Immune response against ocular tissues after immunization with optic nerve antigens in a model of autoimmuneglaucoma

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PURPOSE: In recent years, numerous studies have investigated the involvement of immunological mechanisms in glaucoma. Until now, it has not been determined whether the altered antibody pattern detected in patients is harmful to retinal ganglion cells (RGCs) or triggers disease formation in any way. In a model of experimental autoimmune glaucoma, RGC loss can be induced through immunization with certain ocular antigens. In the current study, the time course of the levels of autoreactivity against ocular tissues after immunization was examined.

METHODS: Intraocular pressure was measured regularly. Ten weeks after immunization with an optic nerve homogenate antigen (ONA) , the number of RGCs was determined. Immunoglobulin G levels in aqueous humor were measured via enzyme-linked immunosorbent assay at the same time point. Serum from different time points was used to analyze the possible occurrence of autoreactive antibodies against the retina or optic nerve in this autoimmune glaucoma model. Additionally, optic nerve and brain sections were evaluated for possible pathological findings.

RESULTS: Intraocular pressure stayed within the normal range throughout this study. A continuous increase of autoreactive antibodies against the optic nerve and retina sections was observed. At 4, 6, and 10 weeks, antibody reactivity was significantly higher in ONA animals (pCONCLUSIONS: Our findings suggest that these modified antibodies play a substantial role in mechanisms leading to RGC death. The slow dissolution of RGCs observed in animals with autoimmune glaucoma is comparable to the slow progressive RGC loss in glaucoma patients, thus making this a useful model to develop neuroprotective therapies in the future.

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