MEDICAL POLICY

Medical Policy Title	**Positron Emission Tomography (PET) – Head Imaging**
Policy Number	**6.01.47**
Current Effective Date	**May 15, 2026**
Next Review Date	**January 2027**

Our medical policies are guides to evaluate technologies or services for medical necessity. Criteria are established through the assessment of evidence based, peer-reviewed scientific literature, and national professional guidelines. Federal and state law(s), regulatory mandates and the member's subscriber contract language are considered first in the determination of a covered service. (Link to Product Disclaimer)

POLICY STATEMENT(S)

I. PET imaging is considered **medically appropriate** for the following indication:

A. For the diagnosis of dementia, **ALL** of the following criteria are required:

1. Date of onset of symptoms with documentation of six (6) months of cognitive decline based on a detailed history of memory loss with impairment of day-to-day activities confirmed by family members or others with knowledge of the individual's status;
2. Results of bedside testing and/or neuropsychological testing can be performed when history and bedside mental status examination cannot provide a confident diagnosis (see policy guidelines);
3. Results of any structural imaging (MRI or CT Head) performed; **and**
4. Presumptive causes or etiology/ies of dementia;
   a. Cannot occur exclusively during bouts of delirium; **and**
   b. Cannot be explained by another mental disorder.

II. Metabolic (FDG) Brain PET (CPT 78608) is considered **medically necessary** for **ANY** of the following indications when **ALL** of the criteria are met:

A. Mild Cognitive Impairment (MCI) evaluation when Alzheimer's Disease (AD) is suspected, and **all** of the following criteria are met:

1. Established diagnosis of mild cognitive impairment based on clinical history, physical examination, and cognitive testing, which may include neuropsychological testing;
2. Documentation of cognitive decline obtained by **either** of the following:
   a. A detailed history of cognitive decline with impairments confirmed by family members or others with knowledge of the individual's status; **or**
   b. Abnormal mental status test score or neuropsychological test results consistent with MCI/mild neurocognitive disorder (see policy guidelines); **and**
3. Results of any structural brain imaging (MRI Brain or CT head) previously performed;

B. Dementia evaluation to distinguish between AD and Frontotemporal Dementia (FTD) when **all** of the following criteria are met:
    1. Established diagnosis of dementia:
        a. Date of onset of symptoms with documentation of six (6) months of cognitive decline;
        b. Documentation of a decline in cognitive function obtained with **either** of the following:
            i. A detailed history of memory loss with impairment of day-to-day activities confirmed by family members or others with knowledge of the individual's status; **or**
            ii. Abnormal mental status testing score consistent with dementia (see policy guidelines);
        c. Results of any structural imaging (MRI Brain or CT Head) previously performed;
        d. Meets diagnostic criteria for AD and FTD;
        e. The results are expected to clarify the diagnosis between FTD and AD and help guide future treatment;
        f. Cause of clinical symptoms is uncertain; **and**
        g. Evaluation has ruled out specific alternative neurodegenerative disease or causative factors;
            i. Cannot occur exclusively during bouts of delirium;
            ii. Cannot be explained by another mental disorder;

C. Differentiate Lewy Body Dementia (LBD) from AD when **all** of the following criteria are met:
    1. Established diagnosis of dementia:
        a. Date of symptom onset, with documentation of six (6) months of cognitive decline;
        b. Documentation of decline in cognitive function obtained with **either** of the following:
            i. A detailed history of memory loss with impairment of day-to-day activities confirmed by family members or others with knowledge of the individual's status; **or**
            ii. Abnormal mental status test score consistent with dementia/major neurocognitive disorder (see policy guidelines);
        c. Presumptive causes of dementia have been excluded including **all** of the following:
            i. Cannot occur exclusively during bouts of delirium;
            ii. Cannot be explained by another mental disorder; **and**

d. Results of any structural imaging (MRI or CT head) performed.

D. Suspected encephalitis evaluation, including autoimmune encephalitis when:

1. Diagnosis remains unclear after evaluation with MRI Brain, CSF analysis, and/or lab testing including serology.

E. Pre-surgical evaluation of refractory seizure when requested by neurosurgeon or neurologist or a provider consulting with either.

III. FDG PET brain (CPT 78608) is considered **not medically necessary** for the purpose of diagnosis and management of other forms of dementia including but not limited to **ANY** of the following:

A. Parkinson's disease;

B. Normal pressure hydrocephalus;

C. Chronic traumatic encephalopathy.

IV. Fluorodopa F18 (F-Dopa) PET Brain (CPT 78608) is **medically necessary** to evaluate motor symptoms in suspected Parkinsonian Syndromes (Parkinson Disease, Multiple System Atrophy, Progressive Supranuclear Palsy, Corticobasal Degeneration) when the diagnosis is unclear, in order to differentiate from non-neurodegenerative disorders.

V. Amyloid Brain PET (CPT 78814 or 78811) is **medically necessary** when a diagnosis of MCI or dementia due to Alzheimer's disease is suspected based on cognitive testing and the evaluation of structural brain imaging, for **ANY** of the following indications:

A. Individuals with MCI or dementia who are less than 65 years of age and in whom AD is suspected;

B. Individuals with MCI or dementia that could be consistent with amnestic AD pathology with onset at greater or equal to 65 years of age;

C. Individuals with MCI or dementia that could be consistent with AD but has atypical clinical features;

D. Individuals with MCI or dementia with equivocal or inconclusive results on CSF biomarkers;

E. To inform the prognosis of individuals with MCI due to suspected AD pathology;

F. To determine eligibility for treatment with amyloid targeting therapy and to monitor response (applicable to the specific drug).

VI. Tau PET Brain (CPT 78814 or 78811) is **medically necessary** for the evaluation of cognitive impairment in individuals with suspected AD with atypical clinical presentation when **ALL** of the following criteria are met:

1. Established diagnosis of mild cognitive impairment or dementia;

2. Evaluation has excluded other causes; **and**

3. Results of structural brain imaging are available to the ordering provider.

VII. Perfusion PET Brain (CPT 78609) is **not medically necessary** in the evaluation of dementia.

VIII. PET imaging is considered **not medically necessary** in the evaluation of individuals with **EITHER** of the following:

A. Autism spectrum disorders;

B. Evaluation of traumatic brain injury/head trauma;

IX. Amyloid Brain PET imaging for the evaluation of stroke is considered **not medically necessary**.

X. FDG-PET/CT imaging for vasculitis is considered **investigational**.

RELATED POLICIES

Corporate Medical Policy

2.01.50 Neuropsychological Testing

6.01.29 Positron Emission Tomography (PET) Oncologic Applications

11.01.03 Experimental or Investigational Services

Pharmacy Policy

100 Anti-Amyloid Directed Therapies

POLICY GUIDELINE(S)

I. Examples of abnormal bedside mental status test results consistent with MCI include:

A. Montreal Cognitive Assessment Survey (MoCA) with score 19-25 <26

B. St. Louis University Mental Status (SLUMS) with score for high school education 21-26, less than high school education 20-24. <21 or the Eight-item Informant Interview to Differentiate Aging and Dementia (AD8) Dementia Score >2.

C. Mini-Mental State Exam (MMSE) with score 22-26 <26, Memory Impairment Screen (MIS) with score <5,

II. Subacute head trauma is defined as trauma to the head within seven (7) days to three (3) months post-trauma.

III. 3D rendering, (CPT code 76376 or 76377), should not be billed in conjunction with PET imaging.

DESCRIPTION

PET scanning is an imaging technology that can reveal metabolic information in various tissue sites. It is the metabolic information that distinguishes PET scanning from other imaging modalities, such as magnetic resonance imaging (MRI) and computed tomography (CT), which provide primarily anatomic information. PET scans measure concentrations of radioactive chemicals that are partially metabolized in the body. PET scans are based on the use of positron emitting radionuclide tracers coupled to organic molecules such as glucose, ammonia, or water. Dedicated PET scanners consist of multiple detectors arranged in a full or partial ring around the patient.

A variety of radiotracers are used for PET scanning, including fluorine-18, rubidium-82, ammonia N-13, carbon-11, oxygen-15, and nitrogen-13. Fluorine-18 is often coupled with FDG as a means of detecting glucose metabolism, which, in turn, reflects the metabolic activity, and, thus, viability, of the target tissue. Because of their short half-life, tracers must be made locally. Except for fluorine and rubidium, all the tracers must be manufactured with an on-site cyclotron.

Florbetapir (Amyvid, Avid Radiopharmaceuticals), is a radioactive dye for visualization of amyloid plaque in the brain. Amyvid is indicated for PET imaging of the brain, to estimate beta-amyloid neuritic plaque density in adult patients with cognitive impairment who are being evaluated for AD and other causes of cognitive decline.

Flutemetamol F18 is not indicated to predict the development of AD or to check how patients respond to treatment for AD. Flutemetamol F18 PET images should be interpreted only by health care professionals who successfully complete training in an image interpretation program.

SUPPORTIVE LITERATURE

Sims et al (2023) conducted the TRAILBLAZER-ALZ 2 trial, it is a randomized, double blinded, placebo-controlled, 18-month phase 3 trial included 1736 participants with early symptomatic Alzheimer disease and amyloid and tau pathology. The primary objective of this study was to assess the efficacy and adverse events of donanemab. Randomized participants received donanemab or placebo every 4 weeks for up to 72 weeks. Amyloid plaque level was assessed at 24 and 52 weeks, and if it was less than 11 CL on any PET scan or less than 25 but greater than or equal to 11 CL on two consecutive PET scans (TRAILBLAZER-ALZ cutoffs), donanemab was switched to placebo in a blinded procedure. The percentages of donanemab-treated participants in the low/medium tau population who reached amyloid clearance were 34.2% at 24 weeks, 80.1% at 76 weeks compared with 0.2% at 24 weeks and 0% at 76 weeks of placebo-treated participants. In the combined population, amyloid clearance was reached in 29.7% of participants at 24 weeks and 76.4% at 76 weeks of donanemab-treated participants compared with 0.2% at 24 weeks and 0.3% at 76 weeks of placebo-treated participants. Limited-duration dosing was a distinct trial design feature reflecting donanemab binding specificity for amyloid plaque and implemented to decrease burden, cost, and potentially unnecessary treatments. Early significant changes on both brain amyloid PET scans and P-tau217 blood test results suggest opportunities for clinical monitoring of therapy.

Scherbinin et al (2022) conducted the TRAILBLAZER-ALZ trial, it is a placebo-controlled, randomized control study, with a double blinded period of up to 76 weeks and a 48 week follow up period. The objective was to perform post hoc analyses of amyloid reduction after donanemab treatment and assess its association with tau pathology and clinical measures. The trial contained 272 Participants from age 60-85 years old, with gradual and progressive change in memory function for 6 months or more, early symptomatic Alzheimer disease. Elevated amyloid, and intermediate tau levels. A blinded dose reduction evaluations occurred at 24 and 52 weeks based on amyloid clearance. To evaluate the effect of baseline amyloid levels on the probability of participants to reach complete amyloid clearance at 24, 52, and 76 weeks, 3 logistic regressions were run respectively, with amyloid PET results (complete clearance or partial clearance) at weeks 24, 52, and 76 as the dependent variables, and baseline amyloid PET as the only independent variable. Probabilities of reaching complete

clearance were provided along with corresponding 95% CIs. Analysis of the 19 participants who underwent all 4 amyloid PET scans and reached less than 11 Centiloids (CL) by 24 weeks (and therefore discontinued donanemab treatment) showed that the achieved amyloid clearance was sustained with a mean (SD) rate of reaccumulation of 0.02 (7.75) CL per year over the 1-year period in the trial. In addition, an exposure-response model (model based on data from 304 participants, including data from the phase 1b donanemab study) of amyloid plaque level for all available scans suggests that in participants who achieved an amyloid load of 11 CL or less at week 24 and discontinued amyloid treatment, the median time to reaccumulate amyloid from an 11 CL to 24.1 CL threshold could be 3.9 years (95% prediction interval, 1.9-8.3 years). This data reinforces the phase 1b results that showed no significant reaccumulation over 72 weeks after a single dose.

Wilkens et al (2022) conducted a secondary analysis of the Imaging Dementia-Evidence for Amyloid Scanning (IDEAS) single-arm multisite cohort study. The objective was to analyze racial and ethnic differences in amyloid PET scan positivity among individuals with mild cognitive impairment (MCI) or dementia using data from the IDEAS cohort of Medicare beneficiaries. Among 17,107 participants, White individuals showed higher rates of amyloid PET positivity compared to Asian and Hispanic participants, but not significantly different from Black participants. Adjusted models revealed lower odds of positive scans for Asian (OR 0.47), Black (OR 0.71), and Hispanic (OR 0.68) individuals compared to White individuals. The study suggests potential differences in the underlying causes of cognitive impairment across racial and ethnic groups, which may inform future diagnostic, treatment, and prevention strategies for Alzheimer's disease.

Swanson et al (2021) conducted a randomized, double-blind, phase 2b proof-of-concept clinical trial analyzing early Alzheimer's disease with lecanemab (BAN2401). Lecanemab (Leqembi) is an IgG1 monoclonal antibody, which preferentially targets soluble aggregated anti- amyloid beta (Aβ), with activity across oligomers, protofibrils, and insoluble fibrils. In the study they utilized the Bayesian design with response-adaptive randomization to assess three doses across two regimens of lecanemab versus placebo in early Alzheimer's disease, mild cognitive impairment due to Alzheimer's disease (AD) and mild AD dementia. They aimed to establish the effective dose 90% (ED90) (the simplest dose that achieves greater than or equal to 90% of the maximum treatment effect). The primary endpoint was Bayesian analysis of 12-month clinical change on the Alzheimer's Disease Composite Score (ADCOMS) for the ED90 dose, which required an 80% probability of ≥25% clinical reduction in decline versus placebo. Key secondary endpoints included 18-month Bayesian and frequentist analyses of brain amyloid reduction using PET; clinical decline on ADCOMS, Clinical Dementia Rating-Sum-of-Boxes (CDR-SB), and Alzheimer's Disease Assessment Scale-Cognitive Subscale (ADAS-Cog14); changes in CSF core biomarkers; and total hippocampal volume (HV) using volumetric magnetic resonance imaging (vMRI). 854 randomized subjects were treated (lecanemab, 609; placebo, 245). BAN2401-G000-201 did not meet the 12-month primary endpoint. However, prespecified 18-month Bayesian and frequentist analyses demonstrated reduction in brain amyloid accompanied by a consistent reduction of clinical decline across several clinical and biomarker endpoints. Numerous clinical trials are underway, but currently there is no literature that supports amyloid brain PET imaging pre- and post- treatment outside of a clinical trial.

PROFESSIONAL GUIDELINE(S)

- Rabinovici et al (2025) reported The Alzheimer's Association and Society of Nuclear Medicine and Molecular Imaging Appropriate Use Criteria (AUC) for amyloid and tau PET. Each clinical scenario is rated on a scale, 1-3 is rarely appropriate, 4-6 is uncertain and 7-9 is appropriate:

Amyloid PET

- Patients younger than 65 years with suspected AD pathology, presenting with mild cognitive impairment or dementia (9).
- Patients 65 years or older presenting with MCI or dementia syndrome, often consistent with AD pathology (amnestic presentation) (8).
- Patients presenting with MCI or dementia syndrome that could be consistent with AD pathology but has atypical features (8).
- Patients with MCI or dementia with equivocal or inconclusive results on recent CSF biomarkers (8).
- Clinically suspected AD pathology in patients with MCI to inform prognosis (8).
- To determine eligibility for treatment with an approved amyloid targeting therapy (8).
- To monitor response in patients that have received an approved amyloid targeting therapy (6).

Tau PET

- Patients younger than 65 years with suspected AD pathology, presenting with mild cognitive impairment or dementia (8).
- Patients presenting with MCI or dementia syndrome that could be consistent with AD pathology but has atypical features (7).
- Clinically suspected AD pathology in patients with MCI to inform prognosis (7).
- Patients presenting with dementia due to clinically suspected AD pathology to inform prognosis (7).
- To determine eligibility for treatment with an approved amyloid-targeting therapy (8).

REGULATORY STATUS

The United States Food and Drug Administration (FDA) is responsible for ensuring the safety, efficacy, and quality of drugs sold in the United States. This includes both prescription and over-the-counter medications. Refer to the FDA Drug website. Available from: https://www.fda.gov/drugs [accessed 2025 Dec 04]

The FDA maintains information for consumers and health professionals on new drug warnings and other safety information, drug label changes, and shortages of medically necessary drug products. Available from: Drug Safety and Availability | FDA [accessed 2025 Dec 04]

Florbetapir (Amyvid, Avid Radiopharmaceuticals) was approved by the United States Food and Drug Administration (FDA) in 2012.

Flutemetamol F18 injection (Vizamyl, GE Healthcare), was approved by the FDA in October 2013.

Florbetaben F18 (Neuraceq, Piramal Life Sciences, Matran, Switzerland) was FDA approved in 2014.

CODE(S)

- Codes may not be covered under all circumstances.
- Code list may not be all inclusive (AMA and CMS code updates may occur more frequently than policy updates).
- (E/I)=Experimental/Investigational
- (NMN)=Not medically necessary/appropriate

CPT Codes

Code	Description
78608	Brain imaging, positron emission tomography, (PET); metabolic evaluation
78609	perfusion evaluation
78811	Positron emission tomography (PET) imaging; limited area (e.g., chest, head/neck)
78814	Positron emission tomography (PET) with concurrently acquired computed tomography (CT) for attenuation correction and anatomical localization imaging; limited area (e.g., chest, head/neck)

HCPCS Codes

Code	Description
A9552	Fluorodeoxyglucose F-18 FDG, diagnostic, per study dose, up to 45 millicuries
A9586	Florbetapir F18, diagnostic, per study dose, up to 10 millicuries (e.g., Amyvid)
A9598	Positron emission tomography radiopharmaceutical, diagnostic, for non-tumor identification, not otherwise classified
A9601	Flortaucipir f 18 injection, diagnostic, 1 millicurie (e.g., Tauvid)
A9602	Fluorodopa f-18, diagnostic, per millicurie
Q9982	Flutemetamol F18, diagnostic, per study dose, up to 5 millicuries (e.g., Vizamyl)
Q9983	Florbetaben F18, diagnostic, per study dose, up to 8.1 millicuries (e.g., Neuraceq)

ICD10 Codes

Code	Description
D33.0-D33.9	Benign neoplasm of brain and other parts of central nervous system (code range)

Code	Description
F01.50-F03.91	Dementia due to known physiological conditions (code range)
G30.0-G30.9	Alzheimer Disease (code range)
G40.001-G40.219	Localization-related (focal) (partial) idiopathic epilepsy and epileptic syndromes with seizures of localized onset (code range)
G40.301-G40.319	Generalized idiopathic epilepsy and epileptic syndromes (code range)
G40.901-G40.919	Epilepsy, unspecified (code range)

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SEARCH TERMS

Not Applicable

CENTERS FOR MEDICARE AND MEDICAID SERVICES (CMS)

FDG PET for Dementia and Neurodegenerative Diseases (NCD 220.6.13) [accessed 2025 Nov 13]

PRODUCT DISCLAIMER

- Services are contract dependent; if a product does not cover a service, medical policy criteria do not apply.
- If a commercial product (including an Essential Plan or Child Health Plus product) covers a specific service, medical policy criteria apply to the benefit.
- If a Medicaid product covers a specific service, and there are no New York State Medicaid guidelines (eMedNY) criteria, medical policy criteria apply to the benefit.
- If a Medicare product (including Medicare HMO-Dual Special Needs Program (DSNP) product) covers a specific service, and there is no national or local Medicare coverage decision for the service, medical policy criteria apply to the benefit.
- If a Medicare HMO-Dual Special Needs Program (DSNP) product DOES NOT cover a specific service, please refer to the Medicaid Product coverage line.

POLICY HISTORY/REVISION

Committee Approval Dates	
01/22/26	
Date	**Summary of Changes**
01/22/26	• This new policy addresses the criteria for PET head imaging which was removed from CMP#6.01.07. Policy statement added for amyloid brain PET imaging with medically necessary criteria. Policy statement added for perfusion PET brain as not medically necessary.
01/01/25	• Summary of changes tracking implemented.