

¹⁸F FDG PET/CT of the Brain

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1. Introduction

Glucose is the main energy supplier of the brain. Therefore the uptake of FDG is high, especially in the cortex, thalamus and striatum. Glucose metabolism in the brain is impaired in many neurological disorders which can therefore be diagnosed with FDG-PET-CT. Specific patterns of hypo-and hyper metabolism have been identified for different types of dementia and movement disorders. Furthermore FDG-PET can be used to localize the functional deficit zone in epilepsy. However due to the high FDG uptake in the brain, FDG-PET is much less useful in detection of primary brain lesions or metastasis.

2. Methodology

This guideline is based on available scientific literature on the subject, the previous guideline (Aanbevelingen Nucleaire Geneeskunde 2007), international guidelines from EANM and/or SNMMI if available and applicable to the Dutch situation.

3. Indications

- Dementias: such as Alzheimer's disease, dementia with Lewy Bodies, corticobasal degeneration and frontotemporal dementia (primary progressive aphasia and its behavioural variant)
- Differentiation of the movement disorders Parkinson's disease versus multiple system atrophy, progressive supranuclear palsy, and corticobasal degeneration
- Some neuro-oncological conditions (detection of recurrence, grading, primary CNS lymphoma)
- Pre-operative analysis in epilepsy

4. Relation to other diagnostic procedures

Although FDG-PET-CT of the brain does not have a role in the initial diagnosis of these disorders, it can be useful when the clinician is not able to make the diagnosis based upon neuro(psycho)logical examination, laboratory findings and/or first choice (anatomical) imaging MRI /CT.

5. Medical information necessary for planning

- Symptoms
- Differential diagnosis
- Medication and when last taken.
- Conclusions of other imaging modalities
- Relevant history (recent interventions, chemotherapy, radiotherapy)
- Special clinical conditions which could complicate the procedure
- Diabetes mellitus
- Patient's length and weight

6. Radiopharmaceutical

Tracer: ¹⁸F-fluorodeoxyglucose
 Nuclide: Fluorine-18
 Activity: 150-250 MBq, children 2-4 MBq/kg
 Administration: Intravenous

7. Radiation safety

FDG-PET-CT should not be performed in pregnancy
 Breast feeding need not be discontinued (ICRP publication 106, Annex D).

Organ receiving the largest radiation dose		Dose (mGy/MBq)	Effective Dose (mSv/MBq)	Recommended Dose (MBq)	Effective Dose (mSv)
Adults	Organ				
Adults	Bladder wall	1,3	0,019	150-250	2,85-4,75
		4,7	1y 0,095	20-40	1,9-3,8
		3,4	5y 0,056	40-80	2,2-4,4
Children	Bladder wall	2,5	10y 0,037	65-130	2,4-4,8
		1,6	15y 0,024	90-180	2,2-4,3

- The effective dose of the CT scan is approximately 20 µSv for low dose and 220-450 µSv for high dose/diagnostic CT scan, depending on the collimation and scan type.

8. Patient preparation/essentials for procedure

Patients should fast for at least 4-6 h.
 Drugs that may affect cerebral glucose metabolism should be avoided. They should be discontinued on the day of the PET examination, clinical situation permitting.
 Diabetic patients should not have their medication altered.

Before scanning:

- Check blood glucose level prior to FDG administration.
- Administer FDG in a quiet room with diminished lights and preferably at least 10 min after placement of the intravenous access.
- The patient must lie still for at least 20 min after the FDG injection, speaking, reading or any other activities are not allowed.
- If sedation is necessary, it should be given at least 20 min after FDG injection.
- Continuous monitoring of patients before and during scanning is necessary. For pre-operative evaluation of epilepsy, EEG monitoring is required. This should start prior to FDG administration and continue for at least 20 min post injection.
- Patients should void their urinary bladder just before scanning.

9. Acquisition and processing

The patient should be placed in a comfortable position with the head fixed, using the

orbitomeatal line as a reference. State of the art PET-CT scanner with full ring detectors LSO/LYSO/BGO/GSO crystal. Acquisitions should not be started less than 30 min after injection and may start up to several hours after injection for detection of tumours. Acquisition-time 6 min (3D-mode) CT-based attenuation correction with low-dose CT scan if only for attenuation purposes. Image reconstruction in the form of transaxial images with a matrix of at least 128x128. The reconstruction methods and choices of iterations, subsets and smoothness depend on the type of camera and recommendations of the manufacturer. Use a continuous colour scale for displaying the images. Absolute quantification is not possible with static FDG images. Only dynamic data acquisition with arterial sampling allows for absolute quantification. These protocols are beyond the scope of this document. See European Association of Nuclear Medicine and Society of Nuclear Medicine guidelines. However, for static imaging, different software programs for voxel based quantification and brain mapping with or without normal databases are available.

10. Interpretation

- a. The images should be carefully checked for :
 - movement artefacts
 - attenuation artefacts
 - misalignment of the head
- b. The physician should be aware of the variations in normal glucose brain metabolism, especially age dependent changes as well as medication related changes.
- c. Preferably use standardized threshold settings and scaling methods to avoid unnecessary variation and for optimal personal reference building.
- d. Additional quantification is recommended. Be aware of the specific limitations of the quantification software being used. The conclusion should never be based upon the quantification results alone.
- e. Specific patterns of FDG metabolism in different forms of dementia have been described. In Alzheimer disease there is hypometabolism in the parietal, temporal and posterior cingulate cortex. There is relative sparing of the primary sensorimotor and primary visual cortices, striatum, thalamus and cerebellum. Whereas the occipital lobe is more likely to be involved in Dementia with Lewy bodies. The latter having an abnormal DAT-scan, which is normal in patients with Alzheimer's disease. In frontotemporal dementias hypometabolism is more pronounced in the frontal and anterior temporal cortex. In vascular dementia the cortical hypometabolism follows the vascular damage and has no specific pattern.
- f. In Parkinson's disease one of the characteristic features is a normal to relatively high metabolism in the striatum (symmetrical, especially in the putamen), whereas striatal FDG metabolism is reduced in multiple system atrophy (symmetrical hypometabolism), progressive supranuclear palsy (predominantly head of the caudate nucleus) and corticobasal degeneration (asymmetrical).

11. Report

Report the amount of activity injected, interval between injection and scanning, the scanned area, use of CT and blood glucose level. Also mention conditions that could influence the images such as movement artefacts.

Systematically describe the cortex, basal ganglia, thalamus and striatum, look for asymmetry and relate to known patterns. Describe areas of abnormal metabolism and demarcation.

Correlate with earlier brain imaging modalities if available.

12. Literature

- ICRP Publication 106 :Radiation Dose to Patients from Radiopharmaceuticals Addendum 3 to ICRP Publication 53.
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