Combined analysis of CSF markers for axonal damage in MS *Teunissen C*, Vanderstichele H\*, VanMechelen E\*, Dijkstra C, Polman C\*\*, Jakobs C\*\*\* NUBIN, Department of Molecular Cell Biology and Immunology, VUmc, Amsterdam, \* Innogenetics, Zwijnaaarde, Belgium, \*\* Department of Neurology, VUmc, Amsterdam, \*\*\*Department of Clinical Chemistry, VUmc, Amsterdam

Axonal loss in MS has now been recognized as an important feature of MS. Axonal loss correlates with disability and likely is the correlate of the transition from the RR to SP phase in MS. Validated biomarkers for axonal loss may help monitoring or predicting disease outcome.

The aim of the present study was to analyse whether possible CSF markers for axonal loss are different between the different clinical subtypes of MS.

Methods: We determined the CSF concentrations of N-acetylaspartate (N-AA), amyloid beta, tau, hyperphosphorylated tau and growth associated protein 43 (GAP-43). Included were 27 relapsing remitting (RR) MS patients, 14 secondary progressive (SP) patients, 8 primary progressive (PP) patients and 12 neurological controls. Mean age was 44.3 year (27-60) and comparable among the groups. Mean EDSS was 4.1 (1.5-7.5) and was higher in SP patients compared to RR MS patients.

Results: There was tendency towards higher values for N-AA in MS patients. The average value of N-AA was increased in the RR compared to the SP MS patients. The N-AA concentration correlated with hyperphosphorylated tau and GAP-43. N-AA and GAP-43 concentrations correlated with EDSS scores.

Conclusions: GAP-43, amyloid beta, tau and hyperphospharylated tau concentration measurements seem not to be useful for discrimination between different clinical subtypes of MS and neurological controls. The correlation of N-AA and GAP-43 with EDSS and with each other may suggest that these markers are related to a common mechanism during progression of the disease.

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