Retinal and Optic Nerve Damage is Associated with Early Glial Responses in an Experimental Autoimmune Glaucoma Model

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It is well established that the immunization with ocular antigens causes a retinal ganglion cell (RGC) decline, which is accompanied by glia alterations. In this study, the degenerative effects of the immunization with an optic nerve homogenate (ONA) and its purified compound S100 were analyzed on retinas and optic nerves.

Since a participation of glia cells in cell death mechanisms is currently discussed, rats were immunized with S100 or ONA. At 14 and 28 days, immune-histological and Western blot analyses were performed to investigate the optic nerve structure (SMI-32) , retinal ganglion cells (Brn-3a) , apoptosis (cleaved caspase 3, FasL) , and glial profile (Iba1, ED1, GFAP, vimentin) . Neurofilament dissolution in S100 animals was evident at 14 days ($p = 0.047$) and increased at 28 days ($p = 0.01$) . ONA optic nerves remained intact at early stages and degenerated later on ($p = 0.002$) .

In both groups, RGC loss was detected via immune-histology and Western blot at 28 days (ONA: $p = 0.02$; S100: $p = 0.005$) . Additionally, more Iba1+ retinal microglia could be detected at early stages (ONA: $p = 0.006$; S100: $p = 0.028$) . A slight astrocyte response was detected on Western blots only on ONA retinas ($p = 0.01$) . Hence, the RGC and optic nerve decline was partly antigen dependent, while neuronal loss is paralleled by an early microglial response.

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