6, 15, 24

Alzheimer's biology behind the biomarkers

Amyloid first, tau follows, neurodegeneration drives symptoms



Amyloid

Aβ plaques accumulate 15-20 years before symptoms

→ **Measured by:** Amyloid PET, CSF Aβ42/40, plasma p-tau217 ratio

Tau

Neurofibrillary tangles spread from medial temporal lobe

→ **Measured by:** Tau PET; p-tau217 and p-tau181 track amyloid-related tau

Neurodegeneration

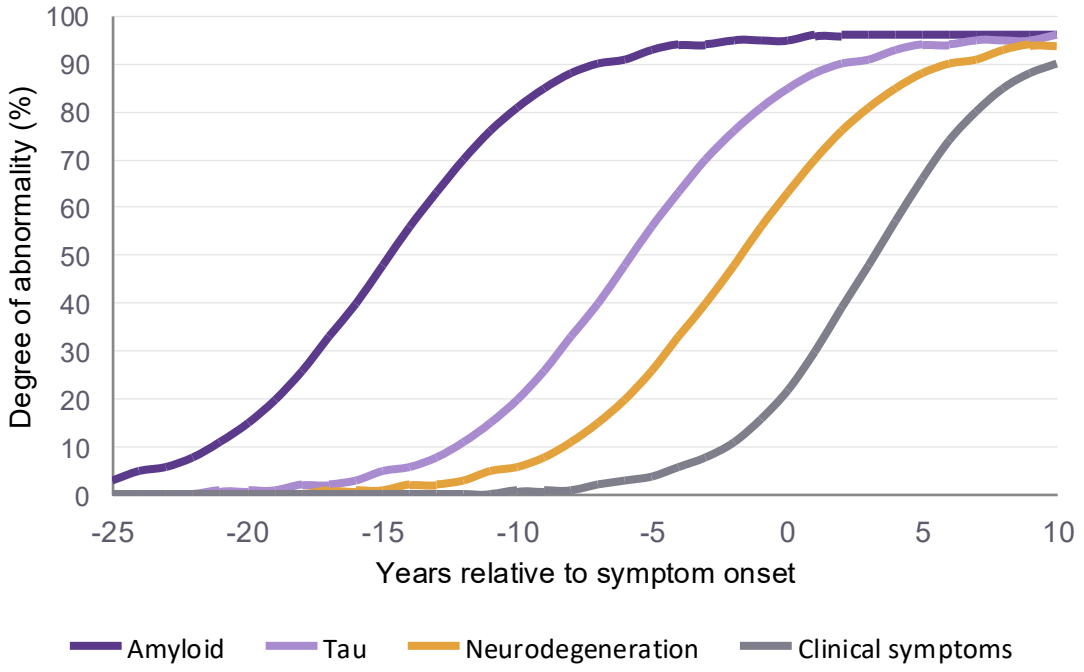
Synapse and neuron loss produce symptoms

→ **Measured by:** MRI atrophy, FDG-PET, plasma NfL (non-specific)

References: 4, 16

The Alzheimer's biomarker timeline

Schematic: biomarkers become abnormal in sequence, years before symptoms



What it means

- Amyloid is abnormal first, often 15-20 years before symptoms
- Tau and neurodegeneration track symptoms more closely
- Why anti-amyloid therapy targets the earliest symptomatic stage
- Why we don't test people without symptoms

References: 16

Vascular cognitive impairment

Clinical clues

- Slowed thinking, poor attention and planning
- Retrieval problems: cues help recall
- Gait change, falls, urinary urgency
- Apathy, depression, emotional lability
- Stepwise decline after strokes, or insidious with small vessel disease

Imaging findings

- White matter hyperintensities (Fazekas grade 2-3)
- Lacunes in basal ganglia, thalamus
- Strategic infarcts (thalamus, angular gyrus)
- Microbleeds: deep pattern suggests hypertension; lobar pattern suggests amyloid angiopathy

Management

- Aggressive vascular risk control: BP, lipids, diabetes, smoking
- SPRINT MIND: intensive BP control reduced MCI
- Antithrombotic only for its usual indication
- Cholinesterase inhibitors: modest, off-label benefit
- Exercise and gait/fall programs

References: 14, 17, 18

Dementia with Lewy bodies

2017 McKeith consensus criteria

Core clinical features

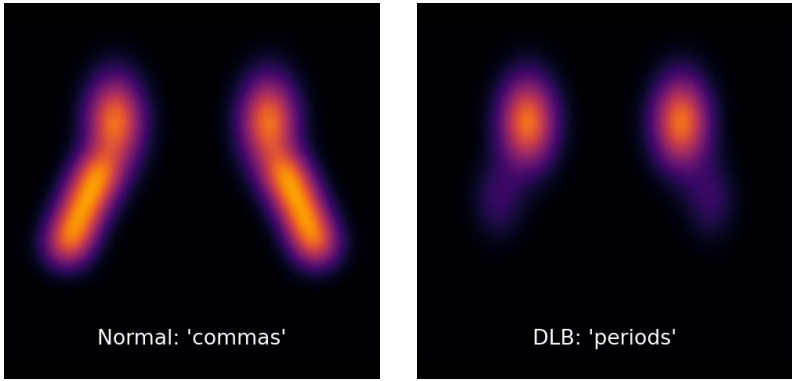
Fluctuating cognition
Good days and bad days, staring spells, daytime drowsiness

Visual hallucinations
Recurrent, well-formed: people, children, animals

REM sleep behavior disorder
Acting out dreams; may precede dementia by years

Parkinsonism
Bradykinesia, rigidity; tremor less prominent

DaTscan (illustration)



Probable DLB: 2+ core features, or 1 core + 1 indicative biomarker (abnormal DaTscan, low cardiac MIBG, or PSG-confirmed RBD). Emerging: α-synuclein seed amplification.

References: 19

DLB vs. Parkinson's disease dementia

The one-year rule: dementia before or within one year of parkinsonism = DLB. Dementia arising in established Parkinson's disease (more than one year of motor symptoms) = PDD. Same pathology, different sequence.

Treatment responses to know

- Cholinesterase inhibitors often work better than in AD, including for hallucinations
- Rivastigmine is FDA-approved for PDD
- Levodopa: modest motor benefit, may worsen hallucinations
- Treat orthostatic hypotension and constipation

Safety warnings

- Severe neuroleptic sensitivity: avoid haloperidol and typical antipsychotics
- If an antipsychotic is unavoidable: low-dose quetiapine or pimavanserin (PDD psychosis)
- Melatonin first-line for RBD; clonazepam with caution
- High fall risk: PT and home safety

References: 19, 20, 21, 22

Frontotemporal dementia

The most common dementia under age 60, and the most often misdiagnosed as psychiatric

Behavioral variant (bvFTD)

- Disinhibition: socially inappropriate, impulsive
- Apathy or inertia
- Loss of empathy or sympathy
- Compulsive or ritualistic behaviors
- Hyperorality: sweets, overeating
- Executive deficits; memory relatively spared
- 3 of 6 features = possible bvFTD (Rascovsky 2011)

Primary progressive aphasias

- Nonfluent/agrammatic: effortful, halting speech, grammar errors; often tau
- Semantic: fluent but empty speech, loses word meaning; usually TDP-43
- Logopenic: word-finding pauses, poor repetition; usually AD pathology
- Genetics: family history in up to ~40%; C9orf72, GRN, MAPT
- Overlap with ALS and parkinsonian syndromes

References: 23, 24

FTD: diagnosis and management pearls

Imaging

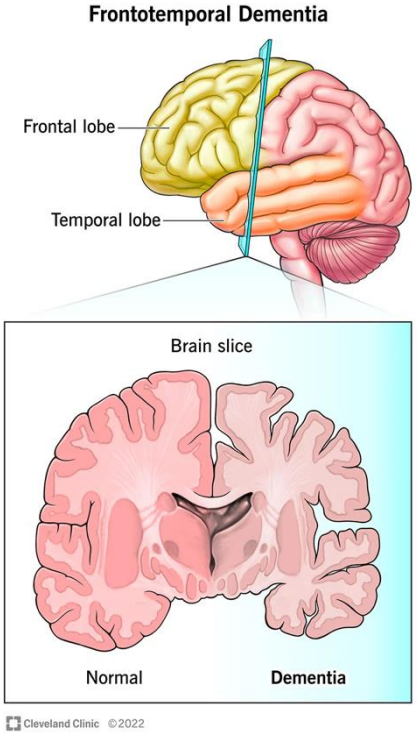
Frontal and/or anterior temporal atrophy, often asymmetric. FDG-PET shows frontal and anterior temporal hypometabolism and is covered by Medicare to separate FTD from AD.

Medications

No FDA-approved therapy. SSRIs and trazodone can reduce disinhibition and compulsions. Cholinesterase inhibitors do not help and can worsen behavior. Memantine did not show benefit.

Safety and family

Protect finances early, address driving and firearms, plan for workplace issues, and connect families with the Association for Frontotemporal Degeneration (AFTD).



References: 23, 25. Illustration: © 2022 Cleveland Clinic

LATE: the under-recognized AD mimic

Limbic-predominant age-related TDP-43 encephalopathy, named in 2019

What it is

Misfolded TDP-43 protein in the amygdala, then hippocampus, then middle frontal cortex; often with hippocampal sclerosis.

Who gets it

Mostly people over 80. Found at autopsy in roughly a quarter to a half of people in their 80s and beyond.

Why it matters

Mimics AD clinically. A meaningful share of patients diagnosed with 'AD' are amyloid-negative, and LATE is a leading explanation.

Co-pathology

Frequently coexists with AD; the combination causes faster decline than either alone.

References: 13, 14, 26

LATE vs. Alzheimer's disease in clinic

Feature	LATE	Alzheimer's disease
Typical age	Usually 80+	Usually 65 to 85
Cognitive profile	Relatively pure amnestic syndrome	Amnestic plus language, visuospatial, executive
Rate of decline	Slow, gradual	Faster, steadier progression
MRI	Disproportionate hippocampal / medial temporal atrophy	Hippocampal plus temporoparietal atrophy
FDG-PET	Medial temporal and inferior temporal hypometabolism	Temporoparietal and posterior cingulate
Amyloid biomarkers	Negative (unless AD co-pathology)	Positive
Anti-amyloid therapy	Not indicated	Candidate if early stage and eligible



Clinical clue: an 85-year-old with slow memory loss, marked hippocampal atrophy, and a negative amyloid blood test. There is no TDP-43 biomarker yet.

References: 13, 26

Mixed dementia and don't-miss causes

Mixed pathology is the norm

- Most brains over age 80 have 2 or more pathologies at autopsy
- Common combinations: AD + vascular, AD + Lewy body, AD + LATE
- Each added pathology lowers the threshold for dementia
- A positive amyloid test does not exclude a second process
- Treat what is treatable in each: vascular risk, Lewy-related symptoms

Rapidly progressive: act fast

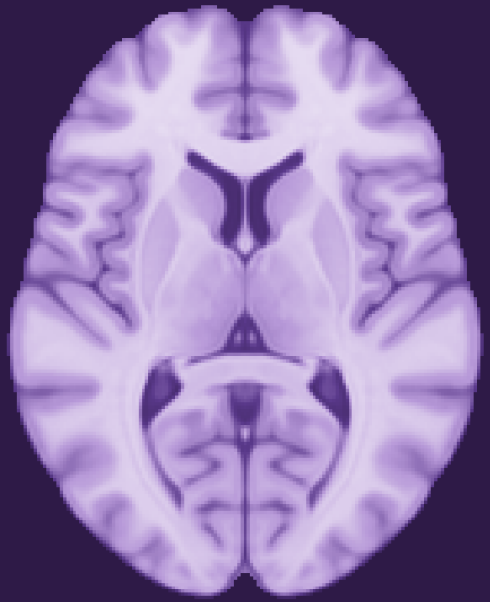
- Decline over weeks to months: urgent evaluation
- Prion disease (CJD): myoclonus, ataxia; MRI DWI ribboning, RT-QuIC
- Autoimmune encephalitis: seizures, psychiatric features; treatable
- Also: CNS lymphoma, infection, subdural hematoma, toxic-metabolic
- Atypical parkinsonism: PSP (falls, vertical gaze), CBS (asymmetric apraxia)

References: 14, 27

PART 2

Biomarkers

Blood, CSF, and genetics: what to order, and what the result really means



Fluid biomarkers for Alzheimer's pathology

Plasma (blood)

- p-tau217 is the most accurate single marker
- p-tau217/Aβ42 ratio or combined algorithms improve accuracy
- Now FDA-cleared; widely available through reference labs
- Best used in symptomatic patients

Cerebrospinal fluid

- Aβ42/40 ratio, p-tau181/Aβ42 ratio
- Well-validated; equivalent to amyloid PET
- Requires lumbar puncture
- Also useful for prion (RT-QuIC), inflammation, α-synuclein

Genetics & other

- APOE genotype: risk, not diagnosis; required before anti-amyloid therapy
- NfL: neurodegeneration of any cause
- GFAP: astrocyte activation
- Genetic panels for early-onset or strong family history

References: 15, 28, 29

FDA-cleared AD blood tests (Sept 2026)

Test	Cleared	What it measures	Intended use
Lumipulse G pTau217/β-Amyloid 1-42 (Fujirebio)	May 2025	Ratio of p-tau217 to Aβ42	Symptomatic adults 55+; aid to diagnosis
Elecsys pTau181 (Roche / Lilly)	Oct 2025	p-tau181	Primary care rule-out before referral
PrecivityAD2 (C2N Diagnostics)	Aug 2026	Mass spectrometry: p-tau217 ratio + Aβ42/40	Symptomatic adults 40+
Elecsys pTau217 (Roche / Lilly)	Aug 2026	p-tau217 (single biomarker)	Symptomatic adults 55+; likely vs. unlikely amyloid



None are screening tests. All are cleared only for people with cognitive symptoms, and every result must be interpreted with the clinical evaluation.

References: 31, 32, 33

Using blood biomarkers well in primary care

Right patient	Objective cognitive impairment after a standard workup. Not for the worried well or asymptomatic family members.
Pretest probability matters	Positive predictive value falls when prevalence is low; negative results are most reliable in lower-risk patients.
Known confounders	Chronic kidney disease can raise p-tau levels; very advanced age and co-pathology complicate interpretation.
Indeterminate results	Two-cutoff tests leave a gray zone. Refer for CSF or amyloid PET if the answer would change management.
Positive ≠ explanation	Amyloid pathology is common in older adults; still consider vascular, Lewy, LATE contributions.

References: 7, 28, 29, 30

A primary care pathway for blood biomarkers

Symptomatic patient
Standard workup done: informant, testing, labs, meds, MRI

Blood biomarker
If result would change management

- Counsel before ordering: what would a positive or negative result mean?
- Check kidney function; CKD can raise p-tau
- Never for asymptomatic screening

Negative
AD pathology unlikely. Look for vascular, Lewy, FTD, LATE, mood, medication causes.

Indeterminate
Refer for CSF or amyloid PET if it would change management.

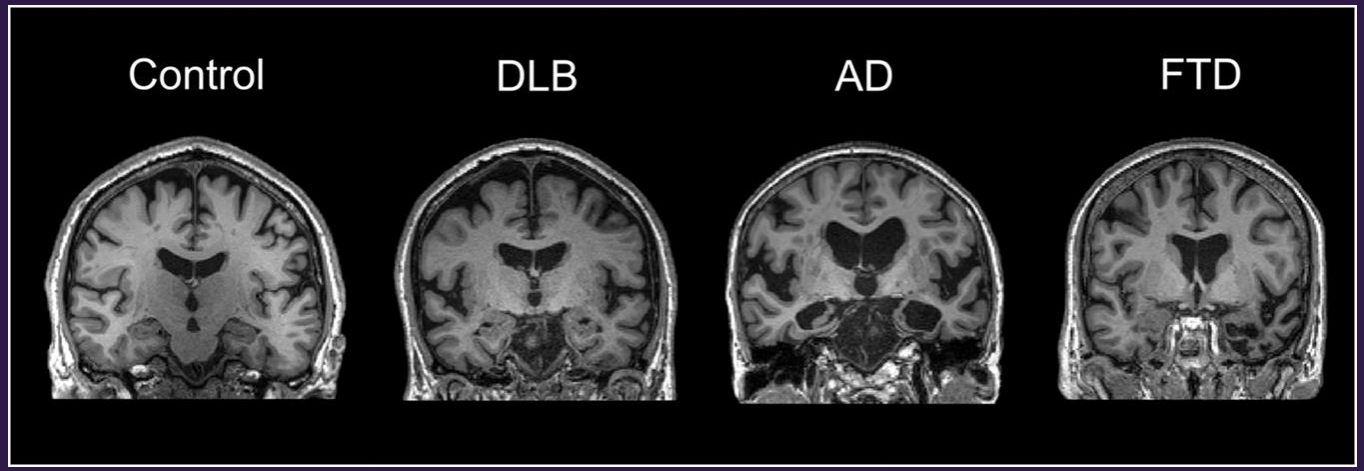
Positive
Supports AD. Disclose, start symptomatic care; if MCI/mild, refer promptly for anti-amyloid evaluation.

Every path: non-drug care, safety planning, and caregiver support.

References: 7, 30

PART 3

Imaging

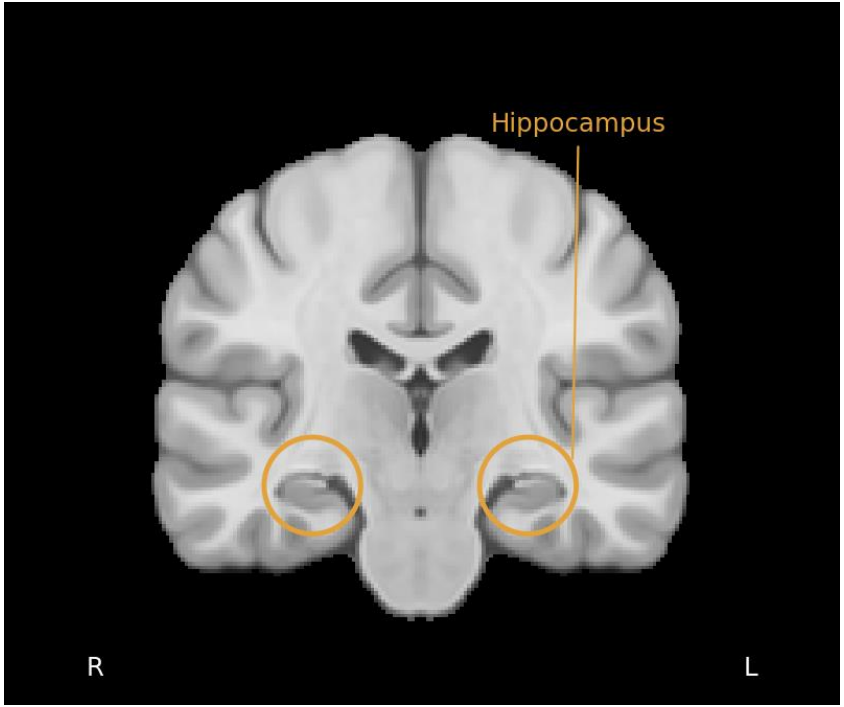


Structural and molecular imaging: what each test can and cannot tell you

Structural imaging: MRI is the workhorse

What to ask the radiologist for

- MRI brain without contrast, with T1 volumetric, FLAIR, DWI, and SWI/GRE
- Comment on hippocampal volume (MTA score) and regional atrophy
- White matter burden (Fazekas grade) and lacunes
- Microbleed count and distribution; superficial siderosis
- CT if MRI is contraindicated or unavailable

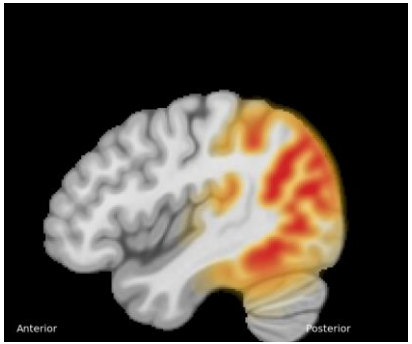


Coronal T1, normal template: grade MTA (0-4) here

References: 34, 35, 36

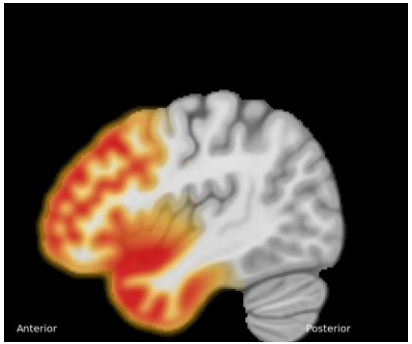
Where each disease strikes

Typical regions of atrophy (MRI) and hypometabolism (FDG-PET); schematic overlays on a normal template brain



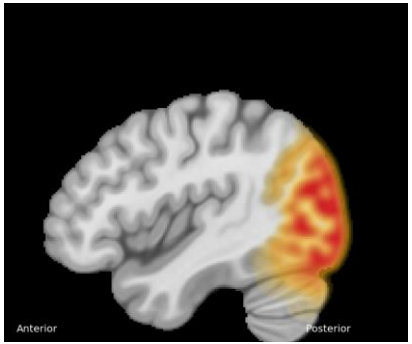
Alzheimer's

Temporoparietal cortex, posterior cingulate, hippocampi



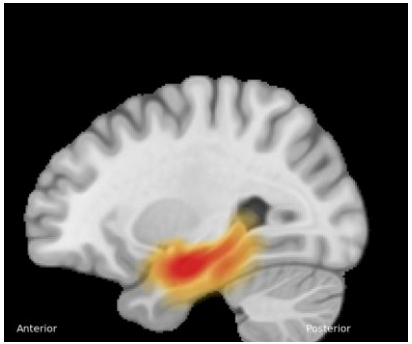
Frontotemporal

Frontal and anterior temporal lobes, often asymmetric



Lewy body

Occipital hypometabolism; medial temporal lobes relatively spared



LATE

Disproportionate hippocampal / medial temporal involvement

Also: WMH and lacunes → vascular | ventricles out of proportion to atrophy → NPH | midbrain 'hummingbird' → PSP

References: 19, 23, 26, 36

Molecular and functional imaging

Test	What it shows	Best use	Coverage notes
FDG-PET	Regional glucose metabolism	AD vs FTD; atypical presentations; LATE pattern	Medicare covers for AD vs FTD question
Amyloid PET	Fibrillar amyloid plaques	Confirm amyloid before anti-amyloid therapy; amyloid removal monitoring	Medicare coverage expanded in 2023
Tau PET	Tau tangle burden and spread	Staging, prognosis; specialty and research use	Limited clinical coverage
DaTscan (SPECT)	Presynaptic dopamine transporter	DLB vs AD; parkinsonism evaluation	Covered; order via neurology in most settings
Cardiac MIBG	Cardiac sympathetic denervation	Supports DLB	Rarely available in the US

References: 19, 37, 38

Putting it together: diagnostic signature by type

Type	MRI	FDG-PET	Key biomarker
Alzheimer's	Hippocampal, parietal atrophy	Temporoparietal, posterior cingulate	Amyloid + (blood, CSF, PET)
Vascular	WMH, lacunes, infarcts	Patchy, variable	None specific; MRI is key
Lewy body	Relative hippocampal sparing	Occipital; cingulate island sign	Abnormal DaTscan; α-syn assays
FTD	Frontal / anterior temporal	Frontal, anterior temporal	Amyloid negative; genetics
LATE	Marked hippocampal atrophy	Medial / inferior temporal	Amyloid negative; no TDP-43 test yet
NPH	Ventriculomegaly, DESH	Not routinely used	High-volume tap test

References: 12, 19, 23, 26, 36

PART 4

Treatment

Symptomatic therapy, disease-modifying therapy, behavior, and care



The foundation: non-drug care for every patient

Exercise

Aerobic and resistance training; balance work to prevent falls

Vascular risk

BP, lipids, diabetes, smoking cessation, alcohol reduction

Hearing and vision

Hearing aids and cataract care; social engagement

Sleep

Treat sleep apnea; avoid sedative hypnotics

Safety

Driving, firearms, finances, medication supervision, wandering

Care partner

Education, respite, support groups, caregiver health

References: 3, 51, 52

Symptomatic medications

Drug	Starting → target dose	Indication	Key adverse effects
Donepezil	5 mg daily → 10 mg after 4-6 wks (23 mg option)	All AD stages; off-label DLB, VaD	Nausea, diarrhea, vivid dreams, bradycardia, syncope
Rivastigmine patch	4.6 → 9.5 → 13.3 mg/24 h	Mild-moderate AD; PDD	Skin irritation; less GI than oral
Galantamine ER	8 → 16 → 24 mg daily	Mild-moderate AD	GI effects, weight loss, bradycardia
Memantine	5 mg → 10 mg BID (or XR 28 mg)	Moderate-severe AD	Dizziness, confusion, constipation; dose-adjust in CKD

Set expectations: benefits are modest and symptomatic. Check heart rate and ask about syncope. Avoid cholinesterase inhibitors in FTD. Reassess and consider deprescribing at advanced stages or with adverse effects.

References: 11, 21, 39, 40

Anti-amyloid monoclonal antibodies

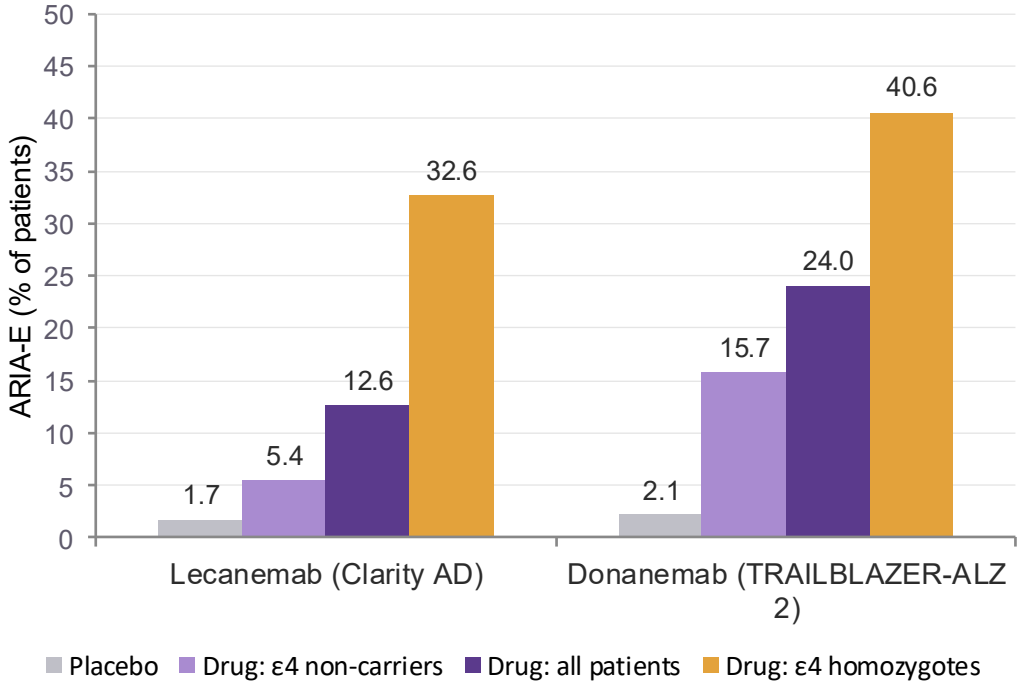
For early symptomatic Alzheimer's disease only: MCI or mild dementia with confirmed amyloid

	Lecanemab (Leqembi)	Donanemab (Kisunla)
Dosing	10 mg/kg IV q2 wk, or SC 500 mg weekly (approved Jul 2026); maintenance IV q4 wk or SC 360 mg weekly after 18 mo	IV q4 wk with modified titration to 1,400 mg; may stop when amyloid clears on PET
Pivotal efficacy	~27% slowing on CDR-SB at 18 months (Clarity AD)	~35% slowing on iADRS in low/medium tau (TRAILBLAZER-ALZ 2)
ARIA-E (edema)	~13% overall	~24% original dosing; lower with modified titration
APOE ε4 homozygotes	Highest ARIA risk; boxed warning	Highest ARIA risk; boxed warning
MRI monitoring	Baseline, then before scheduled doses early in treatment	Baseline, then before doses 2, 3, 4 and 7

References: 41, 42, 43

ARIA: the key safety trade-off

ARIA-E (edema) rates in the pivotal 18-month trials, by APOE ε4 status



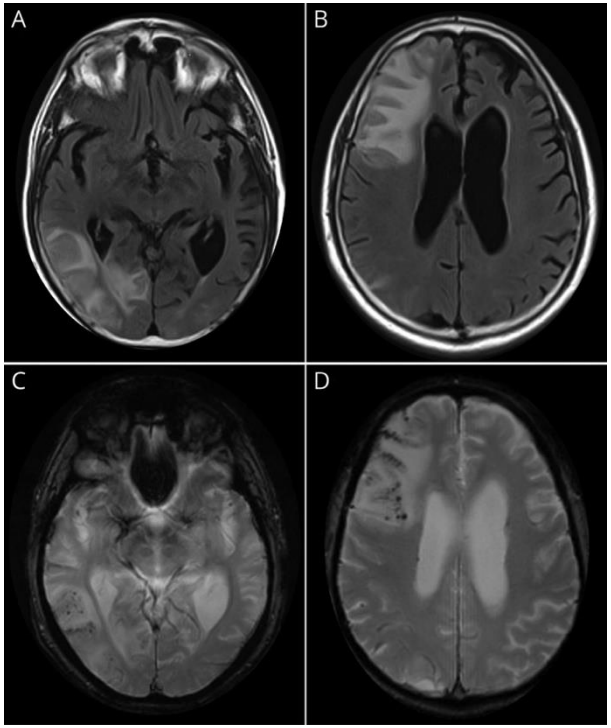
Recognizing ARIA

- Symptoms: headache, confusion, visual change, nausea, gait change, seizures
- New neuro symptoms on therapy: urgent MRI, call the prescribing team, hold doses
- Most ARIA is asymptomatic and found on scheduled MRIs
- Highest risk: ε4 homozygotes, anticoagulation

References: 41, 42, 43, 45

What ARIA looks like on MRI

Severe ARIA on axial MRI in a patient receiving anti-amyloid therapy



Reading the scans

- **ARIA-E (A, B):** FLAIR shows bright vasogenic edema and sulcal effusion
- **ARIA-H (C, D):** gradient-echo (T2*) shows dark dots of microhemorrhage and superficial siderosis
- The two often appear together, most often in the first months of dosing
- Ask radiology to report ARIA type and severity; the prescribing team decides on holding doses

Source: Day GS et al. Neurology 2022;98:619-631, Figure 1. © 2022 American Academy of Neurology

Anti-amyloid therapy: the primary care role

Likely candidates

- MCI or mild dementia due to AD
- Amyloid confirmed by PET or CSF (or validated blood test per specialist)
- Able to attend MRIs and infusions/injections
- Engaged care partner
- Goals aligned with modest slowing

Common exclusions

- Moderate or severe dementia
- Anticoagulation
- More than 4 microbleeds, superficial siderosis, prior macrohemorrhage
- Extensive white matter disease
- Bleeding disorder, uncontrolled seizures
- Non-AD primary cause (FTD, LATE)

What primary care does

- Recognize early symptoms promptly
- Complete the workup and blood biomarker
- Refer early: the window is MCI/mild
- Know ARIA symptoms: headache, confusion, visual change, seizures
- Flag therapy in the chart; avoid thrombolytics without specialist input

References: 43, 44, 45

Behavioral and psychological symptoms

Describe, Investigate, Create, Evaluate (DICE) before prescribing

D

Describe

What, when, where, and who is distressed

I

Investigate

Pain, constipation, infection, sleep, meds, environment

C

Create

A plan with the caregiver: routines, redirection, activity

E

Evaluate

Did it work? Adjust and re-assess

- Brexpiprazole: FDA-approved for agitation in AD dementia; antipsychotic boxed warning for mortality still applies
- SSRIs (citalopram, sertraline) for agitation and depression; trazodone for sleep and FTD behaviors
- Avoid benzodiazepines; avoid typical antipsychotics, especially in Lewy body disease
- Suvorexant for insomnia in AD; treat pain on a schedule (acetaminophen) when pain is suspected

References: 46, 47, 48, 49, 50

Treatment matched to dementia type

Type	What helps	What to avoid or be cautious with
Alzheimer's	ChEI ± memantine; anti-amyloid if early and eligible; brexpiprazole for agitation	Anticholinergics; sedatives
Vascular	Vascular risk control; ChEI (modest, off-label); rehab	Hypotension from over-treatment in frail elders
Lewy body / PDD	ChEI (often robust); rivastigmine for PDD; melatonin for RBD; pimavanserin (PDD psychosis)	Haloperidol and typical antipsychotics; excess levodopa
FTD	SSRIs, trazodone; behavioral and safety planning; genetic counseling	ChEIs (may worsen behavior); anti-amyloid therapy
LATE	ChEI (off-label, limited evidence); safety; caregiver support	Anti-amyloid therapy (amyloid negative)
Mixed	Treat each component; prioritize function and goals	Assuming one cause explains all symptoms

References: 19, 22, 25, 43

Caring well across the long distances

Tools for rural practice

- CMS GUIDE Model: care navigation, caregiver training, and respite payments for participating programs
- Cognitive assessment & care plan visit (CPT 99483)
- Telehealth partnerships with memory clinics for diagnosis and anti-amyloid follow-up
- Project ECHO dementia sessions for case-based learning
- Alzheimer's Association 24/7 Helpline: 1-800-272-3900

Planning ahead

- Advance care planning while the patient can participate
- Durable power of attorney for health care and finances
- Discuss what matters most as the disease progresses
- Hospice when appropriate: FAST stage 7 plus complications
- Revisit goals after hospitalizations, falls, and weight loss

References: 53, 54

Case: putting it together

An 86-year-old retired rancher

- Daughter reports 4 years of slowly worsening memory: repeats stories, misses appointments
- Still drives to town, manages cattle records with help; social, no hallucinations, no dream enactment
- MoCA 22/30, losing points mainly on delayed recall
- Labs normal; MRI: marked bilateral hippocampal atrophy, mild white matter changes, no microbleeds
- Plasma p-tau217: negative

Discuss

- Most likely cause?
- Is he a candidate for anti-amyloid therapy?
- What would you tell his daughter about prognosis?
- What should you address next?

References: 13, 26

Key takeaways

1. Name the syndrome, then pursue the cause: the first symptom and the tempo are your best clues.
2. Mixed pathology is the rule after 80; LATE is a common amyloid-negative mimic of AD.
3. Blood tests for AD pathology are now FDA-cleared: use them in symptomatic patients, and know your assay.
4. MRI with SWI is the key imaging study; add FDG-PET or DaTscan for specific questions.
5. Match treatment to type: no typical antipsychotics in Lewy body disease, no ChEIs in FTD.
6. Anti-amyloid therapy is for early, amyloid-confirmed AD. Early recognition in primary care is what makes it possible.



Questions & discussion

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