

Causes and treatments of traumatic secondary glaucoma

HAI-QING BAI, LIN YAO, DA-BO WANG, RUI JIN, YUN-XIAO WANG

Department of Ophthalmology, Affiliated Hospital of Medical College, Qingdao University, Qingdao - China

PURPOSE. *To study the cause, treatment, and cure of traumatic secondary glaucoma in 103 cases (103 eyes).*

METHODS. *The records of 103 patients (103 eyes) were reviewed. Causes of the high intraocular pressure (IOP) were analyzed according to the time after trauma. Most patients achieved a better visual acuity and ideal IOP after positive medical, neodymium:Yttrium aluminium garnet (Nd:YAG) laser, or surgical treatment. The operations included anterior chamber irrigation, filtering operation combined with mitomycin C, lensectomy, vitrectomy, and combination surgery.*

RESULTS. *Clinical findings of secondary glaucoma associated with ocular trauma are complex. Causes resulting in high IOP include intraocular bleeding, lens dislocation, phacoanaphylaxis, angle recession, and siderosis. After medical, laser, or surgical treatment, the IOP of most patients could be ideally controlled. After follow-up for half a year, the IOP of 3 cases (2.91%) was below 10 mmHg and 92 (89.32%) cases between 10 and 21 mmHg; only 8 cases (7.77%) still had IOP over 21 mmHg.*

CONCLUSIONS. *In traumatic secondary glaucoma, antiglaucoma medication should be used at the early stage, and surgery should be carried out when medical treatment does not reduce the elevated IOP, or in difficult cases to avoid severe complications. The postoperative IOP of most injured eyes was controlled within the safe range. (Eur J Ophthalmol 2009; 19: 201-6)*

KEY WORDS. *Ocular trauma, Glaucoma, Cause, Treatment*

Accepted: August 14, 2008

INTRODUCTION

Ocular trauma, or ocular contusion, often results in hyphema, angle recession, lens dislocation, trabecular meshwork injuries, and inflammation, which can block the outflow of aqueous humor, thus leading to secondary glaucoma (1-5). Along with elimination of the blood and inflammation, the function of the trabecular meshwork may achieve recovery or compensation. Because damage to the visual function induced by high intraocular pressure (IOP) can usually be avoided through positive treatment, intensive follow-up is necessary in these patients. If the IOP cannot be controlled by medicine, in time surgical intervention is necessary. As for the relationship between medically controlling IOP and the need for surgery, we

prefer medical treatment early after surgery, except for anterior lens displacement, which needs emergency surgery because of lens corneal touch. Surgical intervention is indicated when medical treatment does not reduce the elevated IOP, or in difficult cases to avoid severe complications. Many of the complications that may develop are preventable. Management is aimed at preventing further damage to the injured eye. According to the mechanism of intraocular hypertension, the traumatic secondary glaucoma can be divided into inflammation glaucoma, angle recession glaucoma, hyphema glaucoma, lens injury glaucoma, and siderotic glaucoma. We practiced correspondingly medical or surgical treatments on these patients based on their different causes. Anterior chamber irrigation was recommended to hyphema, lensectomy to

the subluxated lens, in some cases combined by vitrectomy, neodymium:Yttrium aluminium garnet (Nd:YAG) laser iridotomy to pupil block, removal of the lens and thorough anterior chamber irrigation to phacoanaphylactic glaucoma, trabeculectomy to angle recession glaucoma and other cases that failed in lowering the IOP after the above surgery intervention, and lensectomy combined with vitrectomy and trabeculectomy to siderotic glaucoma. The postoperation IOP among most patients was controlled within the safe range after at least half a year's follow-up. After one or more subsequent operations, the success rate of surgical intervention was 92.23% (95 among 103 cases) (the inclusion criteria of successful surgery are defined as follows: half a year after one or more subsequent operations, with or without antiglaucoma drugs, IOP ranges from 6 to 21 mmHg, no serious complications). Here we share some experiences treating trauma-related glaucoma, a relatively complex clinical scenario.

METHODS

Case selection and classification

In all, 103 patients (103 eyes) with ocular trauma related glaucoma were admitted into our department during January 2004 until January 2006. They were between 11 and 86 years old, and the mean age was 44.71 ± 19.83 years. A total of 76 patients were male, and 27 female. A total of 70 patients were injured by closed globe trauma, and 33 patients by open globe trauma, including 1 by intraocular foreign body. The IOP of 28 patients was between 30-40 mmHg, 49 patients between 41-50 mmHg, 26 patients beyond 50 mmHg. Their visual acuity varied from light perception to 0.2 (Tab. I). The selected patients were clas-

sified into three categories according to the onset time of glaucoma after ocular trauma, including early stage, within 1 month after ocular trauma, intermediate stage, 1 to 6 months, and advanced stage, more than 6 months. During the early stage, high IOP developed due to inflammation in 33 eyes (32.04%), hyphema in 36 eyes (34.95%), and lens dislocation in 22 eyes (21.36%). In intermediate stage, 3 (2.91%) eyes developed glaucoma due to pupil block, and 2 (1.94%) eyes showed phacoanaphylactic glaucoma. During advanced stage, the IOP gradually rose in 6 (5.82%) eyes due to angle recession, and in 1 (0.97%) eye due to siderosis.

Treatments

Early stage. Eyes with inflammation were given 0.1% dexamethasone eyedrop and dexamethasone 0.1-0.2 mg/Kg/d through intravenous route for 5 to 7 days. Drugs for depressing IOP were also necessary, such as 0.5% timolol, 0.2% brimonidine tartrate, 0.005% latanoprost, and 20% mannitol. Trabeculectomy with or without mitomycin C was carried on when medical treatment failed to depress the high IOP. Eyes with hyphema needed careful inspection and follow-up. If the hyphema showed no signs of resolving for more than 5 days, surgical intervention was indicated to remove the blood, as well as to reduce the high IOP. Anterior chamber irrigation was chosen first. After irrigation, if IOP still could not be controlled with the help of medicine, trabeculectomy with or without mitomycin C was taken.

Eyes with lens dislocation underwent lensectomy. If lens dropped into vitreous cavities, vitrectomy would be done at the same time. If IOP could not be controlled ideally, further trabeculectomy with or without mitomycin C was adopted as well.

Intermediate stage. Patients with pupil block underwent neodymium:Yttrium aluminium garnet (Nd:YAG) laser iridotomy first. If IOP still could not be controlled, trabeculectomy was considered. Patients with phacoanaphylactic glaucoma were given lensectomy combined with thorough anterior chamber irrigation.

Advanced stage. Patients with angle recession underwent trabeculectomy with or without mitomycin C. Patients with siderotic glaucoma underwent trabeculectomy with or without mitomycin C combined with lensectomy.

TABLE I - PREOPERATIVE AND POSTOPERATIVE (final visit) VISUAL ACUITIES

Visual acuity	Preoperatively, n (%)	Postoperatively, n (%)
Light perception	11 (10.68)	6 (5.83)
Hand movements	23 (22.33)	15 (14.56)
Counting fingers	30 (29.13)	22 (21.36)
0.02-0.2	39 (37.86)	22 (21.36)
0.3-0.5	0	28 (27.18)
0.6-1.0	0	10 (9.71)

RESULTS

IOP

Table II shows the different causes, respective treatments, and IOP outcomes according to treatments in different stages of the ocular trauma related glaucoma. All patients were followed for at least 6 months.

Table III indicates the results of IOP after long-term follow-up. However, with time elapsed, many patients gave up further examination, and their data were unavailable.

Visual acuity

The visual acuity of all eyes with early onset glaucoma was enhanced after treatment. Eyes with glaucoma due to intermediate or advanced trauma had improved or equal visual acuity versus that before treatment (Tab. I).

DISCUSSION

Causes of traumatic secondary glaucoma are complicated. Elevated IOP may be caused by a single factor,

but most of the time it results from multiple factors combined. The mechanisms for the various types of traumatic secondary glaucoma are different, depending on the degree and extent of the related injured tissue. The risk factors for ocular contusion secondary glaucoma include age, visual acuity regression, lens injury, and angle recession (1). The key point of open traumatic secondary glaucoma is prevention. The angle of anterior chamber should be carefully preserved by reforming a flat anterior chamber and avoiding the block of aqueous humor during one-stage operation. Usually sodium hyaluronate is necessary to maintain the integrity of the angle of the anterior chamber. According to the mechanism of ocular trauma related glaucoma, we divide traumatic secondary glaucoma into inflammation related glaucoma, angle recession related glaucoma, hyphema related glaucoma, lens injury related glaucoma, siderosis related glaucoma, and other causes like vitreous agent. We also divide ocular trauma related glaucoma into three stages, including early stage, intermediate stage, and advanced stage, based on the time of the high IOP rise after injury. Clinically, therapeutic measures are carried out accordingly based on different causes.

TABLE II - CAUSES, TREATMENTS, AND INTRAOCULAR PRESSURE (IOP)

Stage	Cause	Treatment	No.	IOP 6 mo after treatment, mmHg		
				<10	10-21	>21
Early stage (<1 mo)	Inflammation	Medicine	17	0	16	1
		Trabeculectomy	16	1	14	1
	Hyphema	Medicine	8	0	7	1
		Anterior chamber irrigation	12	0	12	0
	Lens dislocation	Anterior chamber irrigation+ trabeculectomy	11	0	9	2
		Trabeculectomy	5	1	4	0
		Lensectomy	4	0	4	0
		Lensectomy+vitrectomy	15	1	13	1
	Trabeculectomy*	3	0	3	0	
Intermediate stage (1-6 mo)	Pupil block	Nd:YAG laser iridotomy	1	0	1	0
		Trabeculectomy†	2	0	2	0
	Phacoanaphylactic glaucoma	Lensectomy+anterior chamber irrigation	2	0	1	1
Advanced stage (>6 mo)	Angle recession	Trabeculectomy	6	0	5	1
	Siderosis	Lensectomy+trabeculectomy	1	0	1	0

*Trabeculectomy in case of failure in controlling IOP after lensectomy combined with vitrectomy.

†Trabeculectomy in case of failure in controlling IOP after neodymium:Yttrium aluminium garnet (Nd:YAG) laser iridotomy.

Early stage

Within 2 weeks to 1 month after injury, usually, the causes for elevated IOP are inflammation, hyphema, and lens dislocation.

Inflammation-related glaucoma. Inflammation is one of the major reactions from trauma and can induce high IOP secondary to outflow obstruction from inflammatory cells, debris, or protein in the anterior chamber, and possibly due to inflammation at the trabecular meshwork. This elevated IOP can occur without hyphema, angle recession, or disruption of the trabecular meshwork (2). Seventeen cases (16.50%) in this study were given corticosteroid and nonsteroid anti-inflammatory drug systematically and topically for 1 to 2 weeks. The IOP was controlled effectively. Obvious permanent lesion of visual function did not occur according to the scotometer examination. However, there were 16 cases (15.53%) whose IOP could not be controlled by medicine. They were given trabeculectomy with the use of mitomycin C, until the IOP was controlled.

Hyphema related glaucoma. Hyphema may be one of the most common signs of ocular contusion (4, 6), and it is often associated with angle recession (4, 5, 7). Hyphema is also one of the most common causes of ocular trauma related glaucoma. High IOP greater than 50 mmHg lasting for 3–5 days or greater than 35 mmHg for 7 days are good indication for an anterior chamber irrigation to wash out blood. Anterior chamber irrigation should also be performed when patients feel great discomfort because of ocular hypertension. In our study, rebleeding did not appear in the 12 cases (11.65%) given anterior chamber irrigation. Thus, rebleeding after anterior chamber irrigation can be avoided, as long as adequate anti-inflammatory therapy and hemostat are given. Since all cases were treated with medicine at least 1 week before anterior chamber irrigation, the effect of anterior chamber irrigation earlier after ocular trauma should be observed in future study. IOP of 11 cases (10.68%) could

not be controlled after anterior chamber irrigation, and trabeculectomy with mitomycin was finally performed. Two cases among them whose preoperative IOP was beyond 50 mmHg showed serous choroidal detachment after the operation. Therefore, before and after this procedure anti-inflammatory therapy was very important for these patients. Thirteen cases (12.62%) with hyphema did not undergo anterior chamber irrigation because of less bleeding. After medical treatment, hyphema was absorbed. In most cases, the IOP was reduced to normal, while 5 cases (4.85%) underwent trabeculectomy with the use of mitomycin C because of high IOP after blood cleansing.

Lens dislocation related glaucoma. In our study, 22 (21.36%) patients had secondary glaucoma resulting from lens dislocation or subluxation. In case of anterior displacement of the lens, there is not only the high IOP, but also a high risk for early corneal endothelium injury because of lens corneal touch. Therefore, the 4 patients (3.88%) with lens anterior displacement were given lensectomy to relieve the papillary block as emergency case. After lensectomy, their IOP was controlled. The other 18 cases (17.48%) had lens posterior displacement, which underwent lensectomy combined with vitrectomy. The IOP of 15 cases (14.56%) was controlled after surgery, while the other 3 cases (2.91%) failed. They were then given trabeculectomy with mitomycin C in 2 weeks. These cases are often associated with the rupture of the vitreous anterior limiting membrane. Medical treatment is not very effective due to involvement of vitreous, and filtering surgery may easily fail. In early stage, lensectomy combined with vitrectomy is a better choice. Once vitreous prolapses into the anterior chamber, it may result in permanent lesion of the trabecular meshwork.

Intermediate stage

One to 6 months after injury, the cause for elevated IOP is usually the complications of ocular trauma, such as pupil block caused by wide posterior synechia of the iris, goniosynechia, and endophthalmitis caused by lens injury. Few penetrating ocular traumas result in posttraumatic glaucoma. Intraocular inflammation, lens injury, and advancing age may be the main factors (1).

Complications of inflammation in anterior segment. Three cases (2.91%) had secondary angle-closure glaucoma because the inflammation caused by ocular trauma was not controlled in time, so synechia iridis anterior peripherica or pupil block happened, which resulted in angle closure.

TABLE III - INTRAOCULAR PRESSURE AFTER LONG-TERM FOLLOW-UP

	0.5~1 yr	1~1.5 yr	1.5~2 yr	>2 yr
6~10 mmHg	2	2	1	1
10~21 mmHg	65	52	39	32
21~35 mmHg	7	6	6	4

Steroids and nonsteroidal anti-inflammatory drugs should be taken first; at the same time, drugs for depressing IOP were also necessary. Nd:YAG laser iridotomy should be done in case of pupil block. When IOP cannot be depressed by drugs or Nd:YAG laser iridotomy, filtering surgery may be considered. Intraocular and conjunctival inflammation significantly enhanced fibrosis after filtering surgery. Therefore, morphologic change of the bleb should be observed carefully and intensively during early period of follow up in order to detect the blebs that incline to fail. For the failing bleb, 5-fluorouracil (5-FU) was used through subconjunctival injection.

Phacoanaphylactic glaucoma. Two eyes with phacoanaphylactic glaucoma had the main symptom of gradual inflammation and elevated IOP which could not be controlled by drugs. Rupture of the anterior lens capsule and cortex egress was obvious in one case. The other one showed severe iridocyclitis and elevated IOP. Cortex outflow and traumatic cataract became obvious 2 months after ocular trauma. Both eyes had extracapsular cataract extraction followed by sufficient anterior chamber irrigation. Their IOP was still beyond 21 mmHg shortly after surgery, so antiglaucoma drugs were supplied until the IOP declined 1 month later. As corrected visual acuity was better in one case, implantation of intraocular lens was carried out 2 months later. The main reason for high IOP in these cases was the outflow of cortex, so thoroughly removing the cortex should be focused on during operation. After surgery and anti-inflammation treatment, IOP of most patients can be controlled, so filtering surgery is not necessary.

Advanced stage

More than 6 months after ocular trauma, the common cause of high IOP is angle recession, or another uncommon reason such as siderosis.

Angle recession glaucoma. Angle recession glaucoma is an advanced stage complication of blunt ocular contusion. In the presence of hyphema, angle recession has been observed in 71% to 100% (3). Although a common finding, only 7% to 9% of patients with angle recession develop glaucoma (4, 5, 8, 9). Late onset increased IOP related to contusion trauma is the character of angle recession glaucoma. There is a deep slit into the ciliary body, while the tip of the slit cannot be seen by gonioscope. Thus, patients with angle recession should be followed up for a relatively

long time. Gonioscopy examination should be done first in the contralateral uninjured eye for comparison (7, 10), in order to avoid missing diagnosis because of the anterior iris synechia covering the recessed angle. However, the risk of glaucoma caused just by angle recession appears seldom. Therefore, the cause of elevated IOP after ocular contusion should be identified carefully. Surgery intervention cannot be carried on cursorily. Prostanoid is the better antiglaucoma drug for these patients, such as latanoprost, which can efficiently depress the IOP. The possible explanation may be changes of the outflow pathway of aqueous humor in these angle recession patients, which facilitates the egress of aqueous humor. The exact mechanisms are unknown. In this study, 6 cases (5.83%) were identified as angle recession glaucoma by gonioscopy examination. Duration between ocular trauma and onset of high IOP varied from 2 months to 60 months, and the mean time was 23 months. After the failure of antiglaucoma medication, all of them underwent trabeculectomy with mitomycin C. Nonpenetrating trabecular surgery is not fit for these patients because of the severe damage of the trabecular meshwork, according to our previous experiences. In addition, because of the anatomic structure changes of anterior chamber, the Schlemm's canal may also be damaged. Therefore, nonpenetrating trabecular surgery should be avoided in these cases. Trabeculectomy with mitomycin C should be the first choice. Related studies indicated that the surgery achievement ratio could be elevated by using mitomycin C in trabeculectomy for traumatic angle recession glaucoma patients (11, 12).

Siderotic glaucoma. Ironic intraocular foreign body can cause siderotic glaucoma, which is a rare condition. Late elevation in IOP may be related to iron toxicity (13). Surgery should be taken on as early as possible once diagnosed. Lee et al pointed out that in case of intralenticular foreign body, the crystalline lens served as a natural barrier to irreversible intraocular toxicity (14). The barrier could be broken and other intraocular tissue could also be affected by iron toxicity as the iron foreign body stayed long enough in the lens. There was only one patient in this study who was diagnosed with siderotic glaucoma and traumatic cataract, with ironic foreign body in one eye undetected for 3 years, until his vision dropped gradually to light perception and IOP rose to beyond 40 mmHg. The patient underwent trabeculectomy combined with lensectomy and vitrectomy immediately after admission into our department. Though IOP was controlled ideally after surgery, visual function was not

recovered because of persistent iron toxicity to retina and other intraocular tissue. This case reminded us that in patients with repeated and long duration intraocular inflammation, imaging examination should be used to make a definite diagnosis in time. We should be alert to an occult intraocular foreign body in the event of ocular trauma, even if none had been detected during prior imaging examinations (15).

In addition, patients with severe ocular surface disease often displayed high IOP. The overall prevalence of glaucoma in patients with severe ocular surface disease is 65.7%. Early diagnosis and effective treatment can usually depress the onset of concurrent glaucoma (16). However, our data showed little experience in treating this kind of case.

In summary, mechanisms of traumatic glaucoma are complex. The depth of the lesions and the extent along the circumference is variable, without a close relationship between the type and place of the injury. In this study, we artificially classified traumatic glaucoma into early, intermediate, and advanced stages in order to analyze the mechanisms and treatments of injuries more easily. In fact, the evolution of eye injury was continued and uninterrupted. Following the recovery of injured eyes, usually by several weeks to several months, factors such as inflammation and hyphema resulting in high IOP gradually become extinct, while factors such

as scar formation and alteration of the eye structure become the main causes blocking the outflow of the aqueous humor. In the early stage, inflammation, hyphema, and lens dislocation are the main causes. In intermediate and advanced stages, the main causes are inflammatory complications of anterior segment and angle recession. Therefore, choice of treatment is complicated. For patients with definite surgery indication, such as lens dislocation, operation should be done promptly. Conservative treatments are usually needed in patients with ambiguous indication for surgery, because many patients recover from the ocular trauma gradually and eventually achieve controlled IOP without filtering surgery.

The authors have no financial support or commercial interest.

Reprint requests to:
Hai-qing Bai, MD
Department of Ophthalmology
Affiliated Hospital of Medical College
Qingdao University
16th, Jangsu Road
Qingdao, 266003 China
haiqing_bai@126.com

REFERENCES

1. Girkin CA, McGwin G Jr, Long C, Morris R, Kuhn F. Glaucoma after ocular contusion: a cohort study of the United States Eye Injury Registry. *J Glaucoma* 2005; 14: 470-3.
2. De Leon-Ortega JE, Girkin CA. Ocular trauma-related glaucoma. *Ophthalmol Clin North Am* 2002; 15: 215-23.
3. Blanton FM. Anterior chamber angle recession and secondary glaucoma: a study of the aftereffects of traumatic hyphemas. *Arch Ophthalmol* 1964; 72: 39-43.
4. Kaufman JH, Tolpin DW. Glaucoma after traumatic angle recession: a ten-year prospective study. *Am J Ophthalmol* 1974; 78: 648-54.
5. Mooney D. Angle recession and secondary glaucoma. *Br J Ophthalmol* 1973; 57: 608-12.
6. Saunte JP, Saunte ME. Thirty-four cases of airsoft gun pellet ocular injuries in Copenhagen, Denmark, 1998-2002. *Acta Ophthalmol Scand* 2006; 84: 755-8.
7. Tönjum AM. Gonioscopy in traumatic hyphema. *Acta Ophthalmol (Copenh)* 1966; 44: 650-64.
8. Pettit TH, Keates EU. Traumatic cleavage of the chamber angle. *Arch Ophthalmol* 1963; 69: 438-44.
9. Tonjum AM. Intraocular pressure and facility of outflow late after ocular contusion. *Acta Ophthalmol (Copenh)* 1968; 46: 886-908.
10. Salmon JF, Mermoud A, Ivey A, Swanewelder SA, Hoffman M. The detection of post-traumatic angle recession by gonioscopy in a population-based glaucoma survey. *Ophthalmology* 1994; 101: 1844-50.
11. Manners T, Salmon JF, Barron A, Willies C, Murray AD. Trabeculectomy with mitomycin C in the treatment of post-traumatic angle recession glaucoma. *Br J Ophthalmol* 2001; 85: 159-63.
12. Mermoud A, Salmon JF, Barron A, Straker C, Murray AD. Surgical management of post-traumatic angle recession glaucoma. *Ophthalmology* 1993; 100: 634-42.
13. Imaizumi M, Matsumoto CS, Yamada K, Nanba Y, Takaki Y, Nakatsuka K. Electroretinographic assessment of early changes in ocular siderosis. *Ophthalmologica* 2000; 214: 354-9.
14. Lee W, Park SY, Park TK, Kim HK, Ohn YH. Mature cataract and lens-induced glaucoma associated with an asymptomatic intralenticular foreign body. *J Cataract Refract Surg* 2007; 33: 550-2.
15. Lin HC, Wang HZ, Lai YH. Occult plastic intravitreal foreign body retained for 30 years: a case report. *Kaohsiung J Med Sci* 2006; 22: 529-33.
16. Tsai JH, Derby E, Holland EJ, Khatana AK. Incidence and prevalence of glaucoma in severe ocular surface disease. *Cornea* 2006; 25: 530-2.

Copyright of European Journal of Ophthalmology is the property of Wichtig Editore and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.