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Traumatic angle-recession glaucoma: a literature review

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Abstract

Ocular trauma is a main cause of ocular comorbidity worldwide. Cornea or lens injury, secondary glaucoma, vitreous haemorrhage, retinal or choroidal detachment, and endophthalmitis are the sequelae of ocular injury causing vision loss. Many articles have been published identifying the common sequelae of closed globe injuries, including the risk of developing secondary glaucoma from angle recession. This review article aims to cover the sequelae of closed globe ocular trauma, the definition of traumatic angle-recession glaucoma (TARG), and the natural course, detection, management, and prophylactic treatment of TARG.

Keywords: angle recession, closed-globe trauma, traumatic angle-recession glaucoma

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Glaukoma sekunder kemelesetan sudut akibat trauma: tinjauan literatur

Abstrak

Kecederaan pada mata adalah penyebab utama masalah komorbid melibatkan mata di seluruh dunia. Kecederaan pada kornea atau kanta, glaukoma sekunder, pendarahan pada vitreous, lekangan retina atau koroidal adalah kesan kecederaan pada mata yang boleh menyebabkan hilang penglihatan. Terdapat banyak artikel yang dibentangkan untuk mengenalpasti kesan kecederaan bola mata tertutup ataupun kecederaan akibat benda tumpul, termasuklah risiko terbentuknya glaukoma sekunder akibat daripada kemelesetan sudut (angle recession). Artikel ini bertujuan untuk meninjau literatur berkaitan dengan kesan kecederaan akibat bola mata tertutup termasuk mengenai definisi glaukoma kemelesetan sudut akibat trauma (GSKT atau TARG) serta kesan jangka panjang, cara pengesanan, rawatan dan profilaksis.

Kata kunci: glaukoma sekunder akibat daripada kemelesetan sudut, kecederaan bola mata tertutup, kemelesetan sudut

Introduction

Ocular trauma is a common cause of ocular comorbidity with 19 million people suffering from trauma-related monocular blindness or low vision worldwide.[1] Approximately three-quarters of a million patients are hospitalized every year due to eye injuries throughout the world.[2] In Australia, The National Eye Health Survey (NEHS) for 2016 reported approximately 2.4 and 7.9 per 1,000 nonindigenous and indigenous adults suffered monocular vision loss as a result of eye injury.[3] Several studies have shown that 90% of ocular trauma is preventable.[1-4] Agriculture related injury contribute among the most important causes of visual loss in developing countries.[1-4]

In many population-based studies, there is a higher incidence of ocular trauma in males compared to females.[5] This has been also reported in a study in Kuching, Malaysia, where the percentage is greater in males, 85.8% (n = 200), with a male-to-female ratio of 6:1.[6] The highest percentage occurs is young adults in the third decade of life and the second peak is in the elderly.[5,6] There are a variety of causes of ocular trauma, mainly work related injury, while others include home-related injury, violence-related injury, and road related injury.[7] While the greater incidence in males is likely multifactorial, *e.g.*, aggressive behaviour, work or sports related, assault, and alcohol and drug abuse, the incidence of ocular trauma in the elderly

occurs as a result of poor vision probably secondary to other ocular conditions such as cataract, diabetic retinopathy, glaucoma, and age-related macular degeneration.

Ocular trauma has increasingly become a public health burden worldwide. It is estimated that the hospital charges in the United States were between USD 175 million and USD 200 million per year in 1986, which required 227,000 days of hospital care.[8] In Australia, open-globe injuries are responsible for 44% of the expenditure on ocular injuries, with an estimated cost of USD 155 million per year.[9]

For individuals, ocular injuries have a huge psychologic and economic impact due to absence from work, frequent hospital visits, and treatment and rehabilitation costs, especially in the reproductive age group.[8] In younger age groups, ocular trauma affects school performance and family dynamics, in addition to its psychologic and social impact.[10]

Impact of trauma

Ocular trauma has several aetiologies and differs between urban areas and other settings, as well as between countries, world regions, and demographic and socioeconomic classes. It ranges from minor injuries to major injuries resulting in vision loss or loss of the eye. Initial eye assessment and early diagnosis, appropriate first aid from a primary team with prompt referral to an ophthalmology team, and early treatment is very important as ocular trauma can lead to sight-threatening complications. The Birmingham Eye Trauma Terminology (BETT) classification system provides a consistent and comprehensive system for ocular trauma which classifies injuries as closed or open globe.[11] The modified BETT classification system includes periocular injuries with or without retained foreign bodies.[12] However, ocular trauma also can be broadly classified based on the causative object and extent of the injury, such as blunt or penetrating ocular injury.[13] The diagnosis and treatment of various ocular traumas differs depending on the underlying aetiology, severity of the case, and potential complications.

Ocular trauma may result in pathology of the eyelids, lacrimal drainage system, ocular surface and adnexa, extraocular muscles, orbital walls, eyeball, and optic nerve. The ocular structures in the anterior segment, *i.e.*, conjunctiva, cornea, trabecular meshwork, crystalline lens, and iris, are commonly affected by direct trauma compared to the structures within the posterior segment, *i.e.*, optic nerve, choroid and retina.[14] Combined anterior and posterior segment injuries cause worse prognosis and poor visual outcome.[15-17]

Although protected by the bony orbit, the structures around the globe are also prone to traumatic injuries. Orbital compartment syndrome, globe laceration, eyelid laceration, and damage to the lacrimal drainage system are among the injuries reported around the globe.[18] While the anterior segment is commonly

exposed to minor trauma, with corneal abrasion as the most common injury presenting at primary care,[19] other type of injuries also commonly seen, including corneal foreign bodies, corneal laceration, hyphaema, lens dislocation, and traumatic cataract.

Local or systemic injuries may cause abnormalities in the posterior segment. They may be asymptomatic or worsen to cause vision loss. Blunt trauma can cause local injury and may damage the retina (commotio retina), choroid (choroidal rupture), and optic nerve (optic nerve avulsion). Systemic trauma may result in diffuse retinopathy (Purtscher's retinopathy), shaken baby syndrome, or localized retinal abnormalities (whiplash retinopathy, fat embolism syndrome).[20]

Closed-globe ocular trauma

According to Khokhar *et al.*, open-globe injuries occur more frequently than closed-globe injuries; however, other studies have found that closed and open-globe injuries occur at rates of 41.9% and 59.4%, respectively.[21,22] The BETT classification system broadly classifies closed-globe ocular trauma as contusion injury or lamellar laceration involving partial-thickness wounds of the eyeball.[11] The location of injury is further classified into 3 zones base on anteroposterior anatomic location of the injury:

- Zone 1: Superficial injuries limited to the bulbar conjunctiva, sclera, or cornea, including corneal abrasion and subconjunctival haemorrhage.
- Zone 2: Injuries involving anterior segment structures up to and including the lens apparatus, the lens zonules, and the pars plicata.
- Zone 3: Posterior injuries involving the pars plana, choroid, retina, vitreous, and optic nerve.[23]

The location and extent of the ocular injury are significantly determined by the different eye structures exposed to the injury and the mechanism of injury.

Sequelae of closed-globe ocular trauma

In closed-globe injuries, the ocular manifestations can be impacted by a wide range of closed-globe damage. In general, the anterior segment is the most injured structure in an ocular contusion. Traumatic cataract and glaucoma resulting from ocular trauma can also occur in the setting of both open- and closed-globe injuries. In closed-globe injury, the risk of developing secondary glaucoma has been associated with angle recession, hyphaema, iris injury (iridodialysis), lens injury, corneal injury, and vitreal injuries (intravitreal haemorrhage, vitreous loss)[24,25]

Various posterior segment abnormalities have been reported as result of closed-globe injury. The sequelae of closed-globe injury in the posterior segment include traumatic retinal tear, detachment, or dialysis, traumatic macular hole, commotio retina, contusion of the retinal pigment epithelium, choroidal rupture, chorioretinitis sclopetaria, and optic nerve avulsion.[20,26]

In a study among 122 cases of closed-globe contusion injury, 89 (73%) had traumatic hyphaema with 8 of the eyes (8.9%) had re bleeding. One eye out of that eyes complications including secondary glaucoma, cataract and posterior segment injuries that necessitated surgical intervention.[24] Poor visual outcomes are seen in globe ruptures, zone 3 injuries, poor initial visual acuity, wound length > 10 mm, and lens trauma.[24] In another study, closed-globe injury with severe vitreous haemorrhage was associated with a high incidence of retinal defect (retinal tear and dialysis) and retinal detachment.[25]

Visual outcomes appear to depend on the severity of the closed-globe injury. In a study by Shah *et al.*, among 1,010 individuals in the paediatric age group (0–18 years) with closed-globe injury with involvement of 1-6 tissues in all cases, 649 eyes (64.3%) regained > 6/24 visual acuity, whereas 247 eyes (24.5%) did not regain more than 1/60.[21] However, in a study reported by Edita *et al.*, most closed-globe injury (81.7%) cases did not cause any final visual impairment in the affected eye as compared to open-globe injury.[27] However, these findings must be understood the study's retrospective nature, which contained a large number of underreported ocular trauma associated with other multiple head injuries and difficulty in determining visual acuity from the ophthalmic examination.[27]

Definition of traumatic angle-recession glaucoma

Angle recession is a common finding after blunt traumatic injury and is usually associated with traumatic hyphaema. Although it can also occur without haemorrhage in the anterior chamber, the presence of traumatic hyphaema has been reported in 71–100% of eyes.[28] Other conditions also associated with angle recession include iridodialysis, iris sphincter tears, and transillumination defects with pigmented dispersion.[29]

Generally, angle recession is defined as the separation of the longitudinal and circular muscle fibres of the ciliary muscle, which can be seen by gonioscopy at the slit lamp following trauma.[30] Histologically, angle recession is detected by a tear between the longitudinal and circular fibres of the ciliary muscles. It was first described in 1982 by Colin, who observed the deformity in anterior chamber angles of enucleated eyes after nonpenetrating trauma.[31] Later, the correlation between traumatic angle recession and late-onset glaucoma was established in 1962 by Wolff and Zimmerman.[30] The proposed mechanism of angle recession after closed-globe injury is the sudden force in anteroposterior compression causing sudden equatorial expansion, and resultant outward movement of the ocular wall causing the injury.[30] Angle recession may develop into secondary glaucoma in a small percentage of eyes months or years after the trauma.[32] In a 10-year prospective study of 31 eyes by Kaufman and Tolpin, glaucoma developed in 6% of cases of angle recession.[32]

Natural course of traumatic angle-recession glaucoma

The incidence of glaucoma in angle recession after trauma appears at two peaks: early (less than 1 year) and late (after 10 years).[28,32] Some cases have reported traumatic angle-recession glaucoma more than 50 years after the initial injury.[33] In early onset of traumatic angle-recession glaucoma, the intraocular pressure (IOP) can be normal and the decreased outflow facility compensated by ciliary body hyposecretion, increased uveoscleral outflow, small cyclodialysis cleft, or trabecular tear causing communication between Schlemm's canal and the anterior chamber.[31] The IOP may be elevated after these factors are normalized. After a period, the trabecular meshwork regains its function with normal outflow facility and IOP; however, the IOP may be persistently elevated.[31] A small percentage of these individuals go on to develop glaucomatous optic neuropathy and vision loss. This late-onset angle recession glaucoma detection is important as it always presents late. Other causes of secondary open angle glaucoma must be ruled out as it always present unilaterally and years after the injury. In one study , elevated IOP was detected in the contralateral injured eye and this increased the risk of open angle glaucoma.[34] This has led to the hypothesis that angle recession glaucoma accelerates the predisposition to developing open angle glaucoma bilaterally.[34,35]

Detection of traumatic angle-recession glaucoma

Traumatic angle recession is diagnosed based on the history and clinical findings. Angle recession must always be considered in cases of unilateral glaucoma or traumatic hyphaema after blunt trauma.[28,32] Clinically, angle recession is detected by slit lamp gonioscopy examination using indirect gonioscopy lenses (*e.g.*, Zeiss or Goldmann lens).[33]

In 1962, Wolff and Zimmerman described angle recession in 17 of 300 eyes which had been enucleated post-contusion injury.[30] The inner circular muscles of the ciliary body appeared to be separated while the longitudinal muscle remain attached to the scleral spur, thus forming a new boundary to the recessed angle. The circular muscles fibre later became atrophied and advanced degenerative changes were seen in the trabecular meshwork. These degenerative changes showed predominantly atrophy, with or without presence of fibrosis or a hyaline membrane covering the inner surface of the trabeculum. This correlation explained the late onset of glaucoma with angle recession.[36] These changes damage the angle and trabecular meshwork, causing chronic lOP elevation with subsequent optic nerve damage. This rise in pressure may occur many months, years, or even decades after the ocular injury. Similar findings in a study by Melamed *et al.* suggested that collateral damage to the trabecular meshwork and Descemet-like membrane extension from the cornea over the trabecular meshwork results in elevation of IOP.[36]

According to the findings of Wolff and Zimmerman, there may be different gonioscopic findings in early or late cases. The appearance of the ciliary body in angle recession varies depending on the degree and extent of the injury.[37] Soon after the injury, the cleft into the face of the ciliary muscle is sharply demarcated while later the healing process causing fibrosis or development of hyaline membrane makes the cleft less defined. Small peripheral anterior synechiae seen at the lateral limits may extend into the peripheral area of angle recession, which may hide the angle recession from view. Comparison with the uninjured eye is helpful, especially in cases of minor ciliary body damage or in 360° recession, as these signs are occasionally hard to describe precisely. Frequently, the examiner must always change the lens from one eye to the other eye to look for subtle changes.[38] Minor recession appears as a disruption of the regular pattern of insertion of the iris fibres into the ciliary body or scleral spur.[37] For more severe injuries, the cleft extends into the ciliary body, the light grey portion of the ciliary band is broadened, and the scleral spur is more prominent.[37,39]

Howard and coworkers have proposed a classification of angle recession based on the depth of ciliary muscle tears.[37] In shallow tears, there is a separation of the uveal meshwork's processes, making the ciliary body band and the scleral spur more obvious than in the contralateral eye. Pigmented tags are seen at anterior surface on the peripheral iris, on the ciliary body band, on the scleral spur, and on the posterior portion of the trabecular meshwork. Compared with the contralateral eye, the ciliary body band seems darker and wider, and the scleral spur appears whiter. In shallow tears, no cleft is seen in the face of the ciliary body. In moderate tears, a definite cleft appears in the fibres of the ciliary muscle, and the angle looks deeper than that of the contralateral eye. Deep tears are characterized by ciliary body fissure, and the apex of the fissure cannot be seen by gonioscopy.

In moderate to severe angle recession, ultrasonographic biomicroscopy is useful when visualization of the angle structures is limited due to corneal opacity or other associated injury.[40] Angle recession may also be confused with cyclodialysis, in which the ciliary body is separated from scleral spur. It appears as visible white sclera posterior to the scleral spur and is associated with hypotony. Other differential diagnoses for angle deformity include iridodialysis, trabecular tears, and angle abnormalities secondary to previous ocular surgery.[35] Other causes of unilateral or asymmetrical glaucoma, such as uveitis, anterior segment tumours, lens induced glaucoma, pseudoexfoliation glaucoma, and glaucoma secondary to elevated episcleral venous pressure, should also be ruled out.

In a retrospective study by Razeghinejad *et al.* comparing 40 eyes with traumatic glaucoma after closed-globe injury and 52 eyes with no evidence of glaucoma after closed-globe injury, the features that were significantly associated with traumatic glaucoma included hyphaema, angle recession > 180°, lens displacement, and trabecular pigmentation.[29.] All these factors could be attributed to ciliary body

damage. An inflammatory response can occur not only at the site of injury but also throughout the ciliary body, iris, and trabecular meshwork. In the same study, IOP > 21 mmHg or more for at least 3 months was diagnosed as traumatic glaucoma.

Management of traumatic angle-recession glaucoma

The initial treatment for angle recession glaucoma is medical therapy that decreases aqueous formation, *e.g.*, beta-blockers, alpha2-agonists, or carbonic anhydrase inhibitors.[38,41,42] Cholinergic agents may not be beneficial to the patients, as they may paradoxically increase IOP probably by reducing the uveoscleral outflow while the angle recession compromises the trabecular outflow.[43] Treatment with miotics can cause increased vascular permeability and lead to fibrin clot formation at the anterior chamber in the acute phase, which increases the chances of posterior synechiae formation and later secluded pupil.[44] Prostaglandin analogues, which increase the uveoscleral outflow, are a choice after inflammation has resolved in the acute phase. The cases with immediate increased IOP after trauma are usually self-limiting and can be controlled with medication alone.[28,31,44]

Although several studies have demonstrated the efficacy of selective laser trabeculoplasty in treating various types of secondary glaucoma,[31,46] laser trabeculoplasty has had some success in the short term[31,33] and is not effective in the long term, especially for patients with angle recession greater than 180°.[31,46] Good outcomes were observed in 3 of 4 patients in a case series cases early in the post-treatment period and maintained for years after selective laser trabeculoplasty.[47] However, it was suggested that larger studies should confirm the safety and effectiveness of selective laser trabeculoplasty. Laser trabeculoplasty for angle-recession glaucoma was also reported as unsuccessful in cases where maximum tolerable medications failed to control IOP.[46]

Some success has also been observed with Nd:YAG laser trabeculopuncture.[31,48] Argon laser trabeculoplasty led to failure in 7 of 11 patients within 3 months, but patients had controlled IOP with Nd:YAG laser trabeculopuncture over 15 months of follow up.[48] In another study, however, Nd:YAG laser trabeculopuncture was not very effective, and the authors suggested offering it only in eyes where at least part of the trabecular meshwork maintains its normal anatomy without angle recession on gonioscopy.[35]

Surgical intervention in angle-recession glaucoma is more challenging than in primary open angle glaucoma. Trabeculectomy remains the first surgical choice, although failure rates are higher in angle recession (74%) compared to primary open angle glaucoma (43%) as reported in one study.[41] The use of adjunctive antimetabolites with trabeculectomy appears to be successful as first surgical procedure and Molteno implantation as a secondary procedure in angle recession

glaucoma. However, bleb associated infection rates are greater in angle recession patients.[41] The risk factors for surgical failure may be related to younger patients and comorbid trauma related to ophthalmic damage. It is also noticeable that fibrosis at the bleb develops earlier after trabeculectomy and requires additional medical or surgical therapy. The low success rate of glaucoma surgery is probably due to the high tendency for fibroblast proliferation and/or changes in aqueous humour properties.[39,49] This may be due to the absence of fibroblast growth inhibitory factors in the aqueous or the presence of stimulatory growth factors in these eyes.[34,49]

Prophylactic treatment of traumatic angle-recession glaucoma

Glaucoma in angle recession typically develops after 6 months to many years. In patients with angle recession > 270° degrees it often develops earlier. Mooney *et al.* found that angle recession < 180° does not develop late-onset glaucoma.[39] Lifelong annual examination in patients with angle recession > 180° has been suggested for late-onset glaucoma detection. Patients at high risk for developing late-onset glaucoma should be identified through careful evaluation to receive appropriate treatment.

Conclusion

Traumatic angle recession is a common finding in eyes with closed-globe trauma. The sequelae of blunt ocular trauma can be devastating and may cause monocular blindness. Understanding the underlying natural course of traumatic angle recession is important in choosing appropriate treatment approaches. Since it is not common and may be difficult to manage, clinical guidelines on management of traumatic angle recession are needed for early detection of glaucomatous changes. Early recognition and identification of individuals at risk of developing traumatic angle recession glaucoma is critical to ensure follow up examinations and treatment at an early stage before vision becomes seriously impaired.

Declarations

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