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Cerebral venules are major factors in formation of brain microinfarcts at systemic inflammation: implications for dementia

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Statement of the Problem: Despite the widely accepted facts regarding the role of neuroinflammation as well as vascular pathology in the pathogenesis and progression of neurodegenerative diseases no special significance was given to the pathology of different cerebral microvessels. The purpose of this study is to describe the structural alterations in neuro-glio-vascular elements of brain cortex at early stages of acute inflammation. Methodology: Systemic inflammation was induced by intravenous injection of purified lypopolysacharide (LPS) from E. coli [Serotip 0111:B4] at a dose of 1,0 mg/kg dissolved in saline. Semi thin and ultrathin tissue sections were examined by means of light and electron microscopy. Findings: Investigation of brain capillaries revealed insignificant changes in the integrity of their wall. Despite of edematous perivascular astrocytic endfeet (AEF) the architecture of surrounding brain parenchyma was structurally intact. However, the destructive changes in the wall of cortical venules and their obturation with blood cells were found as indicatives of early onset of neuroinflammation. Pathological changes were especially intense in the postcapillary venules draining blood from brain capillary bed and permeability of which is greater than that of other vessels. In fact the disruption of intercellular tight junctions, significant increase in the amount of pinocytotic vesicles in endotheliocytes, the appearance of transendothelial channels and thickening of perivascular basal membranes were indicators of blood-brain barrier disruption in those vessels. The discontinuous adluminal plasma membrane of endothelial cells and amorphous material in the lumen of venules were observed. Though AEF around venules were not swollen the integrity of their membranes was disturbed. Moreover, electron microscopy revealed the apparent indicators of parenchymal damage around venules: degenerative changes in nerve and glial cells, signs of vasogenic edema among neuropil elements etc. Conclusion: the pathology of cerebral venules should be considered as primary causes for brain microinfarcts with subsequent neurodegeneration at inflammatory conditions.

Recent Publications

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