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Investigation of neuroinflammatory markers in Frontotemporal Lobar Degeneration (FTLD)

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State of the art: Neuroinflammation is considered an important pathophysiological process in FTLD. YKL40 is an activated astrocytic marker shown to be upregulated in some FTLD subtypes, but its variability across different proteinopathies and relationship to neurodegeneration remain unclear.
Methodology: YKL40 was measured in CSF of 27 presumed/pathology proven FTLD-Tau [24 progressive supranuclear palsy (PSP), 2 corticobasal degeneration, 1 *MAPT* mutation)], and 14 presumed/pathology proven FTLD-TDP [6 semantic and 1 nonfluent variant primary progressive aphasia (svPPA, nfvPPA), 6 FTD with motor neuron disease (FTD-MND), 1 behavioural variant FTD (bvFTD)]. Multiple linear regression analyses were performed for comparison between these groups. As well, YKL40 levels were compared between AD biomarker-positive [11 corticobasal syndrome (CBS), 6 logopenic variant PPA, 3 PSP, 2 svPPA/nfvPPA] and AD biomarker-negative [8 CBS, 8 PSP, 6 bvFTD, 1 FTD-MND, 3 svPPA/nfvPPA] groups. We also examined the association of YKL40 and neurofilament light chain (NfL) (for neurodegeneration).
Results: After adjusting for age and gender, higher YKL40 levels were found in FTLD-TDP compared to FTLD-Tau (mean=442 vs 379ng/mL, p=0.08), and in AD biomarker-positive compared to AD-negative subjects (mean=380 vs. 293, $p<0.05$). We found a significant interaction effect of dichotomized NfL levels and AD status ($p<0.05$) on YKL40. Only in the AD biomarker-positive group, the high NfL group also had significantly higher YKL40 levels than the low NfL group.
Conclusion: CSF YKL40 levels are increased in FTLD-TDP. Our results also suggest that YKL40 levels may reflect a glial response to AD-specific neurodegenerative processes. Study with other markers is ongoing.

Conflicts of interest

N/A