Original Article

# Current profile of secondary glaucomas

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**Purpose:** To study the current profile of secondary glaucomas for their incidence and to identify risk factors.

**Materials and Methods:** In this retrospective chart review, 2997 patients newly diagnosed and referred with glaucoma to our tertiary glaucoma center in the year 2005 were included. Evaluation of all cases was done on the basis of a detailed history and recorded examination including vision, intraocular pressure (IOP), anterior segment examination, gonioscopy and fundus evaluation by glaucoma specialists. Demographic data, etiology of secondary glaucoma, and any other significant findings were noted.

**Results:** Of 2997 referred patients, 2650 had glaucoma or were glaucoma suspects. Of all glaucoma patients or glaucoma suspects, 579 patients (21.84%) had secondary glaucoma. Age distribution was as follows: 25% were between 0-20 years; 27% were between 21-40 years; 30% were between 41-60 years and 18% were >60 years. The male female ratio was 2.2. Frequent causes of secondary glaucoma were post - vitrectomy 14%, trauma 13%, corneo-iridic scar 12%, aphakia 11%, neovascular glaucoma 9%. Post-vitrectomy glaucoma eyes had vitreous substitutes in 83% cases of which 66% eyes had retained silicone oil for more than three months. Vision ≤20/200 was present in 63% eyes, 57% eyes had baseline IOP > 30 mm Hg. Of all traumatic glaucoma patients, 71% cases were <30 years of age. Fifty per cent had baseline IOP of >30 mm Hg and vision ≤20/200.

**Conclusions:** Most patients with secondary glaucoma have poor vision (≤20/200) with high IOP and advanced fundus changes at presentation.

**Key words:** Aphakic glaucoma, corneo-iridic scar, late presentation, post-vitrectomy glaucoma, secondary glaucoma, traumatic glaucoma

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According to the World Health Organization (WHO) global estimation in 2002,[1] more than 161 million people were visually impaired, of whom 124 million people had low vision and 37 million were blind worldwide. Refractive error, as a cause of visual impairment was excluded. It was also estimated that up to 75% of all blindness is avoidable. Glaucoma is the second leading cause of blindness globally as well as in most regions according to the WHO survey 2002. It accounts for 12.3% of global blindness (Vision <20/200 in better eye).

Most primary glaucomas are managed by early diagnosis and treatment and secondary glaucomas differ from primary by the fact that, if the primary pathology is treated properly and the possibility of secondary glaucoma is kept in mind, glaucomatous damage can be easily prevented. Ophthalmologists need to be careful in this regard while treating primary pathologies which have high chance of developing glaucoma secondarily.

This chart review was undertaken to find the common causes, demographics and clinical characteristics of various secondary glaucomas.

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## Materials and Methods

In this retrospective chart review, 2997 patients referred with glaucoma to our tertiary glaucoma center in the year 2005 were evaluated by glaucoma specialists. The evaluation included a detailed history and detailed examination performed including vision, anterior segment examination, intraocular pressure (IOP) by Goldman applanation tonometry, and gonioscopy. Stereoscopic fundus evaluation was performed wherever possible. Visual fields were available in only a few patients as the rest had very poor visual acuity, so they could not be evaluated.

Secondary glaucoma was diagnosed when the following criteria were met: a positive history and ocular findings of pathologies such as trauma, previous surgery, neovascularization, inflammation, or any other abnormal ocular or systemic findings that could have caused prior or current IOP elevation. Patients with unilateral glaucoma were included as secondary glaucoma only if the other eye had no evidence or family history of a primary glaucoma. The other eye had IOP, gonioscopy and fundus evaluation and if required diurnal variation and visual field to exclude primary glaucoma.

Secondary glaucoma was diagnosed in the presence of chronically raised IOP with or without glaucomatous optic neuropathy. The main outcome measures were demographic data, the etiology of secondary glaucoma, visual acuity, IOP, manipulation or indentation gonioscopy, glaucomatous optic neuropathy and any other positive findings.

Data were analyzed using SPSS 10 statistical software (PC version, USA).

## Results

Two thousand nine hundred and ninety seven patients were referred to the glaucoma service in 2005 of which 347 were diagnosed to have no glaucoma by our specialists. Of the 2650 patients diagnosed as glaucoma or glaucoma suspects, 579 patients (21.84%) had secondary glaucoma.

In patients having secondary glaucoma, male female ratio was 2.2 and the age distribution was as follows: 25% were between 0-20 years; 27% were between 21-40 years; 30% were between 41-60 years and 18% were >60 years. Common causes of secondary glaucoma were post vitrectomy (14%), trauma (13%), corneal pathology (12%), aphakia (11%), neovascular glaucoma (10%), pseudophakia (10%), steroid-induced glaucoma (8%), uveitic glaucoma (8%), and miscellaneous causes (14%). Miscellaneous causes included lens-induced glaucoma, post penetrating keratoplasty glaucoma, glaucoma secondary to tumor, pseudoexfoliation syndrome, pigment dispersion glaucoma, glaucoma secondary to retinopathy of prematurity, aniridia, iridocorneal endothelial syndrome and chemical injury. The etiology of secondary glaucoma in the year 2005 was compared to the available past data of 10 years from 1970-80 from the same center [Table 1].

The distribution of glaucoma in different age groups is shown in Table 2. Young patients below 20 years of age had trauma as the most common cause of secondary glaucoma, whereas those between 21-40 years of age had glaucoma following vitreoretinal surgery with or without vitreous substitute as the leading cause. Between 41-60 years of age, neovascular glaucoma followed by glaucoma secondary to corneal pathology was common. In the older population above 60 years of age, pseudophakic glaucoma was the commonest cause, followed by aphakic and neovascular glaucoma. The sex distribution of various glaucomas is shown in Table 3, with male preponderance in all, except uveitic glaucoma.

We performed further analysis to look for distribution of secondary juvenile glaucomas (below 35 years) and compared with 164 cases in 1984 and 100 cases of juvenile secondary glaucoma in 1988 presented to the same center.[2] Secondary juvenile glaucomas constituted 50% of secondary glaucomas of all ages (277 cases). Common causes of secondary glaucoma were trauma 28%, post-vitrectomy 15%, corneal pathology 12%, steroid-induced 10% [Table 4].

We further analyzed each type of glaucoma for their clinical characteristics and risk factors, summarized in Table 3.

Glaucoma following vitreoretinal surgery accounted for 14% of secondary glaucomas. It was the most common etiology in the 21-60 years age group. Males were more frequently affected (M:F ratio = 4:1). A vitreous substitute was used in 83% cases, of which 73% cases had silicone oil. Sixty-six per cent eyes had retained silicone oil (1,000 centistokes viscosity) for >three months. Other associated risk factors found were trauma in 23%, myopia in 9% and others such as diabetes, vascular occlusion, and steroid use. A vision of ≤20/200 was present in 63% eyes and 57% eyes had an IOP ≥ 30 mm Hg after vitrectomy. Glaucomatous optic neuropathy (CD ratio ≥ 0.7) was found in 65% cases and 42% cases had CD ratio ≥ 0.9.

Traumatic glaucoma was the second most common cause of secondary glaucoma in all age groups and the leading cause

**Table 1: Etiology of different secondary glaucomas in the year 2005 compared to previous result from the years 1970-80**

| Causes of secondary glaucomas | Year 2005 (Number) | Year 2005 (%) | Year 1970-80 (%) |
|---|---|---|---|
| Post-vitrectomy (with or without vitreous substitute) | 82 | 14 | - |
| Post-traumatic | 76 | 13 | 8.4 |
| Corneo-iridic scar | 70 | 12 | 12.2 |
| Aphakic | 64 | 11 | 37.7 |
| Neovascular | 54 | 9 | 9.6 |
| Pseudophakic | 58 | 10 | - |
| Steroid-induced | 47 | 8 | 6.8 |
| Uveitic | 47 | 8 | 8.2 |
| Miscellaneous | 82 | 14 | - |

**Table 2: Age distribution of secondary glaucomas**

| | Traumatic (%) | Post-vitrectomy (%) | Corneoiridic scar (%) | Aphakic (%) | Neovascular (%) | Pseudophakic (%) | Steroid (%) | Uveitic (%) | Post-PK (%) | Others |
|---|---|---|---|---|---|---|---|---|---|---|
| 0-20 yrs (*n* = 146) | 26 | 13 | 13 | 13 | 4 | 4 | 8 | 3 | 5 | 11 |
| 21-40 yrs (*n* = 158) | 15 | 19 | 13 | 3 | 5 | 5 | 13 | 9 | 7 | 11 |
| 41-60 yrs (*n* = 176) | 7 | 12 | 14 | 4 | 17 | 10 | 7 | 10 | 10 | 9 |
| >61 yrs (*n* = 105) | 3 | 15 | 7 | 18 | 17 | 25 | 0 | 4 | 8 | 3 |

**Table 3: Characteristics of different secondary glaucomas**

| Causes of secondary glaucomas | Mean age (years) | M:F | BCVA ≤ 20/200 (%) | Baseline IOP ≥ 30 (%) | GON (%) |
|---|---|---|---|---|---|
| Post-vitrectomy | 38.1 | 4:1 | 63 | 57 | 65 |
| Post-traumatic | 25.6 | 9:1 | 50 | 50 | 50 |
| Corneo-iridic scar | 36.1 | 1.8:1 | 100 | - | - |
| Neovascular | 50.7 | 2.3:1 | 95 | 78 | 75 |
| Aphakic | 42.8 | 1.8:1 | 37 | 45 | 58 |
| Pseudophakic | 53.5 | 1.3:1 | 50 | 60 | 50 |
| Steroid | 31.1 | 3.2:1 | 20 | 50 | 50 |
| Uveitic | 41.1 | 0.8:1 | 70 | 60 | 40 |
| Post-PK | 41.8 | 1.2:1 | 90 | - | - |

**Table 4: Overall distribution of juvenile secondary glaucomas compared with previous studies from the same center**

| Causes | Year 2005 (No.) | Year 2005 (%) (*n* = 277) | Year 2005 (Male) | Year 2005 (Female) | Year 1984 (%) (*n* = 164) | Year 1988 (%) (*n* = 100) |
|---|---|---|---|---|---|---|
| Total | 277 | | 168 | 83 | 164 | 100 |
| Traumatic | 77 | 28 | 68 | 9 | 22.6 | 36 |
| Post-vitrectomy | 41 | 15 | 39 | 2 | - | - |
| Corneo-iridic scar | 34 | 12 | 24 | 10 | lesser | 8 |
| Steroid | 27 | 10 | 19 | 8 | 23.8 | 11 |
| Aphakia | 23 | 8.3 | 16 | 7 | 11.6 | 4 |
| Uveitic | 13 | 5 | 7 | 6 | 20.7 | 9 |
| Post-PK | 12 | 4 | 5 | 7 | - | - |
| Pseudophakic | 12 | 4 | 9 | 3 | - | - |
| NVG | 9 | 3 | 4 | 5 | 4.9 | 3 |
| Miscellaneous | 29 | 10 | 22 | 7 | - | - |

in 0-20 years age group. Of all traumatic glaucoma patients, 71% were below 30 years of age, and male 90%. Blunt trauma was the mode of injury in 85% of cases, play-related - cricket ball, tennis ball, gilli-danda, hockey stick, bamboo stick and stone in 30% cases; fire cracker injury in 20% cases and 50% were work-related, assaults or accidental injury. Gonioscopy could be performed in 78% of eyes, with 66% eyes showing angle recession. In eyes having angle recession, two or more quadrants were involved in 87% cases. Other features of trauma like sphincter tear, hyphema, iridodialysis, subluxation, dislocation, vitreous hemorrhage, retinal detachment, and cataract could be seen in various combinations in about 95% cases. Fifty per cent of traumatic glaucomas had an IOP of ≥30 mm Hg and 56% had a vision ≤20/200. Optic nerve head could be assessed in 50% of eyes, of which 50% showed glaucomatous optic neuropathy (GON). Protective glasses were not worn by the individuals at the time of vulnerable play activities.

Glaucoma secondary to corneal pathologies was also frequent. Fifty per cent of these cases were less than 40 years of age. Bilateral opacities were present in one-fifth of the cases. The most common etiology was a healed corneal ulcer (50%); 10% were post traumatic. All eyes had a vision of <20/200, and fundus evaluation was not possible due to the presence of the opacity.

Aphakic glaucoma affected the two extremes of age. Twenty-five per cent of cases were under 15 years of age, 85% were after congenital cataract and 15% were post traumatic. Fifty-eight per cent cases were over 50 years of age after cataract extraction. Forty per cent cases of aphakic glaucomas were bilateral. Forty-five per cent of aphakic glaucomas had an IOP of ≥30 mm Hg and 37% had a vision of ≤20/200. Glaucomatous optic neuropathy (CD ratio ≥0.7) was found in 58% cases and 26% of cases had CD ratio ≥ 0.9.

Neovascular glaucoma constituted 9% cases of all secondary glaucomas. Common causes of neovascular glaucoma were central retinal vein occlusion (22%), proliferative diabetic retinopathy (22%) and vasculitis (22%). Ninety-five per cent eyes had vision <20/200. An IOP > 30 mm was present in 78% cases. Optic nerve head could be assessed in 38% eyes of which 75% showed GON (CD ratio ≥ 0.7) and 50% of cases had CD ratio ≥ 0.9. In this group we saw some of the end stage chronic primary angle closure glaucoma cases (10%) presenting with neovascular glaucoma.

Steroid-induced glaucoma was present in 8% of all cases. Of all cases of steroid-induced cases, 50% were avoidable and were due to the use of treatment for ocular allergies or postoperatively or drops used over the counter without prescription. The rest were due to treatment for systemic conditions such as sarcoidosis, scleroderma, primary sclerosing cholangitis, polymyositis, ulcerative colitis, nephritic syndrome and uveitis. These could also have been avoidable if the concerned physicians treating them for these systemic diseases were educated on the possibility of steroid-induced IOP elevation and glaucoma and had either

warned the patients or had referred them to ophthalmologists for interventions. Bilateral involvement was seen in 68% of cases. A vision of ≤20/200 was present in 20% eyes, 50% eyes had an IOP ≥ 30 mm Hg. Glaucomatous optic neuropathy (CD ratio ≥ 0.7) was found in 50% cases.

Uveitic glaucoma followed attacks of anterior uveitis in 90% cases, and the rest were seen in panuveitis. Bilateral involvement was seen in 50% of cases. Vision of ≤20/200 was present in 70% eyes, 60% eyes had an IOP ≥ 30 mm Hg reaching up to 59 mm of Hg. Optic nerve head could be assessed in 45% eyes of which 40% showed GON.

Pseudophakic glaucoma affected the older population, 80% of patients being above 40 years of age. Most cases were unilateral (93%). A large number of eyes (37%) had either anterior chamber intraocular lens (IOL) or a posterior chamber IOL placed in the anterior chamber or a pupillary capture of IOL. Ninety per cent cases had undergone a complicated cataract surgery. Sixty per cent of pseudophakic glaucomas had an IOP of ≥30 mm Hg and 50% had vision ≤20/200. Optic nerve could be evaluated in 50% cases of which 50% showed GON (CD ratio ≥ 0.7).

## Discussion

In this institution-based retrospective chart review, secondary glaucomas were seen in 22.07% of all newly diagnosed glaucomas. We have ascertained a demographic and clinical profile of all secondary glaucomas over a year. It is one of the largest series of secondary glaucoma studied.

Secondary glaucoma results from numerous ocular or systemic disorders and shows a poor IOP control with ocular hypotensive agents or filtering surgery in its late stages. Thus, early detection is important to maximize the chance of a therapeutic response. The causative lesion overshadows any symptoms or signs of the secondary glaucoma, so that the diagnosis is often missed and almost invariably delayed. This, we feel, is largely due to the lack of knowledge regarding the relative frequency of the various causes of secondary glaucoma. Since it is secondary to other ocular or systemic pathology, primary prevention is possible by keeping in mind risk factors associated with the development of glaucoma.

Despite its public health significance, there is limited data available on the prevalence of secondary glaucoma and the possible risk factors for secondary glaucoma. The population-based Aravind comprehensive eye survey from south India[3] reported a 0.7% incidence of secondary glaucomas where the total prevalence of glaucoma was 2.6%, i.e. a third of all glaucoma cases. The total number of cases in this study was very small for any further analysis. In another population-based study in the Japanese population,[4] secondary glaucoma and primary angle closure glaucoma had an almost equal incidence of 0.6% and 0.5% respectively among a total incidence of glaucoma of 5%. This amounts to 10% of all cases of glaucoma.

A similar study in the past from the same center has reported secondary glaucoma cases of 10 years (1970-80) from the retrospective review chart.[5] Study from north India reports a 6.72% diagnosis of secondary glaucoma out of all glaucoma referrals in a five-year hospital-based retrospective analysis (1995- 99).[6] Other studies from Finland[7] and Pakistan[8] found an incidence of 33% and 35% respectively.

Secondary glaucomas had very poor vision of <20/200 at presentation in almost all types except steroid-induced and aphakic glaucomas. An IOP of ≥30 was present in ≥50% cases in almost all types except the aphakic group. Glaucomatous optic neuropathy (cup disc ratio ≥ 0.7) was found in ≥50% of the cases where fundus could be visualized. Such figures should alert the ophthalmologists treating the primary pathology regarding the need for early initiation of anti-glaucoma medications.

There has been an increase in the total number of cases of secondary glaucoma referred to our center over time. Total number of cases were 1065 in the decade from 1970-1980,[5] while in one year (year 2005) the number was 585. In this work, post-vitrectomy glaucoma was the most common cause among the secondary glaucomas in the year 2005. Thirty years earlier the commonest cause was aphakic glaucoma, which has dramatically reduced with advancements in cataract surgery. Aphakic glaucoma and glaucoma secondary to senile cataract accounted for nearly 50% of all causes of secondary glaucoma. Lens-induced glaucoma has also shown a decline due to greater awareness in the population about cataract extraction and better surgical facilities. Traumatic and post-vitrectomy glaucoma have shown a dramatic increase in number in the current study. Traumatic glaucoma has increased in incidence from 8.4% to 13%. Other causes maintain an almost similar prevalence as in the past.

In the age group of below 20 years, trauma remains the most common cause, as was seen earlier in a study of secondary juvenile glaucomas.[2] This study shows the emergence of post-vitrectomy glaucoma with or without vitreous substitute as the second most frequent cause of juvenile secondary glaucoma. Juvenile aphakic glaucoma has increased in incidence from 4% to 8.3% this time.

As the most common causes were post-vitrectomy and trauma which were more common in males, they accounted for a higher incidence of secondary glaucoma in males. Also, in India, men are more likely to reach a tertiary center than women.

In post-vitrectomy glaucoma, silicone oil seemed to be a very important factor, especially when retained for more than three months. Honavar *et al*,[9] in their study on glaucoma after vitrectomy in Indian patients has shown silicone-induced glaucoma in 70% cases of all glaucoma. Other causes were preexisting glaucoma, neovascular glaucoma and traumatic glaucoma (5%) in their series. In our cases too silicone oil was used in a majority of cases and other factors were trauma, myopia, diabetes. Silicone oil-filled eyes need to have frequent IOP measurements, and oil should be removed as soon as the tamponade effect is no longer required.

Traumatic glaucoma was associated with angle recession and other features of trauma in 95% cases. Ellong *et al*,[10] have reported a incidence of 4.2% compared to 2.9% in our study, of traumatic glaucoma out of all glaucoma cases. In their study too, monocular blindness was seen in 61.9%, mean IOP was 36.9 ± 13.8 mm Hg and mean recorded C:D ratio was 0.8 ± 0.2. Irido-corneal angle recession was seen in 61.9%. Damage to the iris or lens, vitreous hemorrhage, and inflammation on baseline examination has been shown to be associated with a significantly greater risk of developing glaucoma after penetrating ocular involvement.[11,12] Penetrating injuries were followed by a secondary glaucoma if an adherent leucoma and/

or evidence of lenticular damage or displacement were seen.

In corneal pathologies secondary to infective etiology there may be inflammation and a higher incidence of rise of IOP. However, accurate measurement of IOP is difficult, and pneumotonometry if available, may be of value in these cases. Glaucoma secondary to an improperly managed or non-responsive bacterial or fungal ulcer with subsequent formation of adherent leucoma was common.

It is important to mention that steroid-induced response depends on the duration of therapy, type of steroids used, as well as genetic influence of a person. Most of the cases included in this study had GON due to long-term use of systemic and/or local steroids, indicating their late presentation to this hospital. In this respect, general physicians should be educated regarding the side-effects of systemic steroids and the importance of regular follow-up starting from initiation of steroid therapy with an ophthalmologist. Over the counter issue of topical corticosterids should be strongly discouraged in this regard.

This analysis of secondary glaucomas helps identify the five common primary pathologies as trauma, post vitrectomy, adherent leukomas, pseudophakia and aphakia.

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