

From Target Engagement to Treatment Governance: The Evolving Role of Amyloid PET in Anti-Amyloid Therapy

Massimo Filippi,^{a,b,c,d,e,*} Giordano Cecchetti,^{a,b} Alma Ghirelli,^{a,b,c} Edoardo G. Spinelli,^{a,b,c} Giulia Rugarli,^{a,b,c} Stefano Pisano,^{a,b} Ana Maria Samanes Gajate,^f Arturo Chiti,^f and Federica Agosta^{a,b,c,e}

^aCenter for Alzheimer's and Related Diseases (CARD), Neurology Unit, IRCCS San Raffaele Scientific Institute, Milan, Italy

^bNeuroimaging Research Unit, Division of Neuroscience, IRCCS San Raffaele Scientific Institute, Milan, Italy

^cVita-Salute San Raffaele University, Milan, Italy

^dNeurorehabilitation Unit, IRCCS San Raffaele Scientific Institute, Milan, Italy

^eNeurotech Hub, Vita-Salute San Raffaele University, Milan, Italy

^fNuclear Medicine Service, IRCCS San Raffaele Scientific Institute, Milan, Italy



Summary

The introduction of anti-amyloid monoclonal antibodies has shifted Alzheimer's disease care from diagnostic clarification alone to the problem of treatment governance. While lecanemab and donanemab have demonstrated amyloid reduction at the group level, routine clinical care raises a different question: how should treatment exposure be managed in individual patients within constrained healthcare systems? We suggest that amyloid positron emission tomography (PET) may support this task by providing a quantitative baseline reference for target engagement and by informing decisions on treatment continuation, switching, or discontinuation during follow-up. However, amyloid reduction on PET should not be equated with proven individual-level clinical benefit, and the potential role of longitudinal PET must be considered alongside unresolved questions regarding safety, treatment burden, feasibility, and health-system capacity. Drawing on real-world experience from a high-volume European memory clinic, we argue that amyloid PET may have an increasingly relevant role within structured anti-amyloid treatment pathways.

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Why treatment governance has become a distinct problem in the anti-amyloid era

The introduction of lecanemab and donanemab has changed the clinical landscape of early symptomatic Alzheimer's disease (AD).¹⁻⁴ For many years, biomarker use in memory clinics was primarily directed at etiological clarification: establishing whether cognitive impairment was associated with amyloid pathology and, if so, supporting a biological diagnosis of AD.^{5,6} With the arrival of anti-amyloid therapies, however, a new and partly distinct question has emerged. Beyond identifying eligible patients, clinicians and healthcare systems must now determine how treatment exposure should be monitored and governed over time.

This shift does not resolve the broader debate surrounding the net clinical value of anti-amyloid therapies. Although both lecanemab and donanemab have shown the capacity to reduce amyloid burden, the extent to which biomarker change translates into meaningful patient-level cognitive, functional, and safety outcomes remains actively debated, particularly in publicly funded healthcare systems facing substantial constraints in cost, service capacity, and monitoring burden.⁷⁻⁹ The present Viewpoint does not seek to adjudicate that broader debate. Rather, it addresses a narrower and practical question: when these agents are implemented in routine care, how can treatment exposure be aligned with demonstrable biological target engagement?

Across Europe, amyloid biomarker infrastructures were largely developed for etiological clarification rather than longitudinal treatment decision-making, and their role is now being reconsidered in light of anti-amyloid therapies.^{5,6,10,11}

Within this framework, amyloid PET deserves renewed consideration. Unlike fluid biomarkers, quantitative amyloid PET can provide a baseline estimate of fibrillar amyloid burden and permit

*Corresponding author. Neuroimaging Research Unit, Division of Neuroscience, IRCCS San Raffaele Scientific Institute, Via Olgettina, 60, Milan 20132, Italy.

E-mail addresses: filippi.massimo@hsr.it (M. Filippi), cecchetti.giordano@hsr.it (G. Cecchetti), ghirelli.alma@hsr.it (A. Ghirelli), spinelli.edoardogioele@hsr.it (E.G. Spinelli), rugarli.giulia@hsr.it (G. Rugarli), pisano.stefano@hsr.it (S. Pisano), samanesgajate.ana-maria@hsr.it (A.M. Samanes Gajate), chiti.arturo@hsr.it (A. Chiti), agosta.federica@hsr.it (F. Agosta).

longitudinal assessment of residual target burden under therapy.^{12,13} This potential role is particularly relevant when decisions about treatment continuation, switching, or discontinuation depend on whether meaningful target engagement has occurred. At the same time, any proposal to expand the use of longitudinal amyloid PET must be considered together with its own limitations, including scan availability, tracer access, reimbursement variability, radiation exposure, and organisational burden. The goal, therefore, is not to advocate indiscriminate PET use, but to examine whether amyloid PET may have a strategic role in treatment governance within constrained European healthcare systems, while remaining explicit about the questions it can inform and those it cannot resolve on its own (Fig. 1).

What amyloid PET can uniquely add to treatment-oriented decision-making

Amyloid PET provides information that differs from simple confirmation of amyloid positivity. Quantitative PET can estimate the burden of fibrillar amyloid and define a baseline reference against which subsequent treatment-related changes may be interpreted.^{12–14} This becomes particularly relevant in the setting of anti-amyloid therapies, where the question is not only whether a patient is biologically eligible for treatment, but also whether treatment exposure remains aligned with target engagement.

At treatment initiation, baseline amyloid PET can serve as a quantitative anchor. Fluid biomarkers,

including cerebrospinal fluid (CSF) and plasma measures, may reliably support the biological diagnosis of AD and are often sufficient for etiological assessment.^{5,6,15–17} However, they do not provide the same in vivo estimate of residual fibrillar amyloid burden or its regional distribution for longitudinal comparison under therapy. When treatment-related decisions depend on whether amyloid burden has changed meaningfully over time, a paired PET framework offers a type of information that fluid biomarkers do not fully replicate. In addition, baseline amyloid PET contributes to risk stratification in patients exposed to therapies associated with amyloid-related imaging abnormalities (ARIAs).^{18,19}

This paired framework becomes even more relevant during follow-up. In routine care, the key question is often not whether anti-amyloid therapies reduce plaques at the group level, as shown in pivotal trials,^{1,20} but whether the trajectory of amyloid reduction in an individual patient is sufficient to support treatment continuation, switching, or discontinuation. In this context, follow-up amyloid PET may help identify different patterns of target engagement, ranging from minimal reduction to substantial lowering of residual amyloid burden. These PET-defined patterns are not direct surrogates of patient-level clinical benefit, but they may still inform decisions about treatment exposure when interpreted in conjunction with the broader clinical context.

This treatment-oriented use of paired amyloid PET is consistent with the Treatment-Related Amyloid Clearance (TRAC) framework, which formalized a

Amyloid PET in anti-amyloid therapy

Potential contributions and key limitations

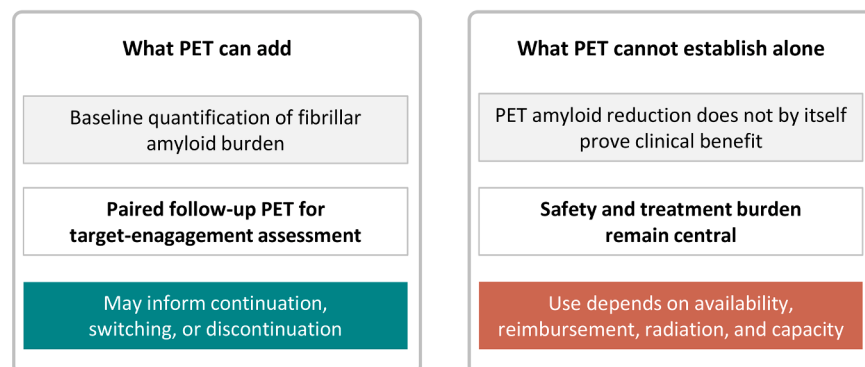


Fig. 1: Potential contributions and key limitations of amyloid PET in the anti-amyloid therapy pathway. Conceptual overview of the potential treatment-oriented role of amyloid PET in patients receiving anti-amyloid therapies. The left panel summarizes how baseline and follow-up quantitative amyloid PET may support assessment of target engagement and inform decisions on treatment continuation, switching, or discontinuation. The right panel highlights key boundaries of this approach: amyloid reduction on PET does not by itself establish patient-level clinical benefit, and any expanded use of longitudinal PET must be weighed against safety considerations, treatment burden, scan availability, reimbursement heterogeneity, radiation exposure, and system-level capacity constraints.

vocabulary for describing treatment-related changes on baseline and follow-up PET imaging.¹³ In the present Viewpoint, we do not seek to expand or redefine that framework. Rather, we argue that its logic may be particularly useful in real-world care, where the practical challenge is not simply to document biomarker change, but to decide whether a given treatment course should be continued, modified, or stopped within constrained healthcare systems.

What amyloid PET cannot resolve on its own

Amyloid PET may document target engagement, but it does not by itself establish patient-level net benefit. Although anti-amyloid therapies reduce fibrillar amyloid burden, the extent to which such biomarker changes translate into meaningful cognitive, functional, or quality-of-life benefit remains debated.^{1,2,7–9} PET-defined amyloid reduction should therefore not be treated as a standalone surrogate of clinical efficacy.

Nor does amyloid PET resolve the safety dimension of treatment governance. Continuation, switching, or discontinuation decisions must also account for ARIA risk, treatment tolerability, comorbidity, patient preference, and the burden of repeated infusions and MRI monitoring.^{19,21,22} In addition, longitudinal PET carries its own implementation constraints, including scan availability, tracer access, reimbursement heterogeneity, and scheduling burden, all of which vary substantially across European healthcare systems.^{7,8,10,11}

Our argument, therefore, is not that amyloid PET settles the broader controversies surrounding anti-amyloid therapy. It is that, when these therapies are used, PET may provide one distinct layer of information for treatment governance, provided that it is interpreted together with clinical outcomes, safety, and system-level feasibility.

Implementation lessons from a high-volume European memory clinic

Within our anti-amyloid treatment program at the Center for Alzheimer's and Related Diseases (CARD), IRCCS San Raffaele Scientific Institute (Milan, Italy), amyloid PET has been incorporated at baseline and during follow-up as part of a structured multidisciplinary care pathway.^{23,24} In this setting, the rationale for longitudinal PET has not been to demonstrate efficacy in the trial sense, but to support treatment-oriented decision-making in routine practice when questions arise regarding whether ongoing exposure remains aligned with observable target engagement.

To date, 27 out of 58 treated patients (46.5%) have reached the 6-month follow-up timepoint and completed amyloid PET, providing the basis for this implementation observation. In the lecanemab-treated cohort, four of nine patients were switched to donanemab after follow-up PET demonstrated minimal or

Treatment	Total patients	Ongoing	Discontinued for efficacy/clearance	Discontinued for ARIA-E	Switched due to insufficient biological response
Donanemab	18	9	7	2	0
Lecanemab	9	5	0	0	4

Treatment decisions were informed by longitudinal amyloid PET findings integrated with clinical context. For donanemab, discontinuation for efficacy was based on the pivotal treat-to-clear stopping criterion (<11 Centiloids).¹ For lecanemab, treatment switching reflected minimal/insufficient amyloid reduction on follow-up PET. Counts are presented to illustrate treatment-governance decisions in routine practice and are not intended for cross-drug efficacy comparisons.

Table 1: PET-guided treatment course at 6-month follow-up in patients treated with anti-amyloid monoclonal antibodies in a real-world memory clinic setting.

insufficient amyloid reduction (Table 1).²² Patients who continued lecanemab showed lower residual global amyloid burden and greater amyloid reduction than those who were switched (Table 2), consistent with a TRAC-informed distinction between partial clearance and limited biological response.¹³

In the donanemab-treated cohort, seven of 18 patients reached amyloid levels below 11 Centiloids at follow-up, consistent with the treat-to-clear stopping criterion applied in pivotal donanemab programs (Table 1).¹ In these cases, follow-up PET enabled early discontinuation of therapy based on biological grounds. Among the remaining patients, nine showed ongoing amyloid reduction with residual burden above 11 Centiloids, supporting treatment continuation, whereas two discontinued because of ARIA-E.^{21,22} In these cases, treatment discontinuation was primarily driven by safety, but follow-up PET still provided useful context regarding the degree of target engagement achieved.

Taken together, these observations are not intended as proof of clinical efficacy, nor as a basis for cross-drug comparison. Rather, they provide proof of principle that, in a high-volume memory clinic operating under real-world constraints, longitudinal amyloid PET can

Treatment and decision group	n	Median 6-month CL	Median ΔCL (baseline – 6 months)	Interpretation
Lecanemab				
Ongoing	5	28.8	53.0	Partial amyloid clearance
Switched	4	84.9	13.5	Minimal/insufficient amyloid clearance
Donanemab				
Ongoing	9	36.9	56.8	Partial amyloid clearance
Discontinued for clearance	7	3.8	86.2	Clearance consistent with treat-to-clear paradigm
Discontinued for ARIA-E	2	NA	NA	Discontinuation driven by safety

Quantitative amyloid PET metrics underlying PET-guided clinical decisions. ΔCL was defined as baseline minus follow-up Centiloids; higher positive values indicate greater amyloid reduction. For donanemab, <11 Centiloids at follow-up reflects the stopping criterion applied in pivotal treat-to-clear programs. Values are reported to illustrate within-group biological trajectories and are not intended for cross-drug efficacy comparisons.

Table 2: Quantitative amyloid PET findings underlying PET-guided treatment decisions.

contribute to practical decisions on continuation, switching, and discontinuation when treatment exposure must be individualized over time.

Organizational and resource implications in constrained healthcare systems

The potential value of amyloid PET in the anti-amyloid therapy pathway is best considered in organisational and resource-allocation terms rather than as evidence of formal cost-effectiveness. Both lecanemab and donanemab require sustained drug exposure, repeated infusion delivery, structured MRI surveillance for ARIA, and dedicated multidisciplinary oversight, thereby generating cumulative demands on healthcare systems beyond the acquisition cost of the drugs themselves.^{7–9,21,22} In this context, decisions regarding treatment duration are not only clinical decisions, but also operational ones.

Our real-world experience suggests that longitudinal amyloid PET may contribute to these decisions by helping determine whether ongoing treatment exposure remains aligned with observable target engagement. In patients whose management was modified after follow-up PET, the implications extended beyond biomarker interpretation and included avoided infusion sessions, reduced continuation of treatment-related monitoring, and reallocation of clinical capacity within a finite treatment programme (Table 3).

In our cohort, PET-informed treatment modification occurred in four lecanemab-treated patients and seven donanemab-treated patients at approximately six months after treatment initiation. Under current regulatory and treatment paradigms,^{4,12,22} donanemab is administered within a treat-to-clear framework with reassessment of amyloid burden up to 18 months, whereas lecanemab is administered at fixed biweekly dosing, with maintenance options described in some regulatory jurisdictions after 18 months.^{3,25} In the absence of PET-guided reassessment, these 11 patients would reasonably have continued therapy through month 18 under standard dosing schedules, with downstream implications for infusion burden, MRI surveillance, and treatment-slot occupancy (Table 3).

At the same time, repeated amyloid PET cannot be regarded as a neutral or universally scalable resource. Constraints such as scan availability, tracer access, reimbursement heterogeneity, scheduling burden, and radiation exposure are likely to determine whether PET-guided treatment governance is feasible in practice. This perspective is especially pertinent in publicly funded healthcare systems, where implementation of anti-amyloid therapies has raised concerns not only about drug affordability, but also about infusion capacity, MRI monitoring requirements, specialist workforce demands, and overall pathway sustainability.^{7–9,21,22,26} Within such systems, a key practical question is not simply whether amyloid PET adds information, but whether it adds information that is likely to change management in a way that justifies its use.

Pragmatic considerations for treatment-oriented use of amyloid PET

Several practical considerations emerge from current evidence and early real-world implementation.

When anti-amyloid therapy is being considered, amyloid PET may be particularly informative when quantitative assessment of fibrillar amyloid burden is expected to influence subsequent treatment decisions. In this context, baseline PET can provide a reference point for interpreting longitudinal target engagement, especially when treatment exposure may later need to be continued, modified, or discontinued on biological grounds. We do not, however, propose amyloid PET as a replacement for established fluid biomarker pathways, which remain central to etiological diagnosis and patient triage. By contrast, purely diagnostic deployment of amyloid PET should remain proportionate and guided by clinical necessity, particularly in settings where CSF biomarkers are accessible, feasible, and reliable.

Longitudinal amyloid PET is likely to be most useful when its findings are expected to alter management. Rather than endorsing a fixed imaging schedule, reassessment strategies should remain flexible and proportionate to baseline amyloid burden, drug-specific

PET-informed decision group	Patients (n)	Potential infusion appointments avoided ^a	Potential downstream implication
Donanemab—discontinuation after amyloid clearance	7	91	Reduced continuation of monthly treatment, MRI surveillance, and infusion-slot occupancy
Lecanemab—switch after limited amyloid reduction	4	104	Reduced continuation under the initial regimen and supported treatment re-evaluation within the programme
Total	11	195	Potential implications for infusion burden, MRI monitoring, and programme capacity

^aAssuming continued treatment from month 6 to month 18 under standard dosing schedules (13 monthly infusions for donanemab; 26 biweekly infusions for lecanemab). These figures are illustrative and are presented to reflect service-utilization burden rather than formal economic impact.

Table 3: Illustrative service-utilization implications of PET-informed treatment modification.

Search strategy and selection criteria

References for this Viewpoint were identified through searches of PubMed and Embase between January 2015 and April 2026, as well as regulatory and health technology assessment documents from the European Medicines Agency (EMA), the US Food and Drug Administration (FDA), and the National Institute for Health and Care Excellence (NICE). Search terms included “Alzheimer’s disease”, “anti-amyloid monoclonal antibodies”, “lecanemab”, “donanemab”, “amyloid PET”, “treat-to-clear”, “TRAC framework”. Additional references were identified through citation tracking of key publications and consensus documents. No language restrictions were applied. The final reference list was selected on the basis of relevance to regulatory frameworks and treatment governance of anti-amyloid therapy implementation in European healthcare systems.

pharmacodynamic profiles, and system-level constraints. This approach is consistent with the view that amyloid PET may support treatment governance, while not functioning as a standalone determinant of efficacy, safety, or net benefit.

Our real-world experience suggests that the additional burden associated with longitudinal amyloid PET may, within a structured follow-up pathway, be offset by downstream effects on treatment intensity. To date, among the 27 patients who had reached the 6-month follow-up timepoint and completed amyloid PET, imaging findings were associated with treatment modification in 11 cases (40.7%), with practical implications for continuation of infusion therapy, MRI surveillance, and treatment-slot occupancy. This does not constitute a formal economic demonstration, nor does it imply that PET should be used indiscriminately. It does suggest, however, that the burden of PET should not be considered in isolation from its potential to support more proportionate use of high-intensity treatment pathways over time.

In summary, our Viewpoint supports a treatment-oriented role for amyloid PET within structured anti-amyloid care pathways. We do not propose it as a replacement for established biomarker pathways or as a surrogate for individual-level clinical benefit. Rather, our real-world experience suggests that longitudinal amyloid PET may become an increasingly relevant tool for governing treatment exposure in routine care, particularly when its findings translate into practical decisions on continuation, switching, or discontinuation.

Contributors

MF, GC, and FA conceived the manuscript and defined its conceptual framework. MF, GC, and FA wrote the original draft of the manuscript. GC and FA directly accessed and verified the underlying individual participant-level data reported in the manuscript. EGS, AG, GR, SP,

AMSG and AC contributed to critical revision of the manuscript for important intellectual content. All authors reviewed and approved the final version of the manuscript. All authors had full access to the data relevant to the manuscript and had final responsibility for the decision to submit for publication.

Declaration of interests

MF is Editor-in-Chief of the *Journal of Neurology*, Associate Editor of *Human Brain Mapping*, *Neurological Sciences*, and *Radiology*; received compensation for consulting services from Almirall, Biogen, Bristol-Myers Squibb, Eli Lilly, Merck, Novartis, Roche, Sanofi; speaking activities from Amgen, Bayer, Biogen, Bristol-Myers Squibb, Celgene, Chiesi Italia SpA, Eisai, Eli Lilly, Fujirebio, Genzyme, Janssen, Merck, Neopharm, Gentili, Neuraxpharm, Novartis, Novo Nordisk, Roche, Sanofi, Takeda; participation in Advisory Boards for Alexion, Biogen, Bristol-Myers Squibb, Eli Lilly, GE Healthcare Ltd, Merck, Neuraxpharm, Novartis, Roche, Sandoz, Sanofi, Takeda; scientific direction of educational events for Biogen, Merck, Roche, Celgene, Bristol-Myers Squibb, Lilly, Novartis, Sanofi-Genzyme; he receives research support from Biogen Idec, Merck-Serono, Novartis, Roche, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla. GC has received speaker honoraria from Neopharm, Gentili and Eli Lilly. AG and EGS have received speaker honoraria from Eli Lilly. GR has nothing to disclose. SP has nothing to disclose. AMSG has nothing to disclose. AC reports consulting or advisory role for Blue Earth Diagnostics, Telix Pharmaceuticals, InnovaRadi Therapeutic, and General Electric Healthcare; and Speaker’s Bureaus for Bracco Diagnostics, General Electric Healthcare, Novartis, Telix Pharmaceuticals, and United Imaging; he is Editor in Chief of *The EANM Journal*. FA is Associate Editor of *NeuroImage: Clinical* and the *European Journal of Neurology*; has received speaker honoraria from Biogen Idec, Bristol Myers Squibb, Eisai, Eli Lilly, GE Healthcare, Neuraxpharm, and Roche; and receives or has received research supports from the Italian Ministry of Health, the Italian Ministry of University and Research, AriSLA (Fondazione Italiana di Ricerca per la SLA), the European Research Council (ERC), the EU Joint Programme—Neurodegenerative Disease Research (JPNDR), and Foundation Research on Alzheimer Disease (France).

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