

Poster presentation

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Progressive supranuclear palsy (Steele - Richardson - Olszewski syndrome) and dementia

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Background

The progressive supranuclear palsy (PSP) constitutes the second most common parkinsonian disorder after the idiopathic form of the disease with an incidence of 5.3 newly diagnosed cases per 100,000 people annually and prevalence 1.39 per 100,000. Clinically, the disease presents itself with not only motor symptoms but also dementia. The aim of this study is to review and present all recent data, specifically those related to the neuropathology and biochemistry, of dementia in PSP.

Materials and methods

Recent advances in molecular and genetic research of PSP are being reviewed. MRI and PET findings are analytically described, while the utility of other exams, like EEG, in the differential diagnosis between PSP and other dementias evaluated.

Results

As depicted from the previous mentioned imaging methods and research, the reported significant learning deficits in PSP are associated with disease-related lesions located not only at subcortical (globus pallidus, mesencephalon, corpus striatum), but also at cortical regions (prefrontal and premotor cortex). A wide spectrum of symptoms is correlated to the differential development of these lesions from the neurofibrillary tangles within the cortex.

Conclusions

The progression of the condition to dementia is a characteristic element of PSP, but does not occur in the same manner in all patients. The type of the neurofibrillary tan-

gles and the range of the regions that they affect, differentiate this condition from other neurodegenerative disorders. Further research is required in relation to the understanding of the neurotransmitter systems involved in the memory and cognitive impairment of PSP, which will be the cornerstone for the discovery and implementation of novel supportive care regimens.

References

1. Dubois B., Slachvesky A., Pillon B., Beato R., Villalpona J.M., Litvan I.: **"Applause sign" helps to discriminate PSP from FTD and PD.** *Neurology* 2005, **64**:2132-2133.
2. Millar D., Griffiths P., Zermansky A.J., Burn D.J.: **Characterizing Behavioral and Cognitive Dysexecutive Changes in Progressive Supranuclear Palsy.** *Movement Disorders* 2006, **21**:199-207.
3. Mott R.T., Dickson D.W., Trojanowski J.Q., Zhukareva V., Lee V.M., Forman M., Van Deerlin V., Ervin J.F., Wang Deng-Shun B.A., Schmechel D.E., Hulette C.M.: **Neuropathologic, Biochemical, and Molecular Characterization of the Frontotemporal Dementias.** *J Neuropathol Exp Neurol* 2005, **64**:420-428.
4. Cotelli M., Borroni B., Manenti R., Alberici A., Calabria M., Agosti C., Ginex V., Ortelli P., Binetti G., Zanetti O., et al.: **Action and Object Naming in Frontotemporal Dementia, Progressive Supranuclear Palsy, and Corticobasal Degeneration.** *Neuropsychology* 2006, **20**(5):558-565.