Secondary Glaucoma - Causes and Management

Uzma Fasih, MS Fehmi, Nisar Shaikh, Arshad Shaikh

Pak J Ophthalmol 2008, Vol. 24 No. 2

..... See end of article for authors affiliations

Correspondence to: Dr.Uzma Fasih B 21 Block 10 Federal B Area Karachi

.....

Purpose: To determine the causes of various types of secondary glaucoma and analyze the efficacy of various management plans for these glaucomas.

Material and Methods: This analytical observational study was conducted at eye department Abbasi Shaheed Hospital Karachi, from June 2005 to June 2007. All patients attended glaucoma clinic were included in the study and their glaucoma was classified and appropriate treatment initiated to achieve the target pressure of 15 mmHg.

Results: In total 106 patient were studied .The number of male patients (66%)was more than the female patients(34%).Most common presenting age group was between 51-60 years forming 28.3%, among secondary glaucomas inflammatory glaucomas were most common (29.2 %). Conservative medical including topical blockers, treatment В prostaglandin analogues, sympathomimetics, miotics, steroids, oral and intravenous hyperosmotic drugs were used. Trabeculectomy was the main surgical option used to control intraocular pressure. Yag laser iridotomies were also done where required. Control of intraocular pressure and visual outcome were quite satisfactory after management.

Received for publication October' 2007

Conclusion: Causes of secondary glaucomas are diverse, inflammatory glaucoma being most common. They could be well managed if present in time. Medical antiglaucoma therapy plays important role in treatment as well as steroids. Trabeculectomy is quite effective in resistant cases.

laucoma is a potentially blinding disease of global importance. It is second leading cause of blindness after cataract¹. Glaucoma is 4th common cause of blindness In Pakistan². The number of people with primary glaucoma in the world by the year 2000 was estimated nearly 66.8 million with 6.7 million suffering from bilateral blindness¹. Information on secondary glaucoma is generally limited but the causes leading to glaucoma are seldom identified. Few studies have described secondary glaucoma as a separate entity but it has been estimated that 6 million people in the world have secondary

glaucoma as compared to 67 million who suffer from primary glaucoma. In India secondary glaucoma represents 6% of total new cases seen annually³.

The causes of secondary glaucoma are diverse but commonly seen entities are inflammatory, lens induced, neovascular and traumatic. It is observed that trauma, cataract and infective uveitis represent special risk factors for development of secondary glaucoma. These are frequent causes of blindness in third world countries⁴.

In Pakistan late presentation of cataract patients, poor management of diabetes, and other diseases leading to neovascular glaucomas, poor management of chronic uveitis and indiscriminate use of topical steroids especially in ocular surface allergies are possible causes of high prevalence of secondary glaucoma³.

Although recent advances in sutureless small incision cataract surgery, phacoemulsification and improved IOL designs have resulted in superior outcome with reduced complications related to wound repair and secondary glaucomas. Special measures and training is required to deal with glaucoma and inflammation secondary to retained lens fragments. These measures will also reduce complications such as pseudophakic glaucoma, another significant cause of secondary glaucoma³.

Lack of facilities in remote peripheries, poverty, illiteracy, ignorance and poor management play an important role leading to complications like uncontrolled IOP, reduced vision, constricted visual fields and glaucomatous optic atrophy. Commonly available current treatment strategies include topical anti glaucoma drugs like topical B blockers, cholinergic agonists, carbonic anhydrase inhibitors topical as well as systemic, oral and intravenous hyperosmotic agents, and prostaglandin analogues. Inflammatory glaucomas respond well to steroids and topical NSAIDS. YAG laser iridotomies do play an important role in angle closure types of secondary glaucoma. Failure of medical treatment leaves no option except to go for surgery as trabeculectomy.

The objective of our study was to determine the causes of various types of secondary glaucoma presenting at our glaucoma clinic and to analyze the efficacy of various management plans for these glaucomas.

MATERIAL AND METHODS

This study was conducted at Eye department Abbasi Shaheed Hospital North Nazimabad Karachi, from June 2005 to June 2007. The patients included in the study were registered at our glaucoma clinic. Complete history regarding ocular complaints and history of any other acute or chronic systemic illness was taken. Information regarding visual aquity, intraocular pressure, slit lamp examination, perimetry, goniocopic and fundoscopy findings was noted on a predesigned performa. Findings like corneal clarity, corneal oedema, anterior and posterior synechia, iris neovascularization and iris atrophy, fundoscopic details regarding condition of optic disc, cup disc ratio, retinal heamorrhages and neovascularization were clearly noted.

Treatment regime of each particular patient was noted on the performa keeping the target intraocular pressure 15 mmHg. Any change in the treatment and details of surgical treatment if any was done were noted down at follow-up visits. IOP was checked with applanation method at every visit. Gonioscopies and perimetries were reevaluated from time to time.

RESULTS

A total of 106 patients of glaucoma were studied who presented at Glaucoma clinic Eye Department Abbasi Shaheed Hospital North Nazimabad Karachi. Male patients were 66% while female patients were 34%. (Table 1). The most common presenting age group was between 51-60 years (28.3%) followed by 61-70 years (18.9%) (Table 2).

Table 1. Gender distribution

| No of patient | Male n (%) | Female n (%) |
|---------------|------------|--------------|
| 106 | 70 (66) | 36 (34) |

| Table 2. | Age | distrik | oution |
|----------|-----|---------|--------|
|----------|-----|---------|--------|

| Age group(years) | Patients n (%) |
|------------------|----------------|
| 1-10 | 1 (0.9) |
| 11-20 | 2 (1.9) |
| 21-30 | 10 (9.4) |
| 31-40 | 15 (14.2) |
| 41-50 | 17 (16.0) |
| 51-60 | 30 (28.3) |
| 61-70 | 20 (18.9) |
| 71-80 | 11 (10.4) |

The common type of secondary glaucoma was inflammatory glaucoma comprising of 45 patients. 31 patients were pseudophakic (29.4%) among them 21 were with posterior chamber implants and 10 were with anterior chamber implants. 2(1.88%) patients presented with aphakic glaucoma with pupillary block. 12(11.3%) patients presented with glaucoma associated with chronic anterior uveitis. 12(11.3%) patients presented with traumatic glaucoma. This was either associated with hyphema and traumatic cataract or angle recession glaucoma. Glaucoma associated with diabetic eye disease was seen in 9 patients (8.4%) patients. 7(6.6%) patients presented with glaucoma secondarily as a complication of hypertension. Among the lens induced group 7(6.6%) presented with phacomorphic glaucoma and 5(4.7%) presented with phacolytic glaucoma. 6(5.6%) patients presented with glaucoma associated with pseudoexfoliation. Among neovascular glaucoma group 3(2.8%) patients were diabetic and 2(1.8%) had central retinal vein occlusion (CRVO) (Table 3).

| Table | 3. Types | of secondar | y glaucoma | and their | management |
|-------|----------|-------------|------------|-----------|------------|
|-------|----------|-------------|------------|-----------|------------|

| Type of secondary glaucoma | No. of patients n (%) | Management |
|---|--------------------------|---|
| 1.Inflammatory | | |
| a.Pseudophakia | 31 (29.2) | |
| PC IOL | 21 | PC IOL=Trabeculectomy 13,Conservative 6, yag laser iridotomies 2 |
| AC IOL | 10 | AC IOL=Trabeculectomy 7, |
| b.Aphakia with papillary block | 2 (1.9) | Conservative 3, Pupilloplasty, synechioplasty, peripheral iridectomy 2 |
| c.Chronic anterior uveitis | 12 (11.3) | Trabeculectomy 5, Conservative 7 |
| 2.Traumatic | 12 (11.3) | Trabeculectomy 5, Conservative 5, yag laser iridotomies 2 |
| 3.Patients with diabetic eye disease | 9 (8.4) | Conservative in all patients |
| 4.Hypertensive patients | 7 (6.6) | Conservative in all patients |
| 5.Neovscular glaucoma | | |
| Diabetic | 3 (2.8) | Diabetic=PRP in 2 diabetic patients, Cryopexy in 1 diabetic patient |
| CRVO | 2 (1.8) | CRVO = Conservative |
| 6.Lens induced | | |
| Phacomorphic | 7 (6.6) | Trabeculectomy + ECCE +IOL 3, *ECCE+IOL 4 |
| Phacolytic | 7 (4.7) | Trabeculectomy + ECCE + IOL 1, ECCE +IOL after conservative treatment 4 |
| 7.Pseudoexfoliation | 6 (5.6) | Trabeculectomy 2, Trabeculectomy +ECCE+IOL 4 |
| 8.Habitual tobacco user | 5 (4.70) | Trabeculectomy 3, Conservative 2 |
| 9.Retinal detachment surgery with silicon oil | 3 (2.8) | Trabeculectomy followed by silicon oil removal 3 |
| 10.Steroid induced | 2 (1.8) | Trabeculectomy 2 |

Habitual tobacco users formed 4.7% (5 patients) of total secondary glaucoma. 3 (2.8%) patients presented

with secondary glaucoma as a complication of retinal detachment surgery with silicon oil. Another group

was steroid induced glaucoma comprising of 2(1.8%) patient both of them had history of vernal catarrh and indiscriminate use of topical steroids.

All of the patients were managed conservatively at the initial stages. The conservative treatment included topical anti glaucoma drugs like topical B blockers, cholinergic agonists, carbonic anhydrase inhibitors topical as well as systemic, oral intravenous hyperosmotic agents, and prostaglandin analogues. Inflammatory glaucomas responded well to steroids and topical NSAIDS.

Yag laser iridotomies were tried where secondary angle closures were suspected in pseuduphakic and traumatic glaucoma group. Pupilloplasty, synechioysis and peripheral iridectomy were done in aphakic patients with pupillary block. Cyclocryopexy was tried in neovascular glaucoma. Trabeculectomy was indicated where conservative treatment failed or there was poor patient compliance. Cataract surgery with intraocular implants was done in lens induced glaucomas along with trabeculectomy where required. Those patients who presented with glaucoma as a complication of detachment surgery with silicon oil underwent trabeculectomies and silicon oil removal later on. Unfortunately removal of silicon oil resulted in hypotony in 2 patients.

Table 4 shows the IOP control with conservative treatment. It could be clearly seen that habitual tobacco users, hypertensive patients, diabetic patients and pseudophakic patients responded well to conservative treatment and where it failed surgical treatment was planned.

| Type of | Conservative | Patients with |
|-------------------|------------------|----------------|
| Glaucoma | treatment | controlled IOP |
| | (Total Patients) | n (%) |
| 1.Inflammatory | | |
| a.Pseudophakic | | |
| PC IOL | 6 | 5 (83.3) |
| AC IOL | 3 | 2 (66.6) |
| b.Chronic uveitis | 7 | 5 (71.4) |
| 2.Traumatic | 5 | 4 (80.0) |
| glaucoma | | |
| 3.Glaucoma with | 9 | 7 (77.7) |
| Diabetic eye | | |
| disease | | |
| 4.Hypertensive | 7 | 6 (85.7) |
| patients | | |

| Table 4. | IOP | control | after | conservative management |
|----------|-----|---------|-------|-------------------------|
|----------|-----|---------|-------|-------------------------|

| 5.Glaucoma in | 2 | 2 (100) |
|------------------|---|---------|
| habitual tobacco | | |
| users | | |

Table 5 shows IOP control of patients who underwent trabeculectomies either due to failure of conservative treatment or poor patient compliance. The table shows an overall success rate of almost 80% for trabeculectomy.

Table 6 shows the visual outcome of these patients. It is well depicted that most of the patients have a visual acuity ranging between 6/60 to 6/12 which is quite satisfactory. Reasons where visual out come was poor were late presentation, corneal pathologies like corneal oedema, endothelial decompensation, corneal opacities etc. and glaucomatous optic atrophy due to persistently raised IOP for a long time.

| Table | 5. | IOP | control | foll | lowing | trabecu | lectomy |
|-------|----|-----|---------|------|--------|---------|---------|
|-------|----|-----|---------|------|--------|---------|---------|

| Type of Glaucoma | Trabeculec tomies | No. of Patients with controlled IOP following Trabeculectomy n (%) |
|--|----------------------|--|
| 1.Inflammatory | | |
| a.Pseudophakia | | |
| • PC IOL | 13 | 11(84.6) |
| • AC IOL | 7 | 5(71.4) |
| b.Chronic anterior uveitis | 5 | 3(60) |
| 2.Traumatic | 5 | 4(80) |
| 3.Lens induced | | |
| Phcomorphic | 3 | 3(100) |
| Phacolytic | 1 | 1(100) |
| 4.Pseudoexfoliation | 6 | 5(83.3) |
| 5.Habitual tobacco users | 3 | 3(100) |
| 6.Retial detachment surgery with silicon oil | 3 | 1(33.3) |
| 7.Steroid induced glaucoma | 2 | 2(100) |

PC=Posterior chamber, AC=Anterior chamber, IOL=Intraocular lens

DISCUSSION

While the prevalence of morbidity and visual impairment due to primary open angle and angleclosure glaucomas have been fairly well established by population surveys in the west and, recently, in the developing world, the issue of blindness from secondary glaucomas has received little attention from most investigators. Individuals with secondary glaucoma tend to report promptly to the ophthalmologist since there is often marked reduction in visual acuity, apart from pain and ocular discomfort. As a consequence, these are largely selfreported².

Table 6. Visual outcome after the treatment

| Type of | Visual acuity | | | | |
|---|---------------|-----------|----------|--|--|
| glaucoma | PL/PR-FC | 6/60-6/18 | 6/12-6/6 | | |
| 1.Inflammatory | | | | | |
| a.Pseudophakia | | | | | |
| • PC IOL | 4 | 11 | 7 | | |
| AC IOL | 6 | 5 | | | |
| b.Aphakia | 2 | | | | |
| c.Chronic uveitis | 4 | 8` | | | |
| 2.Traumatic glaucoma | 9 | 3 | | | |
| 3.Glaucoma with diabetic eye disease | 4 | 4 | 1 | | |
| 4.Hypertensive patients | | 7 | | | |
| 5.Neovascular glaucoma • Diabetic • CRVO | 5 | | | | |
| 6.Lens induced | | | | | |
| • Phacomorphic | 2 | 3 | | | |
| • Phacolytic | 1 | 4 | | | |
| 7.Pseudoexfoliati on | | 6 | | | |
| 8.Habitual | | 3 | 2 | | |

| tobacco user | | | |
|--------------------------------|---|---|--|
| 9.R.D surgery with silicon oil | 3 | | |
| 10.Steroid induced | 1 | 1 | |

PL=Perception of light, PR=Projection of light, FC=Finger counting

Based on the WHO Blindness Data Bank, Thylefors and Negrel, in their world estimate of glaucoma blindness, found it was not possible to determine the number of blind from secondary glaucoma, although they estimated the world prevalence to be 2.7 million⁵.

Secondary open and closed angle glaucomas are an important cause of ocular morbidity and vision loss in our community. Secondary glaucoma occurs with acquired ocular diseases (pigment dispersion, pseudoexfoliation, intraocular infection, intraocular inflammation and retinal vascular disease), blunt anterior segment injury, intraocular surgery (especially corneal grafting and congenital cataract surgery) and topical corticosteroid use. The medical treatment of secondary glaucoma is different from that of primary open angle glaucoma and must be tailored for the individual patient. Awareness of patients at high risk should enable early detection and referral for appropriate management⁶.

Different studies carried out to determine the causes of secondary glaucoma depict different percentages of different causes depending upon the environments in which these studies are carried out and vary with different groups of patients. In a study conducted at Hayatabad Medical complex Peshawar in 2003 secondary glaucoma was found to be 36.14% of the total glaucomas. In the study mentioned secondary glaucoma associated with chronic uveitis was 0.48%, aphakia with pupillary block 2.16%, traumatic glaucoma 7.95%, neovascular glaucoma with diabetes 2.4%, and central retinal vein occlusion 2.89%, phacolytic glaucoma 4.1%, phacomorphic 3.6%, pseudoexfoliation 6.02%, and steroid induced 6.03%. Patients glaucoma was were treated conservatively on topical antiglaucoma treatment, yag laser iridotomies, cryopexy and trabeculectomy where other treatments failed with an overall success rate of 83.7 %. While in our study secondary glaucoma associated with chronic uveitis was 11.3%, aphakia with pupillary block 1.9%, traumatic glaucoma 11.3%,

neovascular glaucoma with diabetes 2.4% and central retinal vein occlusion 1.8%, phacolytic glaucoma 4.7%, phacomorphic 6.6%, pseudoexfoliation 5.6%, and steroid induced glaucoma was 1.8%. These results are somewhat similar to the results of above mentioned study. Patients were treated conservatively as well surgically like trabeculectomy, the over all success rate of trabeculectomy being 80%⁷.

Another study was conducted in New Delhi India from 1970-80 to describe various causative factors responsible for secondary glaucoma. This study shows that aphakic glaucoma and glaucoma associated with complications of mature cataract were responsible for nearly 50% of total secondary glaucoma. This percentage is very high as compared to our study (Aphakic glaucoma 1.8% and lens induced 11.3%), perhaps the cataract surgery techniques have now been improved a lot and surgeons are more skilled. Neovascular glaucoma was 9.6%, traumatic glaucoma 8.4%, glaucoma due to chronic uveitis 8.2% and steroid induced glaucoma 6.8% in the same study which is quite similar to our study⁸.

Another nationwide study conducted at Islamabad included 13 tertiary care hospitals reports that secondary glaucoma was 35% of the total glaucoma burden⁹.

In our study secondary glaucoma due to uveitis encountered 11.3% patients, while study conducted in Boston USA 9.6% patients had secondary glaucoma due to uveitis and chronic anterior uveitis being the most common entity¹⁰. Surgical management of secondary glaucoma after silicone oil injection for complex retinal detachment may achieve good IOP control. Patients who undergo silicone oil removal alone to control IOP are more likely to have persistent elevation of IOP and silicone oil removal and glaucoma surgery are more likely to have hypotony¹¹.

Another interesting group in our study was of habitual tobacco users forming 4.7% of the total secondary glaucoma patients. In a study conducted in the same department for tobacco related eye disease it was found that 16.9% male and 20.5% female patients had secondary glaucoma who were habitual tobacco users¹². Tobacco has been found to be one of the agents having ill effects on ocular circulation and intraocular pressure¹³. In another study it was found that smokers have raised mean intraocular pressure¹⁴.

Tobacco may affect outflow channels of Schelmm's canal which are to some extent dependent

on autonomic nervous control for outflow of aqueous. The trabecular meshwork is innervated by plexus of delicate axons that terminate without specialized endings within endothelium of of canal of Schlemm. Nerves originate from trigeminal and sympathetic nervous system¹⁵.

Another mechanism may be the toxic and ischemic effect causing vascular insufficiency at optic nerve head supplied by short posterior arterioles of Haller Zinn¹⁶.

CONCLUSIONS

It was concluded that causes of secondary glaucoma are diverse but mainly intraocular inflammation is responsible for these types of glaucomas. They can be well managed with conservative medical treatment but where it fails surgery is the only option left. It is suggested:

- Easily accessible and affordable cataract surgery services of high quality should be provided to prevent lens induced glaucoma and pseudophakic glaucoma.
- Early detection and good management of conditions associated with the potential for retinal ischaemia and neovascularisation such as
 - Good management of hypertension to reduce retinal vein occlusions
 - Good control of diabetes to prevent neovascular glaucoma
- Increased awareness among eye care professionals, the public and pharmacists of the dangers of indiscriminate use of topical (and systemic) steroids
- Awareness should be created to prevent ocular trauma and timely presentation for its treatment if one gets such injury.

Author's affiliation

Dr. Uzma Fasih Assistant Professor Eye Department Karachi Medical & Dental College Abbasi Shaheed Hospital Karachi.

Dr. M.S Fehmi Associate Professor Eye Department Karachi Medical & Dental College Abbasi Shaheed Hospital Karachi

Dr. Nisar Shaikh

Assistant Professor Eye Department Karachi Medical & Dental College Abbasi Shaheed Hospital Karachi

Dr. Arshad Shaikh Proessor & Head of Eye Department Karachi Medical & Dental College Abbasi Shaheed Hospital Karachi

REFERENCE

- Quigley HA. The number of people with glaucoma world wide. Br J Ophthalmol. 1996; 80: 389-93.
- Khan MD, Quraish ND, Khan MA. Facts about status of blindness in Pakistan Pak J Ophthalmol. 1999; 15: 15-9.
- 3. Krishnadas R, Ramakrishnan R. Secondary Glaucomas. The Tasks ahead. Community eye health. 2001; 14: 40-2.
- Pozzi SAP, Wahieg R, Roasen B, et al. Secondary glaucoma in Paraguay. Etiology and medicine Ophthalology. 1999; 96: 359-63.

- 5. **Jafers BL, et al.** World Health Organization PBC/94.40 Global data on Blindness an update.
- 6. **Anthony JH.** Secondary glaucoma clinical and Experimental Optometry. 2000; 83: 190-4.
- Baber FT, Saeed N, ZubairM, et al. Two year audit of glaucoma admitted patients in Hayatabad Medical Complex Peshawar. Pak J Ophthalmol. 2003: 19: 32-40.
- 8. Agarwal HC, Sood NN, Kalra BR, et al. Secondary glaucoma. Indian J Ophthalmol. 1982; 30: 121-4.
- Qureshi BM, Khan DM, Shah NM, et al. Glaucoma admissions and surgery in public sector tertiary care hospitals of Pakistan. Results of a national study. Ophthalmic epidemiology. 2006: 13: 115-9.
- Lloves MJ, Power JW, Rodriguez A, et al. Secondary glaucoma in patients with uveitis. Ophthalmologica. 1999; 213: 300-4.
- 11. **Donal L, Bundez MD, Katia E, et al.** Surgical Management of secondary glaucoma after pars plana vitrectomy and silicon oil injection for complex retinal detachment. Ophthalmology. 2001; 108; 1028-32.
- 12. Shaikh A, Alam P, Sami MS. Tobacco related eye diseases. Pak J Ophthalmol. 1999; 15: 113-6.
- 13. Krishna K. Tobacco and Intraocular Pressure. Online J Health and Allied Sciences. 2002; 0922-5997.
- 14. Lee AJ, Rchtchina E, Wang JJ, et al. Smoking effects intraocular pressure, findings of Blue Mountain Study. 2003; 12: 212.
- Newell FW. Anatomy and Embryology. In Ophthalmology Principles and concepts 7th ed. Mosby year book St. Louis. 1992; 3-70.
- 16. **Khaw PT.** The glaucomas. In Aids to Ophthalmology1st ed. Churchill Livingstone, London. 1989: 90-104.