

Tau, microglia and plaque-associated proteins in dyschoric angiopathy

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Introduction. Dyschoric angiopathy is an infrequent finding in elderly patients with congophilic vessels when amyloid (Abeta) is deposited outside the blood brain barrier in the parenchyma around small vessels. In the present study we compare dyschoric vessels, "normal" congophilic vessels and amyloid plaques for the presence of microglia, tau and plaque-associated acute phase proteins such as complement and alpha1-antichymotrypsin. Inflammatory changes with microglia and these proteins in vessels could lead to haemorrhage.

Material and Methods. Nine patients with dyschoric angiopathy were studied. Next to paraffin sections, fresh frozen tissue was used in 4 cases. Using immunohistochemistry we investigated the presence of microglia, tau, complement C3d and alpha1-antichymotrypsin. Nearby sections were stained for Abeta₁₋₂₈. Paraffin sections were used after antigen retrieval (microglia markers and complement) or formic acid (Abeta and Tau). Sections were counterstained for Congo red. Alpha1-antichymotrypsin was used on frozen sections only.

Results and Discussion. Congophilic vessels without dyschoric amyloid did not show inflammatory changes. Dyschoric vessels showed clustering of microglia and signs of neurodegeneration around the amyloid deposits. These vessels were also strongly stained for acute phase proteins ACT and complement whereas the microglial cells bear receptors for complement. This suggests an inflammatory process identical to what is described in AD plaques. These changes could make the dyschoric vessels more prone to haemorrhaging.

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