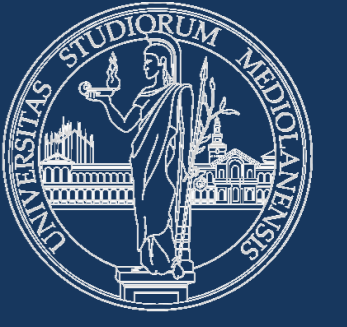


CASES OF POSTERIOR CORTICAL ATROPHY WITH AND WITHOUT AMYLOID PATHOLOGY: IS THERE ANY DIFFERENCE?



UNIVERSITÀ DEGLI STUDI
DI MILANO

A. Arighi¹, P. Basilico¹, G. Marotta², M. Longo², M. Mercurio¹, A. Pietroboni¹, G. Fumagalli¹, L. Ghezzi¹, A. Calvi¹,
M. Scarioni¹, T. Carandini¹, M. De Riz¹, E. Rotondo¹, P. Corti¹, R. Vimercati¹, C. Fenoglio¹, D. Galimberti¹, R. Benti², E. Scarpini¹

Fondazione Ca' Granda IRCCS Ospedale Policlinico, Milan, Italy

1 - Neurodegenerative disease Unit, Department of Pathophysiology and Transplantation, University of Milan

2 - Department of Nuclear Medicine, Fondazione IRCCS Ca' Granda Ospedale Maggiore Policlinico, Milano, Italy

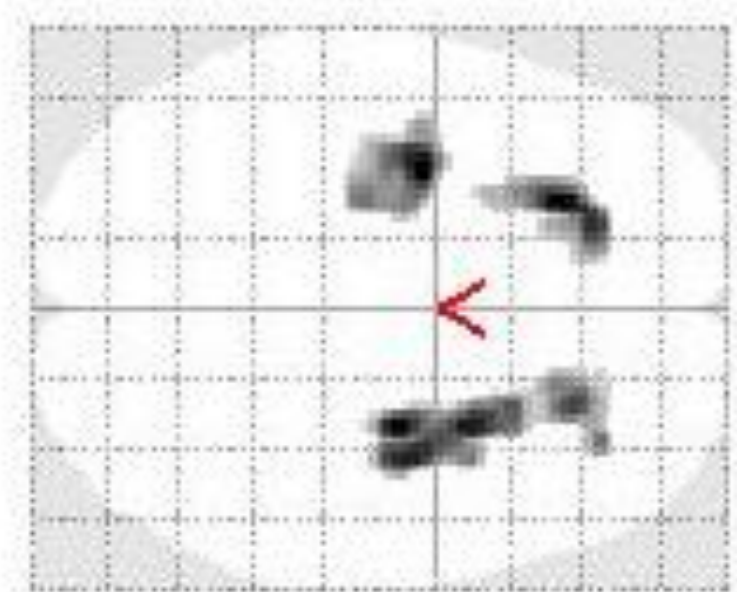
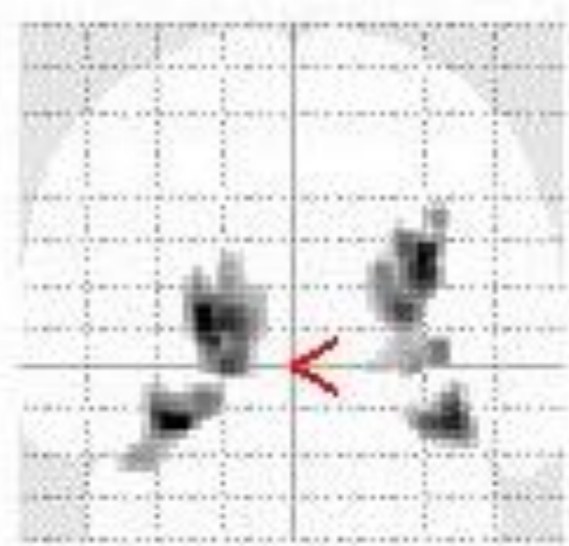
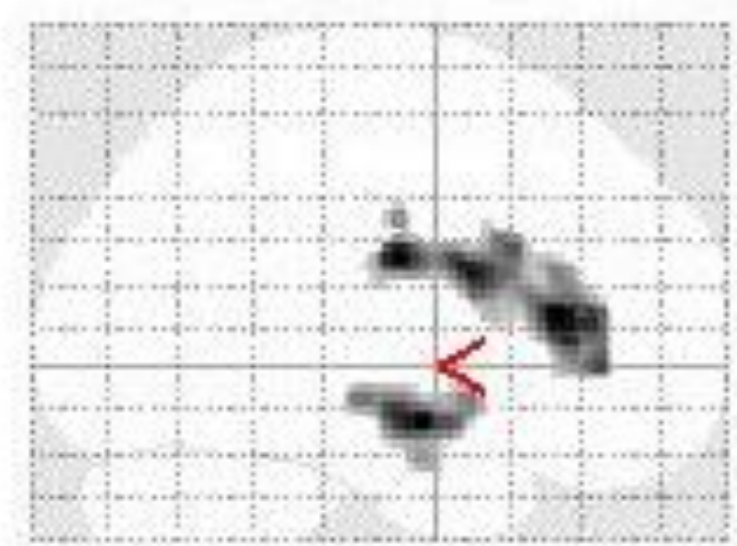
Background

Posterior cortical atrophy (PCA) is a rare variant of Alzheimer's disease characterized by predominant complex visuo-spatial deficits and parieto-occipital or temporo-occipital cortical atrophy¹. As in typical Alzheimer's disease, amyloid cascade with deposition of plaques and tangles seems to play a key role in pathogenesis of PCA. However, a minority of cases without evidence of amyloid deposition may present with similar clinical manifestations².

We describe 6 out of 16 cases of PCA without evidence of amyloid pathology on cerebrospinal fluid (CSF) analysis.

Materials and Methods

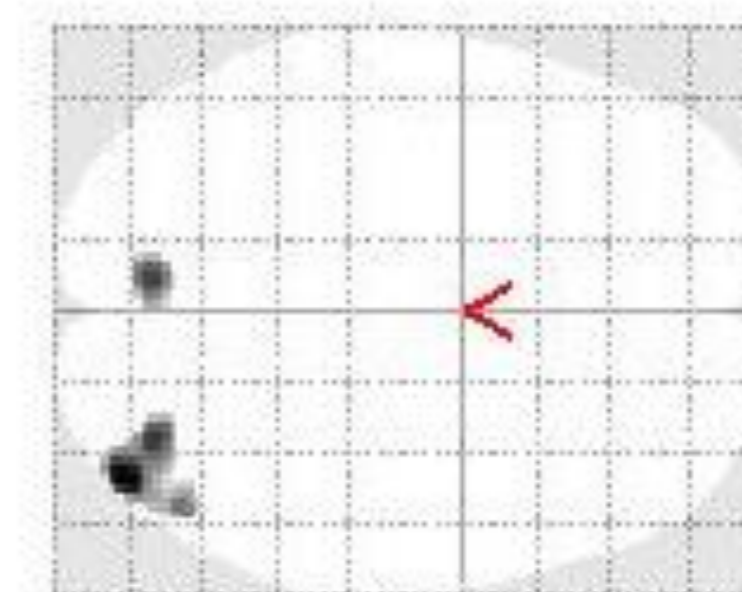
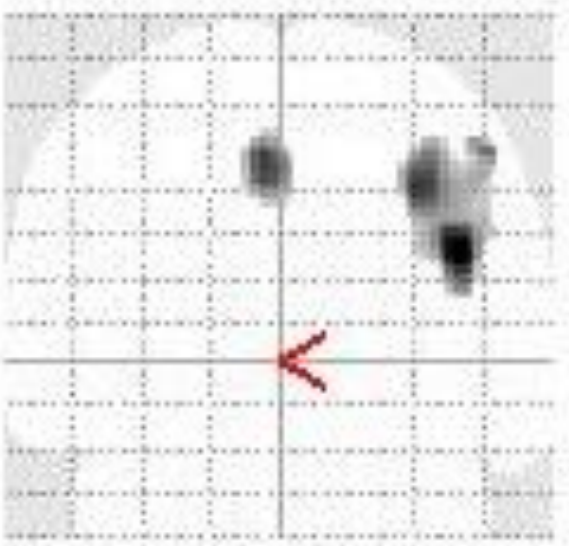
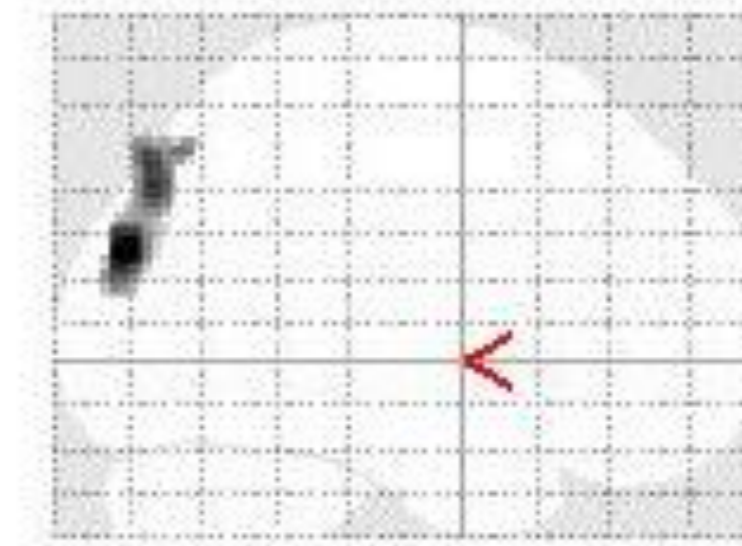
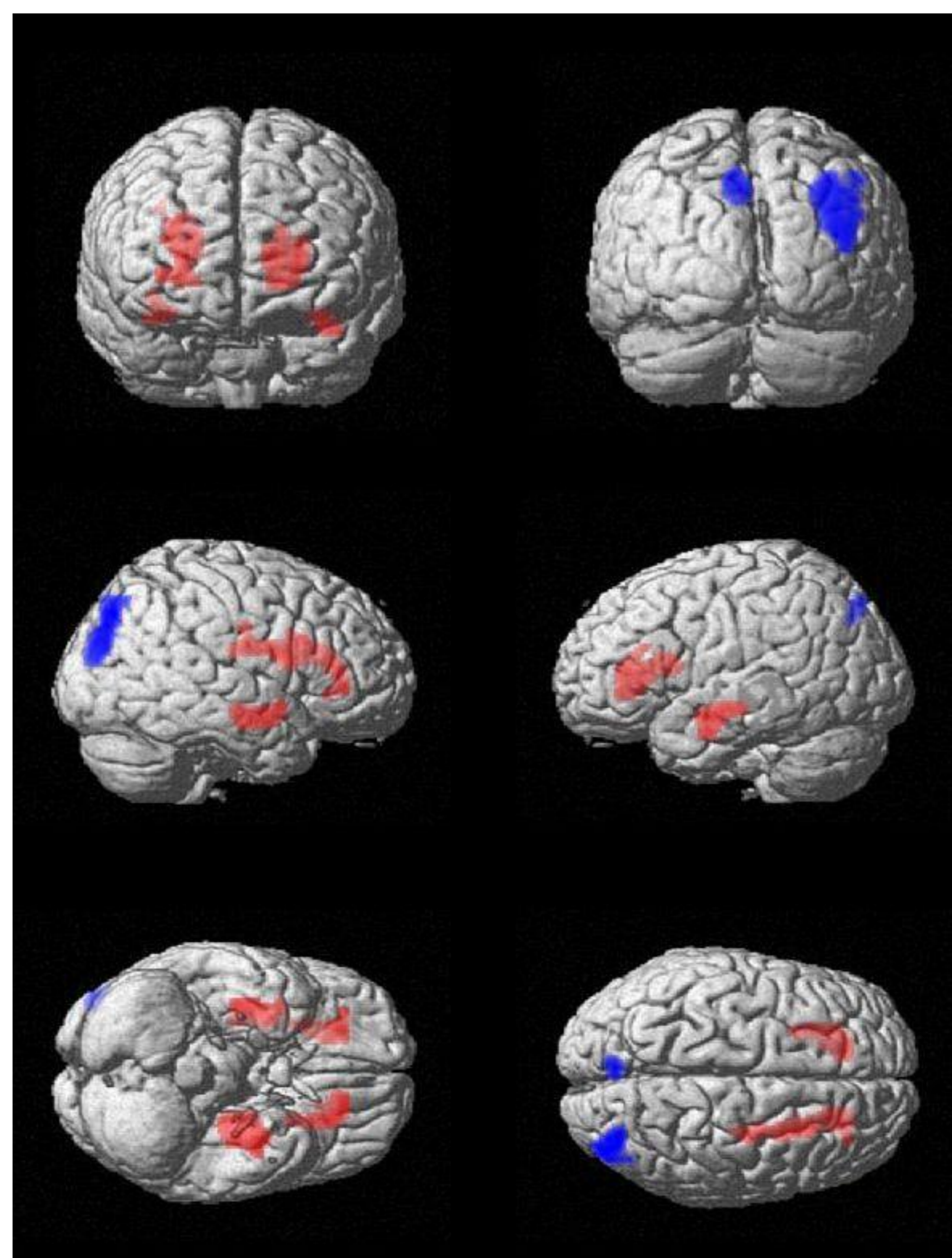
All the patients underwent a neurological examination, neuropsychological evaluation, brain imaging consistent with brain MRI or CT, brain FDG-PET and lumbar puncture. FDG-PET images were analyzed using statistical parametric mapping (SPM8, Wellcome Department of Imaging Neuroscience, London).



SPM{T₁₄}

SPMresults: \StatPCA_LowABvsHighAB
Height threshold T = 3.787390 [p<0.001 (unc.)]
Extent threshold k = 100 voxels

Hypometabolism
High BA vs Low BA



SPM{T₁₄}

SPMresults: \StatPCA_LowABvsHighAB
Height threshold T = 3.787390 [p<0.001 (unc.)]
Extent threshold k = 100 voxels

Hypometabolism
Low BA vs High BA

Results

We identified 16 patients with a clinical diagnosis of PCA, according to 2012 UCL criteria³. CSF analysis showed a pattern of amyloid pathology in 10 cases, while 6 patients had normal A β 42 levels. The two groups were not significantly different from a clinical point of view, but they showed a different pattern on FDG-PET: patients with normal A β 42 levels had a higher temporal and insular hypometabolism bilaterally, while patients with low A β 42 had a higher right posterior parietal and left mesial parietal hypometabolism. Moreover, considering all the patients together, parietal hypometabolism on FDG-PET correlates with low A β 42 CSF levels.

Conclusions

There are cases of PCA without evidence of amyloid pathology and they have a different hypometabolism pattern on brain FDG-PET, involving insular and temporal region. Follow-up will allow us to describe how these forms will evolve. Patients with amyloid pathology have a higher hypometabolism in parietal areas respect patients without amyloid pathology; the right parietal area is the same area in which hypometabolism is correlated with low A β 42 on CSF in all PCA.

References

1. Sebastian J Crutch et al. Posterior cortical atrophy. Lancet Neurol. 2012; 11: 170–78
2. Renner JA, Burns JM, Hou CE, McKeel DW Jr, Storandt M, Morris JC. Progressive posterior cortical dysfunction: a clinicopathologic series. Neurology. 2004; 63(7):1175–1180.
3. Sebastian J. Crutch et al. Shining a light on posterior cortical atrophy. Alzheimer's & Dementia. 2013; 1–3