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The morphological changes in the rabbit cornea in experimental corneal wounds healing with monitoring of the corneal electrolyte contents with an energy dispersive X-ray analyzer (EDAX)

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Background: The corneal epithelium serves as a barrier and contributes to the maintenance of corneal transparency and rigidity. Ulcers and erosions of the corneal epithelium as well as delays in resurfacing of the cornea after wounding are major causes of ocular morbidity and visual loss. Corneal ulceration is very frequent and represents one of the most important eye diseases. Chemical burns of eye presents a major therapeutic challenge to the ophthalmologist. Corneal alkali burns represent between 7% and 10% of eye injuries.

Aim: To investigate the structure and organization of corneal epithelium during the healing process of rabbit corneal wounds with monitoring of the corneal electrolyte contents with energy dispersive X-ray analyzer (EDAX) and to screen through immunohistochemistry corneal cellular changes for apoptosis and proliferation during healing process using Proliferating cell nuclear antigen (PCNA) and Tunnel Apoptosis Assay.

Results: Inflammatory response with massive inflammatory cell infiltrates mainly leukocytes (PML) are the first cells that migrate into the tissues in response to injury insult in the first 24 hours cells were mainly neutrophils. Steadily other mononuclear cells begin to appear along with signs of new capillaries formation. Moreover, progressive increase in basal corneal epithelial cell proliferative activity both in limbal region and leading corneal ulcer edge, stromal keratocytes and endothelial capillaries cells. Labeling for apoptosis was found only on the epithelial surface of normal cornea and not in deeper layers. After alkali injury few positive deeper epithelial cell were seen. In addition, stromal cells apoptosis was present in all the alkali-burned corneas. Observable alterations of mineral concentrations in the rabbit cornea after alkali injury burns.

Conclusion: Basal corneal epithelial cell proliferative activity starts earlier after 24 hour of alkali injury and was headed by massive leukocytes infiltrate in the stroma and within epithelial cells. PCNA was expressed in the vascular endothelium. The PCNA expression starts later in the keratocytes but lasts somewhat longer. In present investigations, we found alterations of mineral concentrations in the rabbit cornea both in epithelial and collagen contents after alkali injury burns. Mo is an essential trace element was found to decline during the healing process of corneal wound injury. Medical intervention with appropriate solutions containing element such as Mo may require with other mineral deficient in restoring and maintaining the normal mineral composition of the denuded corneal stroma.

Biography

Hamdy M. Aly, MBBCh, MSc MD .Ph.D. Ain Shams medical school graduate. 1980. Ph.D. Through joint program with MCV- VCW, Richmond Virginia 1990. Expert in Electron Microscopy, (TEM/SEM-EDAX and AFM) (atomic force microscope). Cell and Tissue Cultures, (Healing injury), Heart, particularly, Aortic valve and Vascular tissues are the areas of my targeted research. He established cell culture facilities at MCV –VCU Virginia, veterans' hospital at Washington, DC. And cardio-thoracic dept. George Town medical school at Washington DC. Former director of aortic valve homograft bank, department of Cardiothoracic Surgery at George Town. Currently he is Professor of Anatomy and Embryology Department and Head of the Research Center also he is the Director of Electron Microscope Center (EM) at Ain Shams faculty of medicine

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