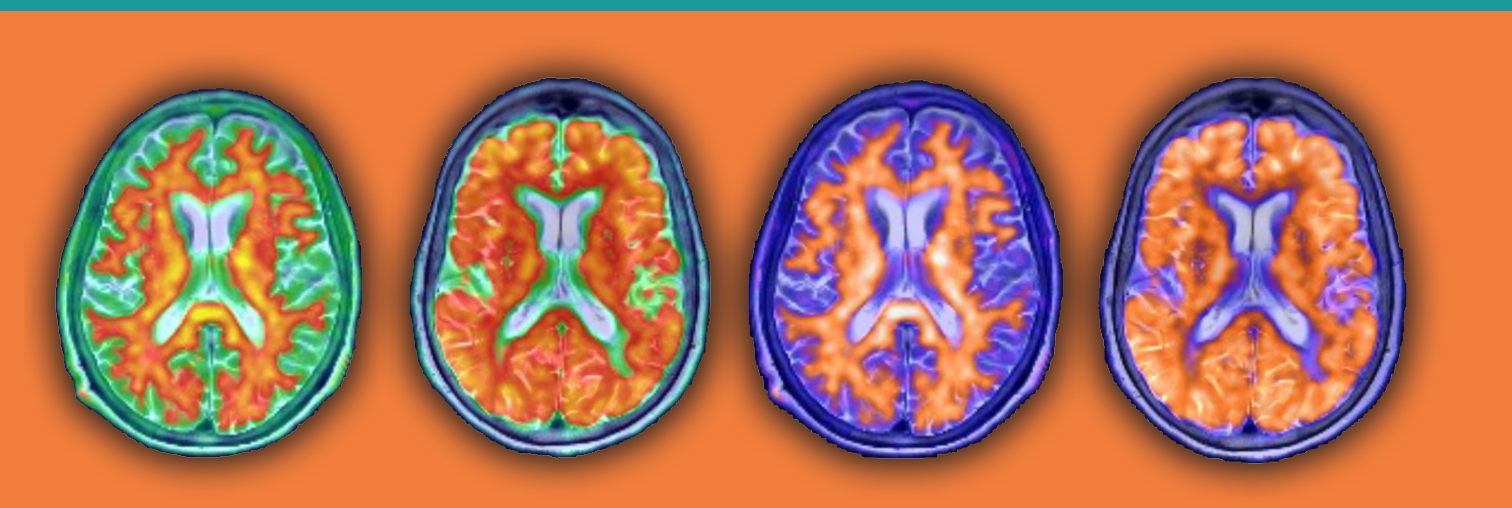
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Imaging Alzheimer's Disease in the Era of Anti-Amyloid Therapy

A Practical Guide to Patient Selection, Safety Surveillance, and Workflow Integration



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Imaging Alzheimer's Disease in the Era of Anti-Amyloid Therapy: A Practical Guide to Patient Selection, Safety Surveillance, and Workflow Integration

Acknowledgement

This white paper highlights the knowledge and experience of the following neuroradiology professionals who participated in a panel discussion: **Lawrence Tanenbaum, MD**, a neuroradiologist and chief technology officer and director of advanced imaging at RadNet, Inc., from 2015 to 2024; **Tammie L.S. Benzinger, MD, PhD,** a professor of radiology and chief of MRI services at Mallinckrodt Institute of Radiology at Washington University School of Medicine in St. Louis; **Suzie Bash, MD,** a neuroradiologist and medical director at RadNet, Inc.; and **Marwan Sabbagh, MD, FAAN**, a professor of neurology at Barrow Neurological Institute in Phoenix and vice chair of research at the Institute's department of neurology.

Important Safety Information

- MRI provides images to be interpreted by physicians; it is not indicated to determine disease stage or prognosis.
- Anti-amyloid therapy in Alzheimer's disease comes with risks. Please refer to the specific anti-amyloid therapy's prescribing information for side effects, contraindications, and risks.

Executive Summary

New anti-amyloid therapies (lecanemab and donanemab) have revolutionized neuroradiology's approach to Alzheimer's disease, from mostly symptomatic management to disease-modifying therapy (DMT). Dementia is a syndrome encompassing multiple etiologies, and accurate diagnosis requires identification of the underlying biological process.

This transition has put greater emphasis on data produced by MRI and PET imaging informing neurologists' clinical decisions. DMT requires confirmation of underlying pathology and introduces new safety considerations, shifting clinical workflows toward precision medicine.

As a result, imaging has moved into a central position to aid clinicians in diagnosis, patient selection, and longitudinal management of Alzheimer's disease (AD). The following white paper, informed by leading neuroradiologists and neurologists, provides a concise overview of AD's emerging biomarker-driven medical model. It also offers practical recommendations to neuroradiologists and multidisciplinary teams navigating this rapidly evolving field, where imaging experts now support decision-making regarding who receives therapy, how treatment is monitored, and when intervention should be modified or withheld.

A Transformational Moment

Historically, patients presenting with dementia underwent clinical evaluation to establish the presence and pattern of cognitive impairment. Phenotypical cognitive testing formed the foundation of this initial assessment. Alzheimer's disease (AD), which is present in 60% to 80% of patients with dementia,[1] was established by identifying its most common symptoms, typically amnestic patterns with memory encoding or retrieval deficits. Because no routine, accessible biomarkers for AD pathology existed in everyday clinical practice, neuroimaging from diagnostic tools like MRI and CT played only a secondary role in this process, usually to help clinicians exclude reversible causes of dementia, such as tumors, strokes, or normal pressure hydrocephalus.

Once diagnosed, AD treatment was entirely symptomatic. Drugs such as cholinesterase inhibitors and memantine could provide temporary relief of symptoms but could not stop neuronal loss or slow disease progression. For the most part, non-pharmacological and supportive care constituted the majority of AD management.

The introduction of anti-amyloid monoclonal antibody therapy for the treatment of cognitive disorders caused by AD (lecanemab in 2023 and donanemab in 2024) fundamentally



At RadNet, our neuroradiologists include a Centiloid value on all amyloid PET interpretations. I would encourage everyone to run their amyloid PET studies through a Centiloid workflow, as it is now becoming standard of care.

—Suzie Bash, MD

changed this traditional model for evaluating and treating dementia. For the first time ever, AD progression can be slowed. However, to achieve this goal, patient selection, treatment, and safety surveillance during treatment now require much greater reliance on precision diagnoses—a careful and ongoing synthesis of patient history, neurological examination, and clinical and biomarker evidence.

Imaging as a Prerequisite

Candidates for anti-amyloid therapy must demonstrate mild cognitive impairment or mild dementia, elevated brain beta-amyloid, and sufficient physical and mental health to withstand the rigors of therapy. Patients must also be screened for co-existing non-AD pathologies that will be unresponsive to treatment or pose treatment risk factors.

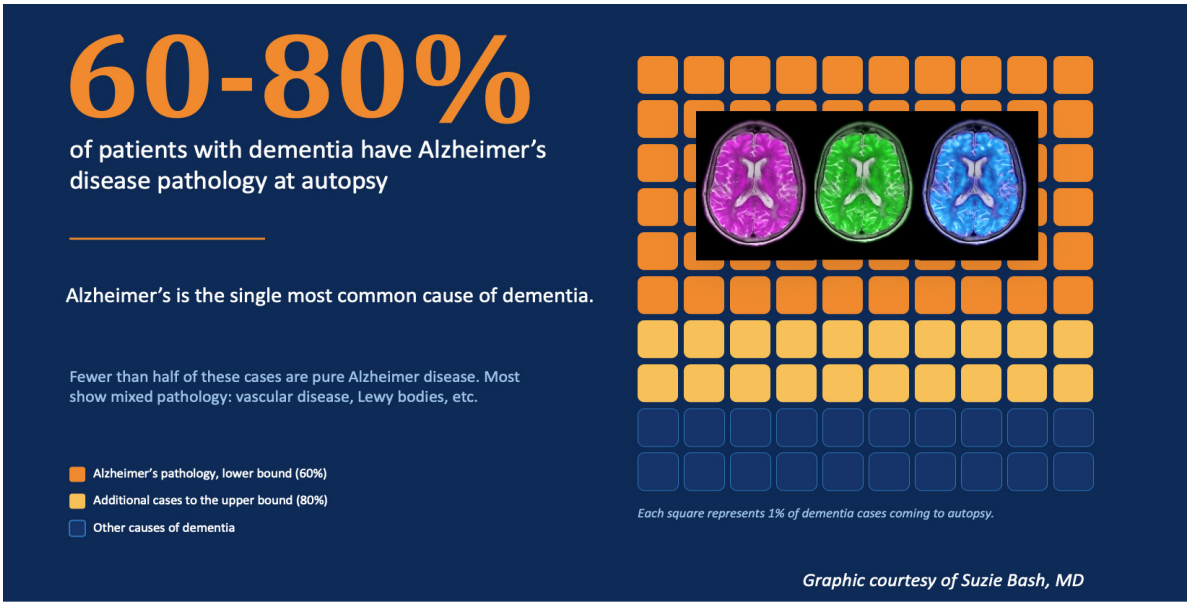
In the U.S., clinicians are increasingly employing quantitative amyloid PET scans combined with visual reads to provide evidence of beta-amyloid levels in the brain and to assist in confirming a diagnosis of AD. Amyloid PET also supports treatment duration and dosing decisions by helping neurologists to quantify beta-amyloid plaque clearance.

Quantitative MRI (QMRI) identifies and longitudinally tracks brain substructure volumes and lesions. It is a biomarker for neurodegeneration, which can help stage AD at the time of presentation. Newly FDA-cleared QMRI solutions can identify and count baseline microhemorrhages and superficial siderosis, as well as provide a Fazekas score to objectively document and screen for severe microvascular ischemic disease. Significant pre-existing vascular disease can increase the risk of the therapy's most serious side effect, amyloid-related imaging abnormalities (ARIA), which in severe cases can be life-threatening. Baseline MRI and QMRI also help screen for potential neurodegenerative conditions that can mimic AD. Finally, neuroradiologists must evaluate surveillance MRIs at regular intervals throughout the treatment to safeguard against ARIA. QMRI tools are commercially available that can assist in automated ARIA surveillance monitoring.

Early and Accurate Detection

Amyloid PET: The Gatekeeper

Amyloid PET scans enable clinicians to confirm the presence of amyloid plaques, which are the primary target of new ATT. Normal beta-amyloid values raise the possibility of alternative neurodegenerative or cerebrovascular

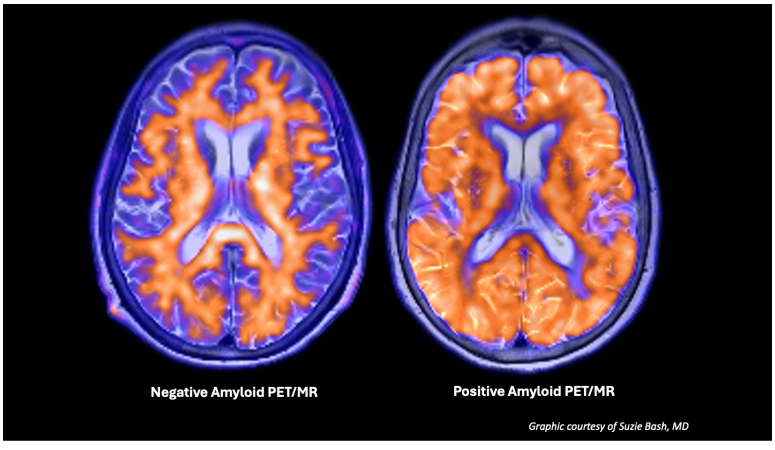




Because these drugs are treating the very earliest stages, you shouldn't see hippocampal atrophy, or if you do, it should be mild.
—Tammie L.S. Benzinger, MD, PhD

contributing etiologies in patients with cognitive impairment.[2] An alternative to PET scans, cerebrospinal fluid (CSF) analysis, can provide evidence of the presence of amyloid pathology, but involves a lumbar puncture, which tends to be unpopular with patients because it is painful and involves the risk of infection. CSF analysis also has less value for measuring plaque clearance success during later stages of the treatment.[2]

In the lecanemab clinical trial, researchers used visual reads of amyloid PETs to distinguish between "not elevated" and "elevated" amyloid beta levels. However, it can be challenging to gauge whether a scan is positive or negative when there is an intermediate amyloid burden by visual interpretation alone.



Therefore, quantitative amyloid PET has emerged as an adjunct precision-medicine approach to support visual reads.[3]

Generated by quantitative processing software, the globally standardized and vendor-neutral unbounded Centiloid scale helps clinicians compare amyloid burden across tracers and brain regions. A value of 0 represents the mean of normal healthy controls, and 100 corresponds to the average amyloid burden seen in patients with clinically typical AD. Intermediate values, around 10 to 25 Centiloid, may indicate early/subtle amyloid pathology.[4]

"At RadNet, we routinely incorporate Centiloid values in the interpretation of amyloid PET studies by processing all scans through quantitative Centiloid workflow," says Suzie Bash, MD, a neuroradiologist and medical director at RadNet. "This approach is critically important because it establishes the baseline amyloid plaque burden at the start of therapy. Once the initial 18-month treatment period is complete, many patients move on to IV or subcutaneous maintenance therapy to prevent plaque reaccumulation. At that point, Bash notes, the neurologist often orders a follow-up amyloid PET so that Centiloid values can be reassessed, quantifying the degree of amyloid clearance[5] and informing maintenance dosing decisions. "A visual read tells us whether amyloid is present," says Bash. "Centiloid quantification allows an objective measure of how much clearance has occurred relative to baseline, which is what those decisions turn on. Quantitative PET is becoming standard of care, and running both the baseline and the follow-up scan through a Centiloid workflow is what makes the comparison meaningful."

Maintenance therapy decisions are individualized and depend on both clinician assessment and the degree of amyloid clearance achieved.

In addition to amyloid PET scans, Tau PET scans could play a more frequent role as AD therapy evolves. Baseline tau burden appears to be superior to amyloid PET and MRI at predicting cognitive decline.[6] Substudy analysis of the clinical trials demonstrated that lower baseline tau levels are associated with slower decline and better treatment response.[7] However, because reimbursement of Tau PETs varies, panel members rarely order them unless patients opt to pay for the scans out-of-pocket or live in a region where the scans are reliably reimbursed.

Although anti-amyloid therapy only directly targets amyloid plaques, there is evidence that it can slow tau accumulation as well.[8] As new generations of tau-targeting therapies advance through clinical trials, Tau PET scans may take on greater importance in personalized AD treatment strategies going forward.[3]

Right Patient, Right Time

Only some patients with evidence of amyloid plaques will be eligible for treatment. The therapies intentionally target the earliest symptomatic window of AD (stages 3 and 4, mild cognitive impairment

or mild dementia due to Alzheimer's) because those patients respond most favorably to treatment and their safety risks are most manageable. Unfortunately, diagnosis is delayed until moderate or advanced stages in 50% or more patients with Alzheimer's, when it is too late to be eligible for anti-amyloid therapy.[9] Additionally, some patients will fail eligibility due to neurodegenerative or cerebrovascular comorbidities.

"To be realistic, these are not drugs for everybody," says Marwan Sabbagh, MD, FAAN, a professor of neurology at Barrow Neurological Institute in Phoenix. "All of the data, whether it's in-house or out or from other centers, say only about 20% of patients would be eligible or appropriate."

Lewy body disease and limbic predominant age-related TDP-43 encephalopathy (LATE) are the two neurodegenerative disorders that occur most commonly with AD as co-pathologies. Absent a definitive phenotypic confirmation, DaTscan, FDG PET, skin biopsies and CSF biomarkers may help clinicians confirm a Lewy body disease diagnosis.[2] Because no fluid biomarker for LATE exists, diagnosis remains provisional. But non-elevated or marginally elevated amyloid beta along with a lack of AD-like topographic pattern of degeneration, as professionally measured by FDG PET, could be a red flag.[2]

Certain cerebrovascular diseases, such as a high burden of white matter hyperintensities, more than two lacunar infarcts, or single regional infarcts, raise the possibility of vascular disease contributing to the cognitive impairment, but do not necessarily exclude treatment. However, cerebral amyloid angiopathy (CAA) with more than four cerebral microbleeds or superficial siderosis contraindicates therapy due to risk of hemorrhage (ARIA-H), panelists say.



"The interesting thing that's positive is that the reported ARIA rates post-approval are lower than what was reported in the trials."
--Marwan Sabbagh, MD, FAAN

Brain volume can be a workflow stumbling block during the patient screening process. "This is one of the common misconceptions radiologists have," notes Tammie L.S. Benzinger, MD, PhD, a professor of radiology and chief of MRI services at Mallinckrodt Institute of Radiology at Washington University School of Medicine in St. Louis. "They'll get the quantitative MRI and see that the hippocampal volumes look normal, and they'll say, 'Oh, this isn't Alzheimer's disease.' So that's the opposite of where we are right now. Because these drugs are treating the very earliest stages, you shouldn't see hippocampal atrophy, or if you do, it should be mild."

ARIA Surveillance

Anti-amyloid therapies remove beta-amyloid from the brain's parenchyma via immune-mediated mechanisms. But this process inevitably involves vascular pathways. Paradoxically, although beta-amyloid plaques are pathology, beta-amyloid itself provides some stability to the blood vessel walls. Removing beta-amyloid quickly can lead to ARIA-H or edema (ARIA-E) or both. Risk for ARIA is highest during the first three to six months of therapy and drops sharply after that.[10]

New Imaging Responsibilities

According to clinical guidelines, surveillance MRIs should occur on lecanemab patients before the third, fifth, seventh and 14th infusion. For high-risk patients, such as those with prior ARIA, an optional safety scan may occur before the 26th infusion.[11]

For donanemab patients, surveillance MRIs should occur prior to the second, third, fourth, and seventh infusion. High-risk patients may receive a safety scan prior to the 12th infusion.[3]

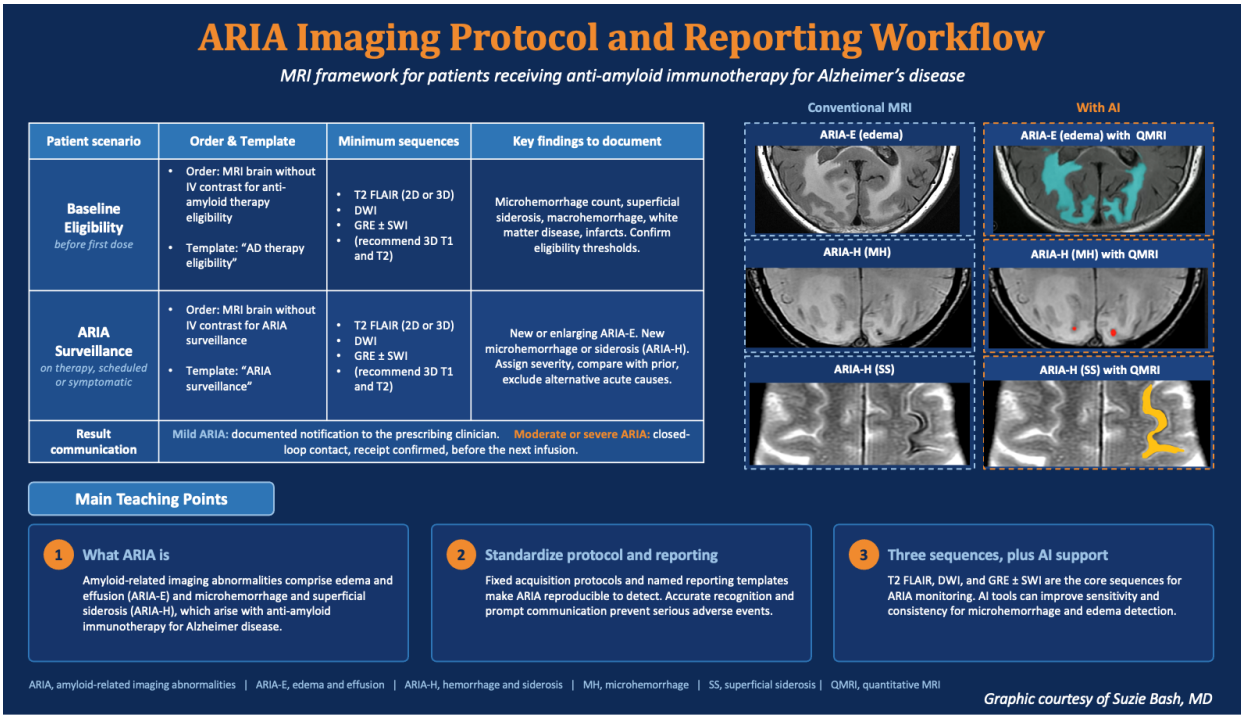
ARIA is graded as mild, moderate or severe. ARIA-E appears as areas of hyperintensity on fluid-attenuated inversion recovery (FLAIR) MRI sequences, with severity based on the size and number of sites involved:

- **Mild:** One location smaller than 5 cm.
- **Moderate:** One location of 5 to 10 cm, or more than one location smaller than 10 cm.
- **Severe:** One or more locations larger than 10 cm.

ARIA-H appears on iron-sensitive MRI sequences as new or worsening hemorrhagic changes compared to baseline. It presents either as new microhemorrhages or focal areas of superficial siderosis:

- **Mild:** Fewer than five new microhemorrhages; or one focal area of superficial siderosis.
- **Moderate:** Five to nine new microhemorrhages; or two focal areas of superficial siderosis.
- **Severe:** More than 10 new microhemorrhages; or more than two focal areas of superficial siderosis.[12]

In cases of mild radiographic ARIA, therapy usually continues but with more frequent monitoring.



Moderate or severe radiographic ARIA typically requires temporary suspension of therapy until the ARIA-H stabilizes and ARIA-E resolves on MRI. New neurological symptoms, such as headaches, confusion, seizures or focal deficits require more cautious management, even if radiographic findings are mild. However, the majority of ARIA cases are asymptomatic, with symptomatic ARIA rates only about 3% for both lecanemab[9] and donanemab, with donanemab reaching this new low level after the titration schedule was recently modified.[13]

In the clinical trials for the therapies, rates for any kind of ARIA averaged around 22% for lecanemab and 24% for donanemab.[14] However, anecdotally, panel members who have treated anti-amyloid patients in real-world settings say their ARIA rates have been lower, around 10% to 15%.

"The interesting thing that's positive is that the reported ARIA rates post-approval are lower than what was reported in the trials," notes Sabbagh. "And the reason is that physicians like me are selecting our cases more carefully. We're seeing ARIA rates of about 12%, most of them asymptomatic. So, it's a less scary and less frequent thing than has been reported." This lower reported ARIA rate of 12% was also observed by both Bash and Benzinger in their respective clinical practices.

MRI and Safe Therapy Initiation

Baseline MRI images provide clinicians with evidence of exclusionary findings that increase the likelihood of ARIA or alternative causes of cognitive impairment. They also provide a reference for future comparisons during surveillance monitoring.

The baseline MRI should be recent. In cases where the baseline scan is older than six months and patients demonstrate risk factors, guidelines strongly encourage clinicians to perform a repeat scan before treatment begins. An MRI within 12 months may be adequate if negative for microhemorrhages, while 1 to 4 microhemorrhages on a 7- to 12-month-old MRI should prompt repeat imaging.[15]

Consistent MRI field strengths are helpful across the Alzheimer's imaging pathway but are not required. 3T MRI is more sensitive than 1.5T and therefore may give the appearance of new microhemorrhages or superficial siderosis due to improved detection rather than true progression.

Both eligibility screening and ARIA surveillance require the same standardized MRI sequences:

- **FLAIR and T2-weighted imaging:** Provides evidence of edema and effusion (ARIA-E), white matter hyperintensities, infarcts, and other structural abnormalities. Severe white-matter disease (extensive confluent hyperintensities) and any findings suggestive of cerebral amyloid angiopathy (CAA)-related inflammation may exclude therapy. (Severity can be graded using the Fazekas scale).
- **Gradient-recalled echo sequences (GRE) or susceptibility-weighted imaging (SWI):** Provides evidence of cerebral microbleeds and cortical superficial siderosis. Five or more microbleeds in any

location, any cortical superficial siderosis, or a single macrohemorrhage greater than 10 mm exclude therapy.

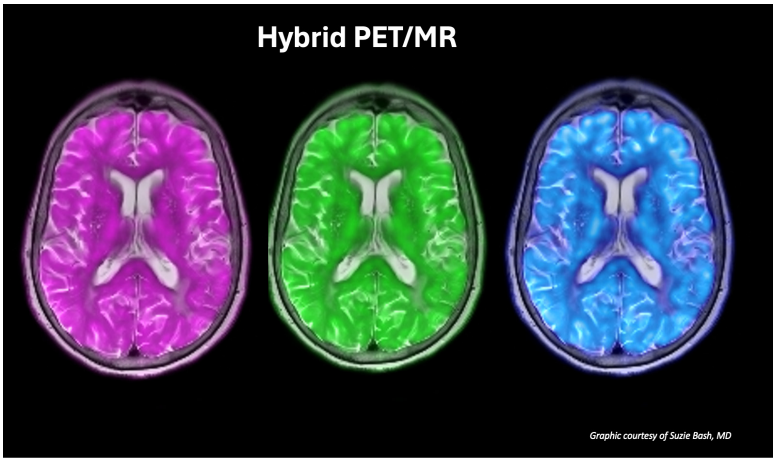
- **Diffusion-weighted imaging (DWI):** Assists clinicians in ruling out acute or subacute infarcts, or other ischemic changes. Large infarcts, multiple lacunar infarcts, or stroke in a major vascular territory may exclude therapy.[16]

The question of GRE vs. SWI has emerged as an important one as it relates to communication between radiologists and neurologists. In the clinical trials for these therapies, grading scales were established by researchers interpreting GRE. But SWI provides greater sensitivity and therefore may help readers uncover more microbleeds, which can directly impact patient eligibility.

"Our neuroradiologists and neurologists find value in having both GRE and SWI, so at RadNet our dementia protocol includes both, when SWI is available on the magnet," says Bash. "It's worth noting that the ARIA grading scales were developed on GRE. SWI is more sensitive for detection of microhemorrhages and superficial siderosis, so the thresholds don't translate directly. For accurate comparison throughout treatment, the blood-sensitive sequence should stay consistent between the baseline MRI and the ARIA surveillance examinations."

Quantitative MRI and AI Integration

Despite these standardized approaches, real-world implementation of baseline eligibility and treatment surveillance with MRI remains imperfect. Inter-reader variability among radiologists who may be unfamiliar with the new protocols and scans performed on different machines using different sequences adds to this challenge. AI software has emerged as a tool to help care teams improve efficiency, detection sensitivity, and reproducibility while also reducing interpretive bias.



There are FDA-cleared QMRI software programs that use deep learning to support clinical decision-making regarding segmenting, measuring, and counting FLAIR hyperintense lesions and microhemorrhages; these programs can help clinicians visualize small and subtle findings critical to baseline eligibility and ongoing surveillance. Neuroradiologists have found these tools particularly useful in speeding up baseline volumetric analysis.[17]

Although the radiologist ultimately bears responsibility for the final report, studies have shown that AI assistance can improve their sensitivity for ARIA detection. However, these findings also suggest that specificity decreases due to more frequent false positives.[18]

"I really like the idea of using an AI assistant tool," says Bash. "It can raise the bar for some people who don't have as much experience in ARIA surveillance. And it can decrease reader subjectivity, even for people who have a lot of experience with this."

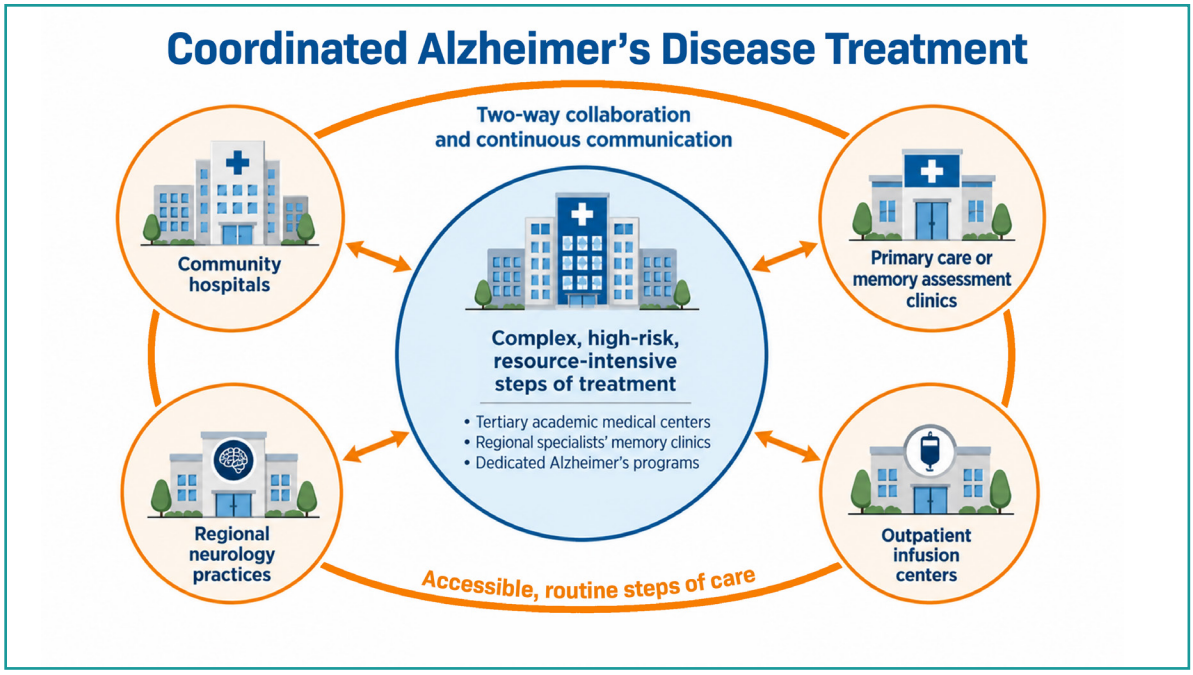
"I've come around 180 degrees," adds Benzinger. "Originally, I said any radiologist can recognize vasogenic edema and siderosis. MR interpretation is our bread and butter." But the new and constantly updated protocols and nomenclature surrounding anti-amyloid therapy can be difficult to keep up with, she acknowledges. "And so, asking a radiologist to know whether these findings are mild or moderate—that's not necessarily something they need to master. That's something a computer could do."

Multidisciplinary Workflow and Care Coordination

Anti-amyloid therapy involves an enormous amount of scanning, diagnosis, decision making, and coordinated multidisciplinary workflows over an extended timeline, which can be onerous to both clinicians and patients.

Subcutaneous anti-amyloid formulations that can be delivered via autoinjectors to patients at home could play a role in lightening workloads and improving patient convenience. Last year the FDA approved a 360-mg maintenance dose of lecanemab that patients can self-administer following an initial 18 months of intravenous treatment.[19] Further, the manufacturer received FDA approval this year for a subcutaneous dose for treatment initiation.[20] Next-generation anti-amyloid therapies are reportedly using subcutaneous injections in their clinical trials.[21]

Another potential time saver involves leveraging hybrid PET/MRI scanners. Combining these scans



into a one-stop site visit during eligibility screening and during surveillance tracking and treatment success evaluations has streamlined Benzinger's and Bash's workflows.

Benzinger explains: "Our community doctors said: 'Our patients have had so many MRIs. They're so burdened. Is there any way we can get the PET and the MRI on the same day at least?' And I said, absolutely, let's use the PET/MRI. They've all gone to the PET/MRI since then, and it's really been a game changer. I'm always so happy when we can do something that makes it easier for these families."

Bash agrees that hybrid PET/MRI has many advantages, including alleviation of multi-test day anxiety for the patient. Memory-loss patients in her region at RadNet are also preferentially scanned on a hybrid PET/MRI.

Shared Decision Making

Because anti-amyloid therapy is most effective on patients with early AD (and is only FDA approved for those stages), it is important for radiologists to provide neurologists with the appropriate imaging data for identifying patients in this narrow, early treatment window, a task that can be challenging.

For example, Sabbagh notes, subtle degenerations in hippocampal volumes may be interpreted as healthy. "The golden rule was fifth percentile or below on hippocampal volumes equals Alzheimer's," he observes. "And if it's above fifth percentile, that equals not Alzheimer's. Well, that's not true anymore. You may have a fifteenth percentile or a twentieth percentile, and you're already well on your way to degeneration. That's one of my pet peeves that I see on reports."

Moreover, radiologic reports for dementia patients must now contain so much new, granular, and nuanced data—white matter rarefaction, microhemorrhages, siderosis, volumetric assessment of the regions, areas of hyperintensity, etc.—that it can be easy to miss one small detail that leads to having to update the report, according to Sabbagh.

Efforts to streamline workflows and heighten efficient collaboration among radiologists, neurologists, primary care physicians, and support staff will be of paramount importance as the therapies evolve.

The Road Ahead

"There's a lot that has been added to our workflow," says Sabbagh. "To the point where we actually have a nurse practitioner in a dedicated clinic, and her only job is to manage these patients." As therapy adoption expands, neuroradiology may also face challenges in keeping up with demand.

There is concern that this rising demand for anti-amyloid therapies could ultimately overwhelm an unprepared health system.[22] One study states, "Healthcare systems around the country are working to respond to this need, which will likely require new partnerships among community organizations, primary care clinicians, memory-care-experienced nurse practitioners, and specialists."[22]

A model put forth as a possible solution is called the "hub-and-spoke" model.[23] In this framework, the complex, high-risk, and

resource-intensive steps of treatment occur at a "hub," typically tertiary academic medical centers, regional specialists' memory clinics, or dedicated Alzheimer's programs. And the accessible, routine steps of care, such as identifying eligible patients, occur at a "spoke," typically community hospitals, outpatient infusion centers, primary care or memory assessment clinics, or regional neurology practices.

Conclusion

Alzheimer's disease care has been substantially changed by the arrival of DMTs. Both MRI and PET are central components of clinical decision-making, with neuroradiologists playing an important role in confirming amyloid pathology, supporting treatment eligibility, ARIA safety surveillance, and longitudinal follow-up.

This new paradigm requires a shift in both practice and mindset. Multidisciplinary diagnostic collaboration, quantification, and standardization will be necessary to meet the demands of modern Alzheimer's care. As diagnostic demand grows, scalable solutions that integrate advanced imaging, AI-enabled analysis, and cross-site data harmonization will be essential.

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