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Florbetapir PET/CT in Parkinson dementia: a preliminary study

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Objectives:

Cognitive impairment is a common nonmotor complication of Parkinson's disease (PD), which increases in severity and frequency over time, with up to 80% of patients developing dementia (PDD). There was a growing evidence of a possible synergistic role of Alzheimer-type brain lesions containing β-plaques (Aβ), with the cortical alfa-syn pathology, in determining PDD. Aim of this preliminary study was to study the prevalence in vivo of amyloidopathy in PDD and to correlate the amyloid pathology with the clinical features of patients.

Materials and Methods:

10 patients with PDD (age 71,8±4,5 years; mean time to onset of dementia 7,0±3,4 years) underwent [18F]Florbetapir PET/CT (a PET ligand that detects fibrillar Aβ). At the time of PET, mean disease duration was 9,2±3,7 years and the mean UPDRS III was 36,2±9,9. All patients performed extensive neuropsychological evaluation. Images were visually and semiquantitatively analized. After spatial transformation of each scan into Montreal Neurological Institute space, cortical-to-cerebellum mean standard uptake values (SUVr) were calculated for each subjects.

Results:

3/10 patients (30%) resulted positive at the visual analysis of [18F]Florbetapir PET scans (Fig. 1). A statistically significant inverse correlation (r=-0,607; p<0.05) was found between global SUVr and the severity of cognitive impairment, as assessed by MMSE score at the time of PET/CT scan (Fig. 2). A significant correlation was furthermore observed between MMSE score and multiple cortical areas, including precuneus (right: r=-0,779;p=0,008; left: r=-0,691; p=0,027) (Fig. 3), parietal (right: r=-0,828;p=0,003; left: r=-0,812;p=0,004) and temporal areas (right: r=-0,781; p=0,008; left: r=-0,702; p=0,024) (Fig. 4). However, no association between the cortical retention of [18F]Florbetapir and time to dementia or disease duration was found.

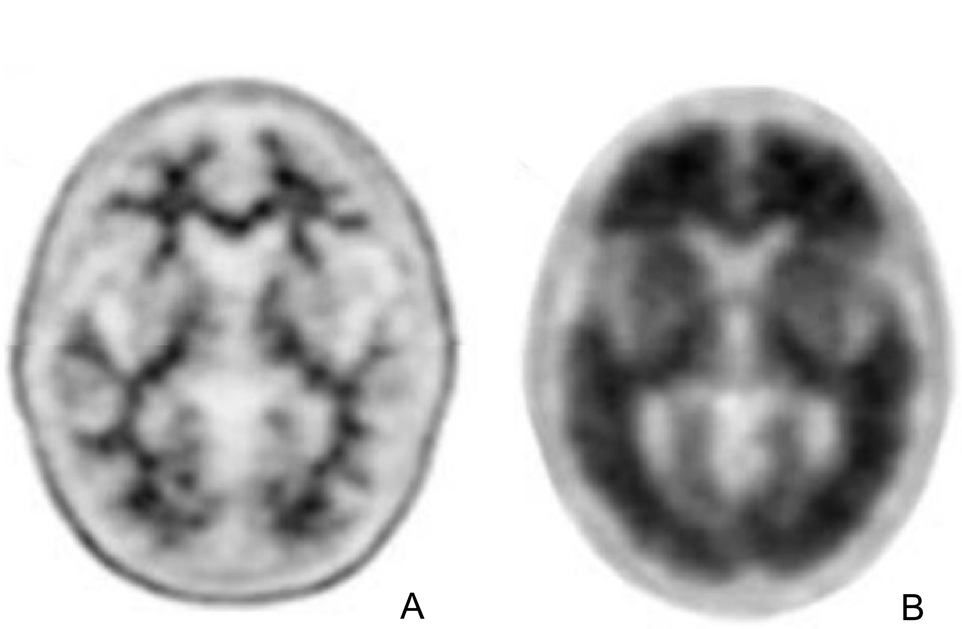
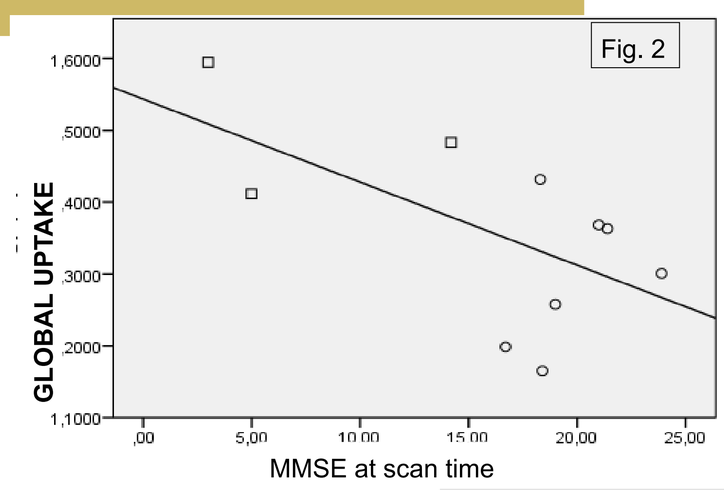
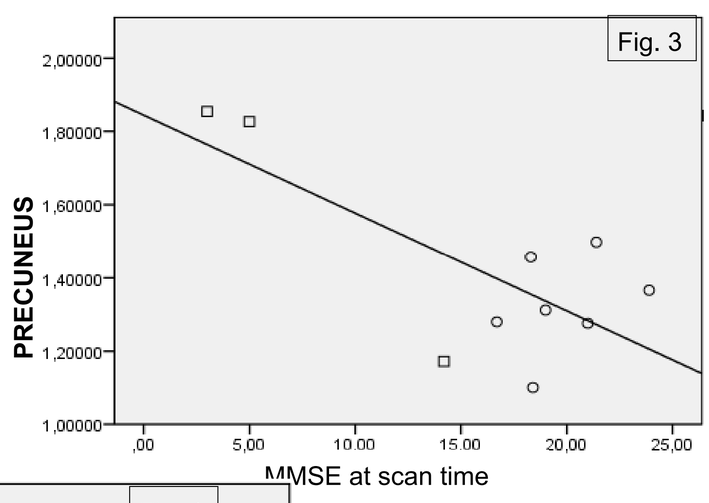
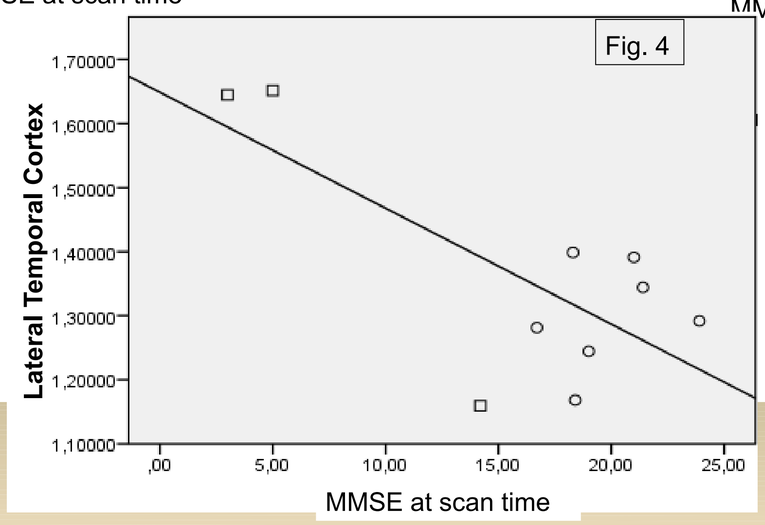


Fig. 1: Example of a negative (A) and a positive (B) Florbetapir PT/CT scan







Discussion:

A few studies are available on the use of PET imaging in assessing amyloid pathology in PD, and so far only one study with florbetapir was conducted. Our result regarding the prevalence in vivo of amyloid pathology in PDD is consistent with previous reports. Although our data are preliminary, the uptake of [18F]Florbetapir PET/CT seems to be related to the severity of cognitive impairment.

Conclusions:

PDD represent a heterogeneous entity, in which the co-existence of cortical alfa-syn and Aβ pathology may act in a synergistic way influencing the severity and/or the phenotype of the cognitive dysfunction; our preliminary study could suggest the significant role of cortical Aβ in increasing the severity of cognitive impairment in PD, quite differently from what reported in Alzheimer Disease. Larger studies are needed to understand the clinical correlates of cortical amyloid in PD dementia.

References:

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