Neuroinflammation in the Choroid Plexus During Alzheimer's Disease

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Alzheimer’s disease (AD) is the most common neurodegenerative disorder contributing to 60–80 % of cases of dementia worldwide, with no effective treatment so far. Alongside the amyloid-beta (Ab) aggregation and neurofibrillary tangles, the presence of neuroinflammation is believed to be the core mechanism in AD pathophysiology. Inflammatory responses and functional abnormalities have been reported in the choroid plexus (CP), crucial for Ab clearance and brain homeostasis maintenance. This study aims to reveal the relationship between CP and neuroinflammation during AD. To determine the Ab aggregation in CP and to develop the AD in vitro model, murine epithelial cells of CP (Z310) were exposed to human Ab1-42 in a concentration and time-dependent manner. Subsequently, the model’s inflammatory profile was time-dependently studied, specifically the cytokines IL-1b and IL-10, and immunoreceptors TLR9 and FPR2 expression. Results were obtained by using immunocytochemistry and western blot. Ab aggregation occurred in Z310 cells in all experimental conditions. After 72 hours, pro-inflammatory IL-1b expression predominated in the model, but after 5-day incubation, the anti-inflammatory IL-10 levels increased significantly. Yet, IL-10 overexpression was consistently lower than IL-1b expression. Interestingly, resulting from Ab aggregation, FPR2 was significantly upregulated but not TLR9. This data suggests novel insight into the molecular mechanism of neuroinflammation initiated by CP during AD, suggesting the activation of FPR2 followed by cytokine release through an undefined signaling cascade. This study supports the CP’s anti-inflammatory nature during pathological conditions. Future research into CP and its strategic properties appears to be a promising therapeutic target.

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