

Clinical Profile of Secondary Glaucoma in Tertiary Care Centre in Western Odisha**Kanhei Charan Tudu¹, Bikash Ranjan Nayak², Pramod Kumar Sharma³, Sharmistha Behera⁴**¹Associate Professor & HOD, Department of Ophthalmology, VSSIMSAR, Burla, Odisha, India²Senior Resident, Department of Ophthalmology, VSSIMSAR, Burla, Odisha, India³Assistant Professor, Department of Ophthalmology, VSSIMSAR, Burla, Odisha, India⁴Associate Professor, Department of Ophthalmology, VSSIMSAR, Burla, Odisha, India

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Abstract:**Background:** Secondary glaucoma is caused by underlying systemic or ocular disorders. The purpose of this investigation was to examine the clinical profile of patients with secondary glaucoma and the different management strategies for these secondary glaucomas in a hospital in western Odisha.**Methods:** This two-year cross-sectional observational study was conducted between October 2020 and November 2022. After receiving informed consent, 57 cases of secondary glaucoma affecting outpatients were included in the study. Evaluation consisted of BCVA, IOP, slit lamp examination, gonioscopy, and medical management.**Results:** Seventeen percent of the 322 patients diagnosed with glaucoma or suspected of having glaucoma had secondary glaucoma. The most prevalent cause was lens-induced glaucoma (25 cases, 43%), followed by post-traumatic glaucoma (10 cases, 18%), pseudophakic (6 cases, 11%), aphakic (5), steroid-induced (4), uveitic (4), and neovascular glaucoma (3 cases, 5%).**Conclusion:** The causes of secondary glaucoma are diverse, and the majority of patients present late with impaired vision, elevated intraocular pressure, and even glaucomatous optic atrophy. In order to prevent secondary glaucoma-related blindness, it is essential to identify and address the causes promptly.**Keywords:** Secondary Glaucoma, Lens Induced Glaucoma, Phacomorphic Glaucoma, Uveitic Glaucoma.This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.**Introduction**

Secondary glaucoma is a group of disorders in which elevated intraocular pressure is caused by a primary ocular or systemic disease. This could result in serious blindness or visual impairment. The global average incidence of secondary glaucoma is 18 per cent of that of primary open-angle glaucoma [1]. Individuals with secondary glaucoma tend to visit an ophthalmologist as soon as possible because, in addition to pain, there is frequently a marked reduction in visual acuity [2, 3]. In secondary glaucomas; there is a blatant obstruction of aqueous outflow with an identifiable underlying cause. There are numerous causes for the development of secondary glaucomas. Causes of elevated intraocular pressure [4] include ocular disorders, systemic diseases, and adverse drug reactions. If the primary underlying cause of the elevated intraocular pressure in secondary glaucoma is treated (if possible), the pressure may normalise [5]. In advanced untreated cases where the optic nerve has been completely injured, however, vision cannot be restored. For an improved visual outcome, a patient with secondary glaucoma needs prompt diagnosis

and treatment [6-10]. On gonioscopic examination, all of these patients had either a secondary open-angle or a secondary closed-angle [8]. This study's objectives were to investigate the common causes, clinical profile, and various types of secondary glaucoma, as well as to assess the efficacy of various management strategies for the various types of secondary glaucoma.

Methods

This cross-sectional observational study was conducted between October 2020 and November 2022 at the Department of Ophthalmology, VSSIMSAR, Burla, Sambalpur, and Odisha. From the patients who visited the outpatient department, 57 cases of secondary glaucoma were admitted, and their informed consent was obtained. Neovascular glaucoma was the least common cause of secondary glaucoma, accounting for only 9% of cases.

Inclusion Criteria: All patients with secondary glaucoma attending ophthalmology OPD of our institution were included in this study.

Exclusion Criteria: All patients with primary glaucomas and developmental glaucomas and those who did not give informed consent were excluded from this study.

Patient Evaluation

After clinical diagnosis, all 57 patients were admitted, and a thorough medical history and physical examination were conducted. Slit lamp examination, routine investigations such as blood pressure measurements, urine sugar, FBS, PPBS estimation, orbit and brain CT scan, and B-scan were performed on the affected eye. Other standard procedures, including lacrimal syringing, A scan

biometry, urine sugar, and blood pressure measurement, were also performed.

Statistical Analysis

The information was compiled on a Microsoft Excel spread sheet. Whenever applicable, version 23 of SPSS was used to analyse qualitative data with the chi-square test and quantitative data with the independent t-test. A value below 0.05 was considered statistically significant.

Results

A total of 57 cases with secondary glaucoma were admitted for treatment and studied in detail.

Table 1: Prevalence of various forms of secondary glaucoma

Types secondary glaucoma	Number	Percent
Lens induced glaucoma	25	43
Post-traumatic glaucoma	10	18
Pseudophakic glaucoma	6	11
Aphakic glaucoma	5	9
Steroid-induced glaucoma	4	7
Uvetic glaucoma	4	7
Neovascular glaucoma	3	5
Total	57	100

There were 57 cases diagnosed with secondary glaucoma out of a total of 322 admitted for glaucoma, making the prevalence of secondary glaucoma among admitted glaucoma cases 17.70%. Table 1 reveals that lens-induced glaucoma accounted for 43%, or 25 out of 57, of all secondary glaucoma, while neovascular glaucoma accounted for 5%, or 3 out of 57.

Table 2: Association with systemic disease in participants

Diseases	Cases(57)	Percentages (%) (100)
Diabetes mellitus	16	29
Hypertension	20	35
Tuberculosis	5	8
Syphilis	1	2
No association	15	26

Systemic diseases such as Diabetes mellitus and hypertension were more prevalent among the participants in our study, accounting for 29% and 35%, respectively. The prevalence of tuberculosis and syphilis was discovered to be 5% and 2%, respectively, among the study population.

Table 3: Incidence of presenting symptoms among participants

Presenting symptoms	No of cases	Percentage (%)
Diminution of vision	57	100%
Redness of eye	56	98%
Eye pain	56	98%
Watering	52	91%
Photophobia	45	79%
Haloes	10	18%
Blurring attack	15	26%

In 100% of cases, there was a reduction in vision, in 98% of cases there was eye redness and pain, in 91% of cases there was eye watering, in 79% of cases there was photophobia, followed by haloes in 18% of cases and distortion in 26% of cases.

Table 4: Complications seen due to various treatment modalities

Complications	No of cases (N=31)	Percentage (%)
PCR with VL	15	48
Striate keratitis	22	70
Uveitis	11	35
Hyphaema	4	13
Shallow AC	12	38
Chemosis	2	6
Iritis	2	6

In 15 cases, intracapsular complications such as posterior capsular tear and vitreous loss were observed. Striate keratitis was identified in 22 cases, uveitis in 11 cases, hyphaema in 4 cases, inadequate anterior chamber depth in 12 cases, chemosis in 2 cases, and iritis in 2 cases.

Discussion

From October 2020 to November 2022, the current investigation was conducted on 57 patients at the Department of Ophthalmology, VIMSAR, and Burla.

The sample size of Sherpa et al.'s investigation was comparable to that of the present study [11]. Azam et al. conducted a study with a larger sample size (80 samples) than mine [12]. Bendel et al. [13] had a relatively limited sample size (27 cases). The prevalence of secondary glaucoma among admitted glaucoma patients was found to be 17.70% in my study. Sherpa et al. [11] discovered an almost identical prevalence of secondary glaucoma, 22.07%.

The most prevalent type (43%) of secondary glaucoma was found to be lens-induced glaucoma. Thus, my research is consistent with prior literature [14–16]. The phacomorphic type of lens-induced glaucoma (62%) was more prevalent than the phacolytic type. Sitoula et al. [17] discovered a similar prevalence of phacomorphic type (64%).

Diabetes and hypertension are the most prevalent systemic diseases among our study participants, accounting for 29% and 35%, respectively. Stein et al. [18] discovered that diabetic patients have a 35% risk and hypertensive patients have a 17% risk of developing POAG, which is consistent with my study. In this study, the majority of cases (76%) occurred in patients older than 60 years. In accordance with previous research, the incidence was highest in the 61-70 age group (43.1% [19], 60% [20], 50% [21], 62.50% [22], and 52.0% [23]). This is due to the fact that LIGs are a consequence of ageing.

In all cases, ciliary congestion, corneal edoema, and mid-dilated pupils were observed. A shallow anterior chamber is observed in all cases of phacomorphic glaucoma and two cases of lens dislocation, totaling 65.5%. Other investigations

[24-26] have found that phacomorphic glaucoma is associated with a shallow anterior chamber. In this series, cases of phacolytic glaucoma exhibited either a profound anterior chamber or a normal anterior chamber depth, as noted in numerous other studies [27-29].

Surgical management

In the preponderance of 41 (64.1%) cases, SICS with PCIOL implantation was the chosen treatment. In 16 (25%) instances where intraocular pressure remained above 25 mmHg despite medical treatment, combined surgery was performed.

According to multiple studies, ECCE with PCIOL implantation in LIG is an effective and safe procedure for obtaining visual recovery and maintaining intraocular pressure below 20 mm Hg on early post-operative days [30-33]. For the treatment of LIG, manual small-incision cataract extraction with PCIOL implantation is currently preferred.

Post-operative BCVA

In this research, BCVA 6/12 or better was gained in 43.8% of cases at last follow-up, which is slightly higher than Damodar Pradhan et al's study series (31.40%) but lower than Sharanabasamma and K Vaibhav (54%) [13,9]. In a study by Chandrasekhar G et al. [19], it was found that 44% of participants had vision superior than 6/12.

In the present study, 38.5% of phacomorphic glaucomas and 59.1% of phacolytic glaucomas recovered to a visual acuity of 6/12 or higher. Several investigations [19, 23, 27, 31] supported this conclusion. 57 per cent of phacomorphic glaucomas and 61 per cent of phacolytic glaucomas attained postoperative corrected visual acuity of 6/12 or higher, according to a study by Prajna N V [36] and colleagues.

Conclusion

The conclusion that can be derived from this study is that there are numerous causes of secondary glaucoma, but intraocular inflammation is primarily responsible for these types of glaucoma. To avoid ocular morbidity, a thorough history, clinical examination, and, if necessary, anterior segment imagings such as UBM are required to identify the

specific cause, if possible, and formulate a treatment plan.

In our region, lens-induced glaucoma remains the most prevalent cause of secondary glaucoma. Additionally, neovascular, steroid use, trauma, and post-vitrectomy are common causes. The implementation of educational campaigns aimed at preventing ocular injuries has the potential to mitigate the increasing prevalence of traumatic glaucoma. Furthermore, individuals who are at a heightened risk of developing this condition are advised against the misuse of steroids.

In conclusion, the expeditious detection of common aetiologies of secondary glaucoma has the potential to mitigate the profound visual impairment and blindness documented in the present study.

References

1. Khurana AK, Principles and practice of glaucoma, first edition. Noida UP, India: CBS Publisher and distributors; 2015.
2. Government of India, National Survey on Blindness and Visual Outcome after cataract Surgery, 2001-2002, National Programme for Control of Blindness, Ministry of Health, Government of India, New Delhi, India, 2002; 77.
3. Cook C, Foster P. Epidemiology of glaucoma: what's new? *Can Ophthalmol.* 2012 Jun; 17(3): 223-6.
4. Quigley H. The number of people with glaucoma worldwide. *Br J Ophthalmol.* 1996; 80:389-393.
5. Gadia R, Sihota R, Dada T, Gupta V. Current profile of secondary glaucomas. *Indian J Ophthalmol.* 2008; 56(4):285-289.
6. Gurung J, Sitoula RP, Singh AK. Profile of Secondary Glaucoma in a Tertiary Eye Hospital of Eastern Nepal. *Nepal J Ophthalmol.* 2021 Jan; 13(25):98-103.
7. Abdull MM, Chandler C, Gilbert C. Glaucoma, The silent thief of sight": patients' perspectives and health seeking behavior in Bauchi, northern Nigeria. *BMC ophthalmology.* 2016 Dec 1; 16 (1): 44.
8. Gadia R, Sihota R, Dada T, et al (2008). Current profile of secondary glaucomas. *Indian J Ophthalmol.* 2008;56: 285-289.
9. Khurana AK, Khurana Indu, Anatomy and Physiology of Eye. 3rd edition. Noida UP, India: CBS Publisher and distributors. 2017; 72-76
10. Soe Ni Ni, J. Tian, Pina Marziliano, Hong-Tym Wong, Anterior Chamber Angle Shape Analysis and Classification of Glaucoma in SS-OCT Images, *Journal of Ophthalmology*, vol. 2014, Article ID 942367, 12 pages, 2014.
11. Sherpa D and Pokhrel S. Current Pattern of Secondary Glaucoma. *J of Chitwan Medical College;* 2017;7(21): 21-24.
12. Shua Azam., et al. "Causes of Secondary Glaucoma among Patients presenting in Glaucoma Clinic at Al-Ibrahim Eye Hospital, Karachi". *Acta Scientific Ophthalmology* 4.5 (2021): 15-18.
13. Bendel RE, Patterson MT. Observational report: Improved outcomes of trans scleral cyclo photo coagulation for glaucoma patients. *Medicine (Baltimore).* 2017 Jun; 96(23):e6946.
14. Allingham Glaucoma. RR, Sixth Damji edition. KF, Boston, Freedman Massachusetts, S, Moroi SE, USA: Rhee Wolter DJ. Shields Kluwer/ Lippincott textbook of Williams & Wilkins; 2011;72-74.
15. Inomata H, Tawara A. Anterior and posterior parts of human trabecular meshwork. *Jpn J Ophthalmol.*1984;28(4):339- 348
16. Borges- Giampani AS, Junior JG. Anatomy of ciliary body, ciliary processes, anterior chamber angle and collector vessels. *Intech Open;* 2013 Apr 17:9.
17. Sitoula RP, Sarkar I, Nayak D, et al. Lens induced glaucoma: An experience in a tertiary eye care center in eastern Nepal. *Nepal J Ophthalmol;* 2016;8(16): 161-166.
18. Stein JD, Kim DS, Niziol LM, et al. Differences in rates of glaucoma among Asian Americans and other racial groups and various Asian ethnic groups. *Ophthalmology.* 2011; 118:1031-1037.
19. Nanwani D, Dev S, Shilpa N, et al. Profile of secondary glaucoma cases in a tertiary eye care centre. *J of Dental and Medical Sciences;* 2015;14(12):53-58.
20. Rao VP, Bandaru S. a clinical study on lens induced glaucoma and its visual outcome in patients attending government general hospital, kadapa. *International Journal of Medical and Biomedical Studies.* 2019 Oct 28; 3(10).
21. V Sree Kumar, E. Satya Narayana Murthy, B. Preethi. Clinical study of visual prognosis in lens induced glaucoma. *International Journal of Contemporary Medical Research.* 2018; 5(3): C6-C8.
22. Jarwal PN. Clinical study of lens-induced glaucoma at community health center in India. *TNOA J Ophthalmic Sci Res.* 2020; 58: 162-8.
23. Mandal AK, Gothwal VK. Intraocular pressure control and visual outcome in patients with phacolytic glaucoma managed by extracapsular cataract extraction with or without posterior chamber intraocular lens implantation. *Ophthalmic Surg Lasers.* 1998Nov; 29(11): 880-9.
24. Grierson I, Howes RC. Age-related depletion of the cell population in the human trabecular meshwork. *Eye (Lond)* 1987; Pt2):204-2

25. Buller C, Johnson DH, Tschumper RC. Human trabecular meshwork phagocytosis. Observations in an organ culture. Invest Ophthalmol Vis Sci. 1990; 31:2156-2163.
26. Rohen JW. Why is intraocular pressure elevated in chronic simple glaucoma? Anatomical considerations. Ophthalmology 1983;90:758-765
27. Gong IL Y, Trinkaus-Randall V, Freddo T F. Ultra structural immune cytochemical localization of elastin in normal human trabecular meshwork. Curr Eye Res. 1989; 8:1071-1082.
28. Grant WM. Experimental aqueous perfusion in enucleated human eyes. Arch Ophthalmol. 1963; 69: 783- 801.
29. Grant WM. Further studies on facility of flow through the trabecular meshwork. AMA Arch Ophthalmol. 1958; 60: 523- 533.
30. Overby DR, Stamer WD, Johnson M. The changing paradigm of outflow resistance generation: towards synergistic models of the JCT and inner wall endothelium. Exp Eye Res. 2009; 88: 656- 670.
31. Keller KE, Aga M, Bradley JM, Kelley MJ, Acott TS. Extracellular matrix turnover and outflow resistance. Exp Eye Res. 2009; 88: 676-682.
32. Rohen J W, Lutjen- Drecoll E, Flugel C, Meyer M, Grierson I. Ultrastructure of the trabecular meshwork in untreated cases of primary open-angle glaucoma (POAG). Exp Eye Res. 1993; 56: 683-692.
33. Rohen J W, Linner E, Witmer R. Electron microscopic studies on the trabecular meshwork in two cases of corticosteroid-glaucoma. Exp Eye Res. 1973; 17: 19- 31.
34. Lutjen- Drecoll E, Shimizu T, Rohrbach M, Rohen J W. Quantitative analysis of 'plaque material' between ciliary muscle tips in normal- and glaucomatous eyes. Exp Eye Res. 1986; 42: 457-465.
35. Johnson D, Gottanka J, Flugel C, Hoffmann F, Futa R, Lutjen-Drecoll E. Ultrastructural changes in the trabecular meshwork of human eyes treated with corticosteroids. Arch Ophthalmol. 1997; 115: 375-383.