

[¹⁸F]-Fdg Pet Identified Superior Colliculi Hypometabolism in Progressive Supranuclear Palsy

Alice Jaillard^{1*}, Grégory Petyt¹, Maxime Morelle¹, Caroline Moreau^{2,3} and Franck Semah^{1,3}

¹CHU Lille, Nuclear Medicine Department, F-59000 Lille, France

²CHU Lille, Neurology Department, F-59000 Lille, France

³Inserm, U1171, F-59000 Lille, France

Abstract

Hypometabolism in the fronto-mesial cortex and in the anterior cingulate cortex were reported in patients with progressive supranuclear palsy (PSP). Severe neuronal loss, associated with gliosis and neurofibrillary tangles were reported in the superior colliculi of patients with PSP in the initial clinico-neuropathological description of the disease by Steele, Richardson and Olszewski. Nowadays, high-resolution positron emission tomography with [fluorine-18] fluoro-D-glucose (¹⁸F-FDG PET) with magnetic resonance (MR) image fusion can reveal hypometabolism in the superior colliculi in patients with PSP.

Keywords: Progressive supranuclear palsy; 18F-FDG PET; Superior colliculi

Introduction

Clinical use of ¹⁸F-FDG PET is well established in neurodegenerative disorders, especially in the diagnosis of dementia, since the early 90's [1,2]. Nevertheless, in recent years major progress has been made in the spatial resolution of brain scans using high resolution PET camera and fused MR images. These technological advances should improve the semiology of brain abnormalities demonstrated by ¹⁸F-FDG PET. We report here that ¹⁸F-FDG PET could demonstrate brainstem changes in patients with progressive supranuclear palsy (PSP).

Case Report

A 56 year old woman presented with parkinsonism associated with apathy, cognitive impairment, vertical supranuclear gaze palsy, walking difficulties, postural instability with falls and dysarthria. Progressive supranuclear palsy was suspected.

A ¹⁸F-DOPA cerebral PET/CT, performed after injection of 72 MBq of [¹⁸F]-DOPA, showed striatal dopaminergic uptake decrease (predominating in the left side) as previously described [3] (Figure 1) and a MRI demonstrated midbrain atrophy, consistent with PSP diagnosis.

A ¹⁸F-FDG cerebral PET/CT (mCT Flow 20, Siemens Medical System) was performed 30 min after injection of 185 MBq of [¹⁸F]-FDG. High-resolution reconstruction (image size=400 × 400, 3 iterations, 21 subsets, gaussian filtration 2mm) was performed. Visual analysis was performed on PET images coregistered with MR images. FDG scan revealed hypometabolism in the frontal cortex and in the anterior cingulate cortex. Hypometabolism in the left caudate nucleus was also seen (Figure 1). These anomalies have been previously reported in patients with PSP [4-6].

Statistical analysis was made using a direct comparison of the patient's PET scans with a normal database of aged-matched control subjects using Scenium VD20 software (Siemens Medical Solutions). Database comparison detected hypometabolism in the midbrain, particularly the superior colliculi (Figure 2) where neuronal loss, gliosis and neurofibrillary tangles were previously described on microscopic findings in PSP patients [7,8]. The midbrain is implicated in balance and eye movement control [9,10].

Discussion

These new images in a patient with PSP underline the progress made by MRI and PET, which are now able to show very small changes associated with neurodegenerative diseases. Imaging is playing an increasingly important role in the management of dementia patients; the objective is to find specific signs of each disease. This example of hypometabolism in patient with PSP in the superior colliculi, confirmed by the statistical analysis (with a Siemens Medical Solution software available on all recent Siemens PET cameras), will also lay emphasis on the absolute necessity to look at new PET images in a different manner: look for small changes on PET/MR fused images.

Hypometabolism of the superior colliculi, which has not yet been described to our knowledge, is consistent with the first clinico-neuropathological description of the disease by Steele, Richardson and Olszewski published in 1964, but further investigations are required to consolidate this finding in PSP patients. Comparison of superior colliculi's metabolism should be made between patients with PSP and patients with Parkinson disease and other atypical or secondary parkinsonism.

Conclusion

High resolution ¹⁸F-FDG PET allowed detecting hypometabolism on very small midbrain structures damaged in PSP patients.

References

1. Berti V, Pupi A, Mosconi L (2011) PET/CT in diagnosis of dementia. *Ann N Y Acad Sci* 1228: 81-92.
2. Brown R, Bohnen N, Wong K, Minoshima S, Frey K (2014) Brain PET in suspected dementia: Patterns of altered FDG metabolism. *Radiographics* 34:684-701.

*Corresponding author: Alice Jaillard, Service de médecine nucléaire et Imagerie fonctionnelle, Hôpital Roger Salengro- Centre Hospitalier Régional Universitaire de Lille, 2 avenue Oscar Lambret-59037 Lille Cedex, France, Tel: 0033 20445656; E-mail: alice.jaillard@gmail.com

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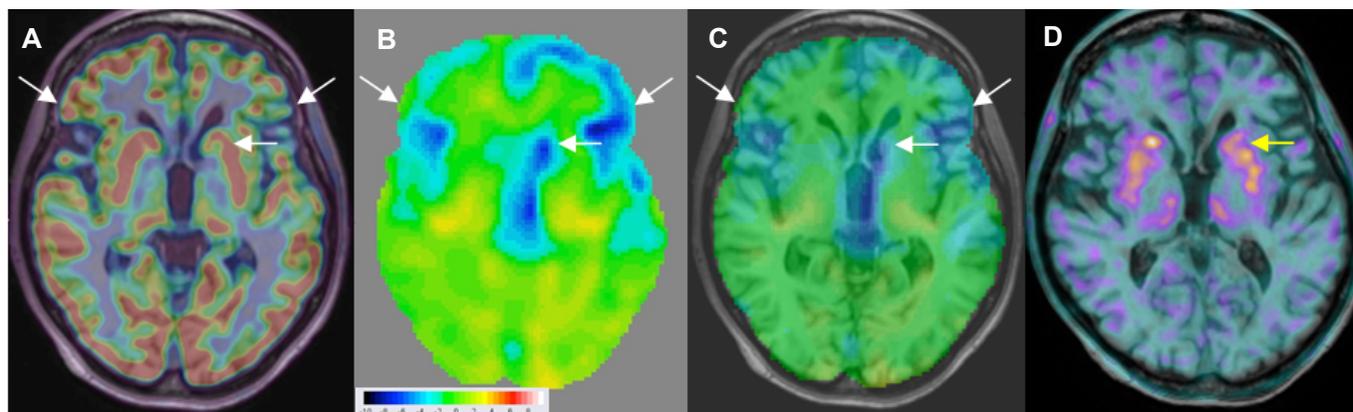


Figure 1: Hypometabolism in the frontal cortex, predominating on the left side and on the left caudate nucleus on ¹⁸F-FDG PET and MR image fusion (white arrow, A) confirmed by normal subjects database comparison (B) and image fusion between database comparison and MRI (C). Bilateral ¹⁸F-DOPA striatal uptake decrease on ¹⁸F-DOPA PET predominating on the left side (yellow arrow, D).

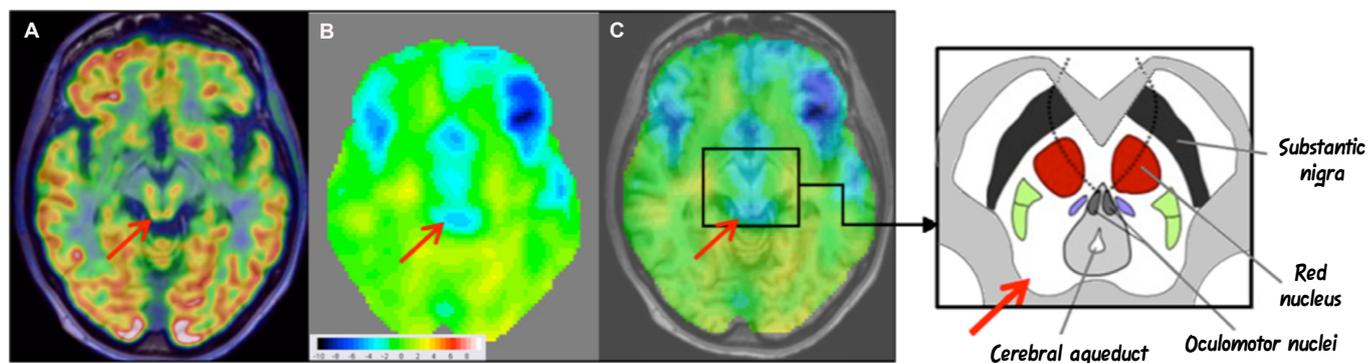


Figure 2: Hypometabolism in the superior colliculus (red arrow) on ¹⁸F-FDG PET and MR image fusion (A) confirmed by normal subject database comparison (B), and image fusion between database comparison and MRI (C). In the black square, schematic anatomical axial view of the location of the superior colliculus (red arrow).

3. Petyt G, Kreisler A, Demailly F, Olivier A, Defebvre L, et al. (2014) Undetectable specific striatal [¹²³I]-Ioflupane SPECT uptake in patients with parkinsonism. *J Neurol Sci* 314: 167-171.
4. Renard D, Collombier L, Castelnovo G, Labauge P (2010) Teaching neuroimages: FDG-PET in progressive supranuclear palsy. *Neurology* 74: e60.
5. Broski SM, Hunt CH, Johnson GB, Morreale RF, Lowe VJ, et al. (2014) Structural and functional imaging in parkinsonian syndromes. *Radiographics* 34: 1273-1292.
6. Teune LK, Bartels AL, de Jong BM, Willemsen AT, Eshuis SA, et al. (2010) Typical cerebral metabolic patterns in neurodegenerative brain diseases. *Mov Disord* 25: 2395-2404.
7. Steele JC, Richardson JC, Olszewski J (1964) Progressive supranuclear palsy. A heterogeneous degeneration involving the brain stem, basal ganglia and cerebellum with vertical gaze and pseudobulbar palsy, nuchal dystonia and dementia. *Arch Neurol* 10: 333-359.
8. Dickson DW, Rademakers R, Hutton ML (2007) Progressive supranuclear palsy: Pathology and genetics. *Brain Pathol* 17: 74-82.
9. Sparks D (2002) The brainstem control of saccadic eye movements. *Nature Reviews Neuroscience* 3: 952-964.
10. Horn A, Fröschl A, Feige J, Röber, H, Kretschmar (2016) Correlation of tau pathology in eye movement related brainstem nuclei in cases of progressive supranuclear palsy (PSP) and a proposed role of perineuronal nets. *Clinical Neurophysiology* 127: e2.