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Alkali burn to the eye: Protection using TNF-a inhibition

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Purpose: The aim of this study was to evaluate early retinal damage after induction of ocular surface alkali burns and the protective effects of tumor necrosis factor alpha (TNF-a) blockade.

Methods: Alkali injury was induced in mouse corneas by using 1 N NaOH. Retinal damage was assessed using a terminal deoxynucleotidyl transferase 29-deoxyuridine 5-triphosphate nick end labeling (TUNEL) assay, 15 minutes to 14 days postburn. Immune cell infiltration was assessed by CD45 immunolocalization. Retinal cytokines were quantified using the enzyme-linked immunosorbent assay for interleukin (IL) 1b, IL2, IL6, TNF-a, CCL5, and macrophage inflammatory protein-1a. Protection against retinal damage was attempted with a single dose of either anti–TNF-a antibody (infliximab, 6.25 mg/kg) or control immunoglobulin G (IgG), administered intraperitoneally 15 minutes after the burn was inflicted. Corneal injury was evaluated by using TUNEL and CD45 immunolocalization and by quantifying corneal neovascularization.

Results: There was significant damage to the retina within 24 hours of the corneal burn being inflicted. TUNEL+ labeling was present in 80% of the retinal ganglion cells, including a few CD45+ cells. There was a 10-fold increase in the retinal inflammatory cytokines in the study groups compared with that in controls. A single intra- peritoneal dose of anti–TNF-a antibody, administered 15 minutes after the burn was inflicted, markedly reduced retinal TUNEL+, CD45+ labeling, and inflammatory cytokine expression, compared with that in the controls. Additionally, TNF-a blockade caused a marked reduction in corneal neovascularization, and in cornea TUNEL and CD45 labeling, 5 days after the burn was inflicted.

Conclusions: This study shows that alkali corneal burns can induce significant retinal damage within 24 hours. A single dose of anti–TNF-a antibody, administered 15 minutes after inflicting the burn, provides significant retinal and corneal protection. This could lead to the discovery of novel therapies for patients with alkali injuries.

Biography

Eleftherios I. Paschalis completed his doctorate in Medicine, Ophthalmology, with Excellence from Democritus University of Thrace, Greece in 2010. He began to work full-time as a senior research fellow at the Massachusetts Eye and Ear Infirmary (MEEI) under the guidance of Dohlman in 2011, and since then has become an integral and prolific member of the lab and the Boston Keratoprosthesis group. Since His arrival, Paschalis' Work has led to the filing of two patent applications, several published manuscripts in prominent peer-reviewed journals, and multiple presentations in national as well as international conferences. Paschali's research at MEEI Extends to various areas of ophthalmology. His work involves improvements in Boston Keratoprosthesis, Development of novel implantable glaucoma valves and materials for vitreoretinal surgery. His recent research focus involves the mitigation of the ocular injuries following chemical burns. In 2013 Paschalis was promoted to Instructor at Harvard Medical School and Investigator at MEEI. In This role he continues to conduct research

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