Intrathecal levels of IL-6 in Alzheimer's disease

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In a recent study, Galimberti et al. [2] reported a significant increase of IL-11 disease (AD $n = 43$) and frontotemporal lobar degeneration (FTLD $n = 24$) in patients with Alzheimer's disease in comparison to age-matched controls ($n = 30$). At the same time, IL-6 showed only a non-significant increase in demented subjects. Measurement of CSF IL-6 levels in AD have revealed contradictory results—unchanged [3, 5] and increased [1, 8, 9]. In our personal studies in a cohort of 169 aged subjects, we found probable AD ($n = 27$), non-AD dementia ($n = 24$, including FTLD $n = 10$ and vascular dementia $n = 5$), and other CNS disorders without cognitive impairment (infections, vascular, neurodegenerative diseases $n = 69$). A specific immunoabsorbent assay (ELISA, R&D Systems, Minneapolis, MN, USA) revealed a significant increase of IL-6 immunoreactivity in CSF of both AD and non-AD dementias compared to controls ($P < 0.03$), despite broad overlap. AD patients with late onset (>65 years) had significantly higher values than those with early onset ($P < 0.05$), but, as in Galimberti's cohort, there were no correlations of CSF IL-6 with age, MMSE scores, disease duration, or CSF A β -42 and T-tau [4, 6]. These data and increased CSL IL-6 levels in hypertensive encephalopathy [7] suggest that it may reflect IL-mediated immunologic phenomena in the course of neurodegenerative processes.

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