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“Accidental and Iatrogenic Ocular Trauma in Canine and Feline Eyes”

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Introduction

Penetrating ocular damage due to surgical or accidental ocular trauma results in the breach of the blood-aqueous barrier, disrupts the visual pathway, and may have important implications for patient vision and comfort. The functional and structural interdependence of intraocular structures predisposes the eye to loss of function with relatively little damage. Accidental trauma with open globe injuries alters ocular architecture markedly. Great effort is expended by the intraocular surgeon to minimize trauma because of the potentially disastrous effects of intraocular inflammation.

Depending on the type and extent of damage, wound healing mechanisms that normally preserve function after tissue damage may alter the alignment or clarity of ocular components, causing visual deficits. In the early stages post-injury, the light path may be obstructed by corneal stromal edema or the presence of hemorrhage or fibrin in the anterior or vitreal chambers. Septic or non-septic endophthalmitis, especially in globes with retained organic foreign bodies, may develop and cause damage to adjacent intraocular structures. Globes in the late stages of an inflammatory response may be affected by contracture of organizing fibroplasia, causing tractional retinal detachment, fibroplasia obstructing the iridocorneal angle or pupil (fibrovascular membrane), or, ultimately, end-stage phthisis bulbi. A negative sequela of ocular trauma is obstruction of aqueous fluid outflow, resulting in elevation of intraocular pressure (glaucoma).

In addition to variances in ocular structure, considerable differences in the response to injury exist among vertebrate species. The acute ocular inflammatory response post-injury is most intense in rabbits and guinea pigs, followed by cats and ducks, while primates have the least severe acute response to a topically applied irritant.[1] Anecdotally, horses and dogs are reported by veterinary ophthalmic surgeons as having relatively higher incidence of surgical complications due to inflammation than other veterinary species (R. David Whitley, personal communication). Pre-iridal fibrovascular membranes, a manifestation of a chronic intraocular inflammatory response,[2] had the highest relative frequency in horses, followed by dogs, cats, and cows.[3]

Ocular trauma occurs in a variety of settings, including accidental injury and surgical interventions. Accidental trauma may be blunt or sharp, and may result in rupture or...
penetration of the globe and uveal prolapse in its most severe form. Blunt force may, however, be severe enough to cause structural damage in a closed globe.

**Hemorrhage and Fibrin**

Vascular damage results in hemorrhage and/or fibrin accumulation within the chambers of the globe. Hemorrhage within the globe is found as hyphema (blood in the anterior chamber), iridal stromal hemorrhage, or within various combinations of the anterior, posterior, and vitreous chambers. Minimization of vascular damage during intraocular surgery is critical to success and fibrinolytic therapy is used clinically to prevent or remove fibrin clots. Differences in levels of intrinsic plasminogen activators exist among anatomic structures and species.[4, 5] Leakage of plasma proteins from damaged vessels results in intraocular fibrin accumulation, activation of the clotting cascade, and stimulating fibroplasia and synechiae formation, which may progress to tractional distortion or damage with organization.

**Corneal Stromal Wound Healing**

Molecular mechanisms associated with successful corneal wound healing may also result in development of corneal opacity. Traumatic disruption of the regularly packed fibrils within corneal stromal lamellae is expected to result in altered light scatter, reducing corneal transparency. Incisional wounding of the corneal epithelium and stroma signal the activation and transformation of previously quiescent keratocytes, with resulting cell phenotype dependent on microenvironmental signals.[6] Transforming growth factor-β (TGF-β) and insulin-like growth factors (IGF-I/II) are key regulators of the wound healing process and drive metaplasia of stromal keratocytes to myofibroblast or fibroblast phenotypes. While wound fibroblasts produce orderly arranged collagen fibrils, myofibroblasts produce disorganized stromal matrix fibrils, resulting in scar formation and opacity.[7] Strategies to reduce myofibroblast development and fibroplasia are hypothesized to provide methods to reduce corneal opacity. [8, 9]

**Consequences of Trauma to the Lens**

Blunt or sharp ocular trauma may damage the lens, causing lens fiber swelling and degeneration (cataract), rupture of the lens capsule, or rupture of zonules causing displacement of the lens (subluxation or luxation). Traumatic cataract may result from focal injury to the lens, such as a penetrating injury, sometimes with sealing of a small lens capsule defect. Lens opacities associated with ocular trauma may occur acutely or may develop weeks after the initial injury.

Release of lens proteins into the eye after capsular rupture is likely to induce severe uveitis.[10] Phacoclastic uveitis results from release of very antigenic non-degenerate lens proteins and is characterized by perilenticular accumulation of lymphocytes,
plasma cells, macrophages, and fibroplasia. In contrast, phacolytic anterior uveitis, associated with leakage of lens proteins from a hypermature cataract, is typically a relatively mild inflammatory response composed of lymphocytes and plasma cells.

Malignant mesenchymal neoplasms occasionally develop after trauma or chronic uveitis in feline globes.[11, 12] In many cases, rupture of the lens capsule and damage to lens fibers is demonstrated. Telomerase activity has been identified in the majority of a series of post-traumatic intraocular sarcomas.[13] Characteristics typical of lens epithelium have been identified in some feline ocular sarcomas, supporting origination in lens epithelium.[14]

**Post-operative complications of lens removal and intraocular lens implantation**

In dogs, phacoemulsification or extracapsular cataract surgery to remove degenerate lens material is frequently followed by implantation of an artificial intraocular lens. Development of intraocular lenses for implantation after lens removal balances material biocompatibility and design for insertion through a small incision. Complications encountered after lens removal include incomplete lens fiber removal with subsequent regrowth, post-operative proliferation of cells within the capsular bag (posterior or anterior capsular opacification). Residual lens epithelial cells on the anterior capsule proliferate and differentiate into myofibroblasts, and migrate along the interior of the lens fiber, sometimes producing extracellular matrix and forming a posterior capsular opacification (PCO), a form of secondary cataract. Incomplete removal of cortical material may become clinically significant if it is present in the visual axis.

**References**


