

P12.07

Neurochemical markers in the cerebrospinal fluid in frontotemporal dementia. Relation to other dementia disorders

M. Sjögren*. *Institute of Clinical Neuroscience, Göteborg University, Mölndal, Sweden*

Background: The pathophysiology of frontotemporal dementia (FTD) is currently unknown. However, several findings point at the cytoskeleton as the vulnerable spot of degenerative changes in this disorder. In hereditary FTD, tau gene mutations have been found. Some studies, but not all, have suggested that the cerebrospinal fluid level of tau is increased in FTD and yet others suggest that another cytoskeleton protein, the neurofilament light protein (NFL), is increased in this disorder. Studies on brain tissue have suggested that the tau pathology in AD differs from that in FTD and that the difference may be related to the degree of phosphorylation.

Objective: To the involvement of total CSF-tau (here referred to as tau) and phosphorylated CSF-tau (phosphotau) in FTD, Alzheimer's disease and various types of dementia.

Methods: Using ELISAs for total tau and tau phosphorylated at Thr181 (phosphotau), the CSF levels of total tau and phosphotau were determined in patients with probable and possible Alzheimer's disease (AD; n=41 and 19, respectively), frontotemporal dementia (FTD; n=18), and Parkinson's disease (PD; n=15) and in age-matched controls (n=17).

Results: Both CSF-tau and CSF-phosphotau were increased in probable AD compared with FTD ($p<0.001$), PD ($p<0.001$) and controls ($p<0.001$). CSF-phosphotau was increased in possible AD compared with FTD ($p<0.001$). CSF-tau and CSF-phosphotau were positively correlated in all the groups.

Conclusion: The results suggest that the CSF levels of tau and phosphotau are increased in about two-thirds of probable AD and in half of possible AD but are normal in FTD, and PD compared with normal aging. Results from previous investigations on CSF biomarkers such as β -amyloid42, GAP-43, and APP in FTD and AD will also be presented.

P12.08

Blood pressure dysregulation and Alzheimer's disease: a case-control study

T. Sobow*, I. Kloszewska. *Department of Psychiatry, Medical University of Lodz, Poland*

Objectives: It has been hypothesized that dysregulation of blood pressure might be involved in both initiation and progression of the degenerating process in Alzheimer's disease (AD). A potential relation between blood pressure (mid-life and current), orthostatic phenomenon and the presence of symptoms of AD has been explored.

Method: Forty-five AD patients were compared with 39 age and gender matched healthy controls. A detailed history of arterial hypertension, orthostatic hypotension and cardiac disease was taken. Additionally, an orthostatic test and serial blood pressure measures have been performed.

Results: Mid-life "jumping" blood pressure pattern (proportions 0.22 vs 0.05, Fischer's exact test, $p=0.03$; OR=5.1, 95%CI 2.3 to 8.9), lower systolic blood pressure ($p=0.06$) and positive orthostatic reaction were noted more commonly in cases than in controls (proportions 0.36 vs 0.15, Fischer's exact test, $p=0.03$; OR=2.9, 95%CI 2.0 to 5.4).

Conclusions: Dysregulation of blood pressure control and resulting brain metabolic inadequacy might possibly be related to AD. Hypertension, as well as other vascular risk factors, should be considered in planning preventive strategies for AD.

P13. Dementia – other**P13.01**

Which tests are renounceable in the diagnosis of dementia without relevant losses in discrimination?

M. Damian¹*, M. Kreis¹, B. Krumm³, M. Syren², S. Speck², F. Hentschel¹. ¹Neuroradiology, ²Memory Clinic and ³Biostatistics, CIMH, Fac. Clin. Med. Mannheim, University of Heidelberg, Germany

Objective: Identifying the most accurate neuropsychological tests in the (differential) diagnostics of dementia to reduce time of administration.

Methods: 127 patients of a memory clinic were examined clinically, neuropsychologically and neuroradiologically. The neuropsychological test battery comprised the CERAD, the MMSE, the Clock Drawing- and the speed tests: Trail-making-, Maze-, Color-Word- and Digit-Symbol-Test. Their administration takes about 2 hours per patient. Stepwise discriminant analyses examined which of the variables could be discarded without relevant losses in the diagnostic validity of the test battery.

Results: The CERAD and the clock-drawing-test proved to be sufficient to discriminate between dementia and no dementia. Their administration takes only 30 minutes. Due to the shortening of the test battery, sensitivity sinks from 95% to 93.9%, specificity from 97.9% to 96.4%. These losses are acceptable. Important timesaving potentials were also found with regard to the differential diagnosis.

Conclusion: Depending on which types of dementia are to be differentiated, the test battery can be abridged of definite measures to reduce the administration costs without unacceptably reducing the diagnostic accuracy.

P13.02

Neuroimaging and frontal-subcortical dysfunction in a stroke cohort

J. Looi¹*, P. Sachdev², M. Valenzuela², H. Brodaty³, W. Wen², L. Lorentz³, J. Sims², J. Kinch², D. Gillies³, R. Schnier. ¹Department of Aged Care, St. George Hospital, Sydney; ²University of New South Wales; ³Prince of Wales Hospital, Australia

Aims: To assess structural-functional correlations between MRI and neuropsychological function in a large Australian cohort of stroke patients.

Method: 200 stroke patients recruited from the stroke units of 3 major Sydney teaching hospitals and 100 healthy community dwelling elderly controls underwent detailed neuropsychological testing and MRI neuroimaging 3 months after their stroke. The MRI scan data were reconstructed via computer & analysed using standardised rating methods for atrophy, subcortical hyperintensities (caps, rims etc.).

Results: Preliminary data analysis demonstrates a high prevalence of frontal-subcortical neuropsychological dysfunction in the stroke patients which may correlate with load of subcortical hyperintensities in frontal, periventricular regions of the brain and frontal atrophy.

Conclusions: Association of frontal and subcortical neuroimaging findings with frontal-subcortical dysfunction in stroke patients suggests a possible substrate for the cognitive deficits seen post-stroke.