007 THE ROLE OF CHRONIC INFLAMMATION ON AMYLOID BURDEN IN MS

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Alzheimer's disease (AD) is a degenerative disorder wherein β-amyloid deposition and inflammation are key pathological features. The complex interplay between inflammation and β-amyloid burden in AD is becoming increasingly recognised. To evaluate the influence of chronic inflammation on β-amyloid deposition, a cohort of pathologically confirmed multiple sclerosis (MS) cases (n=67) was compared to non-demented age- and sex-matched controls (n=55). Formalin-fixed paraffin embedded cortical tissue from the mesial temporal gyrus was immunostained for β-amyloid and myelin. Quantitative measures of β-amyloid burden in MS normal appearing grey matter (NAGM) and cortical lesions were compared to those derived from control NAGM. MS cases lying at the extremes of β-amyloid burden (n=25) were additionally immunostained for microglial inflammation. Preliminary data showed that β-amyloid burden was significantly reduced in MS NAGM compared to controls ($p=0.08$), with MS cortical lesions demonstrating significantly less β-amyloid compared to MS NAGM ($p<0.01$), particularly at the lesional border. Microglial activation was significantly less in the high compared to low amyloid groups ($p<0.05$). These findings suggest the chronic inflammatory milieu in the MS brain protects against β-amyloid deposition, with microglial activation playing a central role. Studies evaluating the relationship between MS-specific microglial activation and amyloid processing are warranted.

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