CSF ANALYSIS OF PROGRESSIVE SUPRANUCLEAR PALSY: NO INDICATION FOR THE PRESENCE OF SPECIFIC TAU FORMS

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Introduction: The differential diagnosis of progressive supranuclear palsy (PSP) is complicated since patients with PSP are often difficult to distinguish from those with other parkinsonian syndromes. A main neuropathological hallmark of PSP is the presence of tau aggregates in the brain. Besides aggregation, differential processing of tau has been identified in PSP brains. Recently, it has been proposed that the ratio of 33/55 kDa tau forms in cerebrospinal fluid (CSF) is specifically reduced in PSP CSF\(^1\), a finding that was reproduced by the same authors in a larger cohort of patients\(^2\).

Aims: To reproduce these results, and to evaluate the use of the ratio of 33/55 kDa tau forms in CSF as a promising and valuable biomarker for PSP.

Methods: We used immunoprecipitation to isolate the tau protein from CSF, followed by western blotting to identify and quantify the precipitated tau proteins, as previously described\(^1,2\).

Results: We were not able to detect the 33/55 kDa tau forms in CSF. We demonstrated that i) CSF total tau levels are too low to be detected by the published protocol, and ii) the described 33 and 55 kDa bands are likely the heavy and light chains of IgG used in the assay.

Conclusions: We were not able to confirm the presence of 33/55 kDa tau forms in CSF. Our data refute the suggestion that these proposed tau isoforms are a reliable biomarker for PSP.

\(^1\)Borroni et al., Neurobiol Aging, 2009;30:34-40

\(^2\)Borroni et al., Neurology, 2008;71:1796-1803