FDG PET IMAGING IN DEMENTIA (FTD vs AD)

Requirements for getting an FDG PET

- FDG PET imaging of dementia is not an FDA approved indication, but CMS has agreed to reimburse if certain prerequisites are met.
- Initial medical visit to document symptoms that meet criteria of both AD and FTD
- Neuropsychological testing confirming dementia that meets some of both diagnoses
- 6 month follow-up that demonstrates persistence of symptoms
- Anatomical MRI
- Insurance preauthorization

Case Study: Neuropsychological testing

- Evaluates:
  - Intellectual/overall functioning
  - Attention/processing speed
  - Motor skills
  - Visuospatial/construction
  - Language
  - Comprehension
  - Memory
  - Executive functioning
  - Psychological functioning

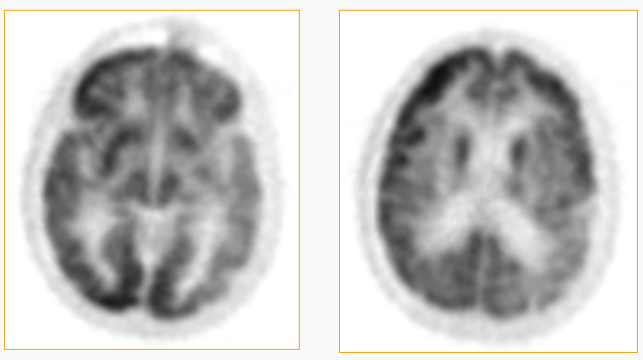
- *A more extensive battery of tests administered by a neuropsychologist that provides more in depth and specific results and takes 3-4 hours*
- *Given the multiple areas of cognitive deficits and the indication of some functional decline, a diagnosis of an early dementia appears supported at this time. The etiology of this dementia, however, is less clear. There are some indicators (e.g., severe impairments in verbal memory that do not improve with cueing, naming difficulties, relatively worse semantic than phonemic fluency) that might suggest Alzheimer's disease (AD) as the pathological condition. There are also indicators (e.g., relative impairments on executive functioning measures, notable language difficulties, early behavioral presentation) that suggest frontotemporal dementia (FTD) as the possible cause. Additionally, the relatively early onset of the cognitive symptoms is more suggestive of FTD than AD. PET imaging may be useful this differential diagnosis. It should also be noted that cognitive data suggests some asymmetry in the cognitive deficits, with relatively greater impairments in the language-based skills (i.e., left cerebral hemisphere) than the visually-based skills (e.g., right cerebral hemisphere).*

Case Study: 2nd clinical visit – 6 months later

- Reviews previous mental status and compares to current level
- There needs to be evidence that cognitive decline has been present for six months

- *The primary encounter diagnosis was Dementia without behavioral disturbance, unspecified dementia type.*
- *Diagnoses of Aphasia syndrome, Obstructive sleep apnea syndrome on CPAP, Cardiac pacemaker, and Essential tremor, head tremor were also pertinent to this visit.*
- *Increase in medications: busPIRone (BUSPAR) 5 mg tablet (Depression), cyanocobalamin (VITAMIN B-12) 1000 mcg tablet, cyanocobalamin 1000 mcg/mL injection (unknown reason), meclizine (ANTIVERT), traZODone (DESYREL) 50 mg tablet (sleep aid), donepezil (ARICEPT) 10 mg Tab, losartan (COZAAR) 50 mg Tab, Memantine HCl ER 28 mg CAPSULE SR 24 HR, sertraline (ZOLOFT) 100 mg Tab*
- *MoCA score is 17. This is equivalent to an MMSE score of 23 and represents a 1 point improvement from his previous MoCA score.*
- *2nd Assessment: Mild dementia. Evaluation incomplete. This patient has an atypical dementia syndrome with prominent language deficits. Included in the differential diagnosis are language predominant Alzheimer's disease dementia, and progressive aphasia due to frontal temporal degeneration.*

Case Study: FDG PET After 2nd clinical visit



PROCEDURE: The patient was fasted for 6 hours, resulting in a blood glucose of 84 mg/dl. 9.58 mCi of 18-F FDG was injected intravenously. 30 minutes later, a PET/CT scan of the brain was performed. Emission data was corrected using the CT acquisition.

FINDINGS: There is generalized atrophy. The sylvan fissures are prominent bilaterally, particularly on the left. There is minimally diminished FDG uptake in the left temporal cortex. Metabolism in the right temporal cortex is preserved. There is a heterogeneous mild to moderate reduction in metabolism in the left parietal cortex. There is preservation of metabolism in the right parietal cortex. Metabolism is preserved in the frontal cortex bilaterally. There is mild metabolic reduction noted in the left precuneus and posterior cingulate cortex. The associated right posterior cingulate and precuneus cortex has preserved metabolism. Metabolism in the primary visual cortex is preserved. There is subtly diminished FDG metabolism in the right cerebellum consistent with crossed cerebellar diaschisis.

IMPRESSION: Metabolic deficits as described above. There is heterogeneous reduction of metabolism in the left temporal, parietal, posterior cingulate and precuneus cortex. The metabolic reduction is mild with only one area of moderate metabolic reduction noted in the left parietal cortex. The most profound reduction is in the parietal and precuneus on the left. In this particular patient the metabolic reductions are primarily mild with some more focal moderate FDG reduction. The observed metabolic pattern at this point in time is not classic for a particular neurodegenerative process. Early frontotemporal dementia cannot be totally excluded but is unlikely. The lack of a metabolic reduction in the frontal and anterior temporal cortex makes the diagnosis of primary progressive aphasia, a subtype of FTD, unlikely. Early Alzheimer's disease is a possible diagnosis due to the metabolic reductions noted in the left lateral temporal, parietal and precuneus cortex. If the patient's cognitive difficulties continue to worsen, repeating the FDG-PET/CT scan in approximately 18-24 months may be appropriate.

SOCIETY OF NUCLEAR MEDICINE AND MOLECULAR IMAGING

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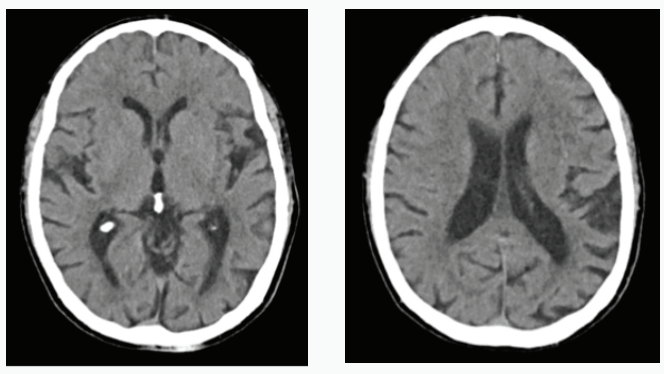
Case Study Example: 72 year old male

INITIAL CLINICAL VISIT

- Primary complaint
  - 72 year old male with memory loss
  - Initial symptoms presented 8 years ago (aphasia & involuntary movements)
- Possible causes
  - Anxiety and Depression
  - Sleep apnea, untreated
  - Vascular disease
  - Significant head injury 60 years ago and 3 years ago
  - Neurodegenerative disease
  - No family history of dementia
- Initial assessments
  - MMSE = 23
  - MoCA = 16
- Initial Diagnosis
  - Moderate dementia

- *Patient was taking at the time of visit : Donepezil 10mg, Memantine 28mg, Sertraline 100mg, Losartan 60mg*
- *MoCA is the Montreal Cognitive Assessment, a cognitive screening tool for Mild Cognitive Impairment. It is a 30 point test that takes 10 minutes. Scores are from 0 to 30. A score above 26 is considered normal. MCI average is 22 and dementia average score is 16. Patient had cognitive deficits in visuospatial/executive, attention, abstraction, and recall*
- *MMSE is the Mini-Mental State Examination, is a 30 point questionnaire to assess cognitive impairment. Score is 0-30, above 24 is normal, 19-23 mild, 10-18 moderate and <9 severe cognitive impairment.*

Case Study: Anatomical imaging (CT)



- *Examination: Non-contrast head CT*
- *Pacemaker precluded use of MRI*
- *Technique: Axial 5 mm non-contrast images were obtained from the skull base to the vertex, with no contrast administered.*

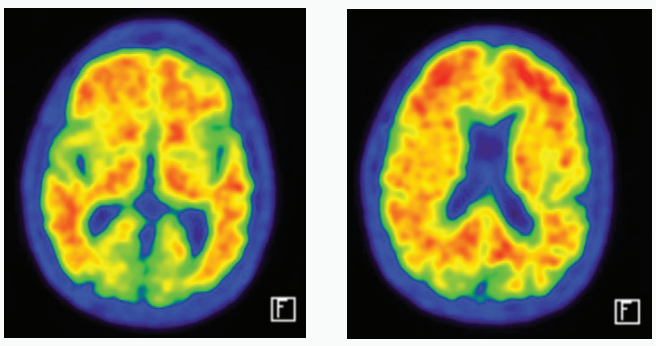
FINDINGS:

- *Gray-white matter differentiation is preserved. There is no evidence of acute territorial infarct, intracranial hemorrhage or mass effect. No extra-axial fluid collection is present.*
- *Diffuse cerebral and cerebellar atrophy.*
- *Ventricles are symmetric. There is no hydrocephalus. Basal and suprasellar cisterns are not effaced. No tonsillar or uncal herniation is present.*
- *Orbits appear normal. Osseous structures are unremarkable. Right maxillary mucoid retention cyst.*

IMPRESSION:

- *Global atrophy of the cerebrum and cerebellum without evidence of lobar pattern dementia.*

Case Study: Amyloid PET



PET CT BRAIN VIZAMYL

INDICATION: Beta-amyloid neuritic plaque density assessment in an adult patient with cognitive impairment who is being evaluated for Alzheimer's Disease (AD) and other causes of cognitive decline

EXAMINATION: Vizamyl (flutemetamol F-18 Injection) PET/CT scan of the brain

PROCEDURE: 4.79 mCi of Vizamyl (flutemetamol F-18 Injection) was injected intravenously. 90 minutes later, a PET/CT scan of the brain was performed. Emission data was corrected using the CT acquisition. Reconstructed emission images were interpreted using transaxial, coronal, and sagittal views. Images were interpreted by comparing the radioactivity in cortical gray matter cerebral cortex with activity in the adjacent white matter. Semi-quantitative analysis was performed by Cortex ID software, comparing activity in specific cortical regions to that in the pons.

FINDINGS: There is diffuse intense increased deposition of radiopharmaceutical throughout the cerebral cortex, including the basal ganglia. Areas of most prominent accumulation include inferior frontal, anterior cingulate, posterior cingulate and precuneus. The composite CortexID uptake ratio is 0.96, which is strongly abnormal.

IMPRESSION: Positive for cortical beta amyloid. Diffuse prominent uptake throughout the cerebral cortex and basal ganglia. CortexID uptake ratio is 0.96.

Post imaging visit

- This patient has Alzheimer's disease dementia.

More specifically he meets NIA–AA criteria for probable Alzheimer's disease dementia with biomarker probability high. This is our highest degree of clinical diagnostic certainty despite his atypical presentation of progressive aphasia. Now with clear and confident diagnosis we will initiate proactive care planning. He is already taking standard drug treatment for Alzheimer's disease dementia which should be continued. Continued anxiety and sleep problems will need to be addressed. I also recommend local speech therapy to improve communication and to provide alternative communication strategies since I expect that his language problems will worsen over time.