Management and Outcome of Secondary Angle Closure Glaucoma: Analytical Study

^{*1}Dr. Jitendra Kumar, ²Dr. Jyoti Sharma, ³Dr.Kanhaiya Prasad, ⁴Dr.Vijay Pratap Singh.

¹ Associate Professor & Head, Department Of Ophthalmology, Maharani Laxmi Bai Medical College, Jhansi, UP, India.

^{2, 3, 4} Junior Resident, Department Of Ophthalmology, Maharani Laxmi Bai Medical College, Jhansi, UP, India Corresponding Authour: Dr. Jitendra Kumar

Abstract: Glaucoma is a potentially blinding disease of global importance. It is second leading cause of blindness after cataract. In secondary angle closure glaucoma, the underlying cause can close the angle directly by local iris and angle factors or by acting to move the crystalline lens forward causing pupillary block (secondary pupillary block). The aim of our study is to find the common causes, demographics and clinical features and outcomes of therapy in various secondary angle closure glaucomas in Bundelkhand region. A total of 137 Patients who were diagnosed as a case of secondary angle closure glaucoma, included in this analytical study conducted in the Department of Ophthalmology, MLBMC Jhansi. Patients with recently diagnosed as a case of secondary angle closure glaucoma, included in this analytical on secondary angle closure glaucoma in which the male female ratio was1.36:1. Most of the cases of secondary angle closure glaucoma was Phacomorphic glaucoma 43.07% (59 patients) followed by uveitic 24.82 (34 patients), traumatic 15.33% (21patients), but in this study we found no case of Drug-induced glaucoma. In this study there is higher incidence of Phacomorphic glaucoma due to lack of awareness, poor socio-economic status, and lack of tertiary care facility in Bundelkhand region.

Keywords: Cataract, Drug-induced glaucoma, Pupillary block, Phacomorphic glaucoma, Secondary angle closure glaucoma.

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I. Introduction

Glaucoma is a potentially blinding disease of global importance. It is the second leading cause of blindness after cataract. ^[1] 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 blindness. ^[1] 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 annually. ^[2] In secondary angle closure glaucoma, the underlying cause can close the angle directly by local iris and angle factors or by acting to move the crystalline lens forward causing pupillary block (secondary pupillary block). This is important since some of these patients with secondary pupillary block will respond to laser iridotomy. They are common causes of glaucoma and can produce high elevations of intraocular pressure (IOP) and ocular morbidity. This review will discuss the risk factors, signs and symptoms, pathophysiology, imaging and the treatment modalities of secondary angle closure glaucomas. In most eyes with this sort of glaucoma, the pressure in the eye is constantly higher than 'normal' because there is a blockage of, or damage to the trabecular meshwork. In some eyes, the pressure is intermittently higher than normal because the iris blocks the trabecular meshwork only some of the time

1. IClassification of	of secondary angle closure glaucoma
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With pupillary block	Without pupillary Block
 Aqueous misdirection syndrome 	Neovascular glaucoma
	 Iridocorneal endothelial syndromes (ICE)
	Infammatory glaucoma
	Ciliary body cysts, tumors
	 Scleral buckling and postvitreoretinal procedures.
	 Ciliary body swelling secondary to:
	1. Central retinal vein occlusion (CRVO)
	2. Panretinal photocoagulation (PRP)
	3. Drugs or inflammation

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1.2 Risk Factors				
 Hyperopic ^[3] Family history of angle closure ^[4] Advancing age ^[5] Female gender^[6] 	 Asian or Inuit descent^[7] Shallow anterior chamber depth^[8] Shorter axial length^[9] Thicker lens^[10] 			

1.3 Symptoms

In the early stages of secondary angle closure glaucoma, there may be no symptoms – vision may seem perfec tly ormalandt herei snopain. The condition is usually first picked up when patient's eves are examined by the optomet rist (optician), or when the condition causing glaucoma is id entified. For this rea on, r egular visits everv vear are essential if glaucoma is to be detected early. In later stages of glaucoma, when a considerable amount of field of vision has been lost, although reading vision and vision for recognizing people is usually still good. However, if the glaucoma is untreated, even the centre of the field of vision may be damaged so that reading vision becomes affected and sight may be lost. In some eyes, there may be intermittent symptoms of eye ache with cloudy vision, where the vision becomes milky or hazy, like looking through smoke.

1.4 Classifications

1.5 Neovascular Glaucoma -Neovascular glaucoma (NVG) arises in response to retinal ischemia, the common predisposing factors being central retinal vein occlusion and diabetic retinopathy. An NVG patient requires a broad diagnostic workup to determine the underlying cause ^[11] and also a predisposed patient requires careful monitoring to detect NVG in its earliest stages.

1.6 Inflammatory Glaucoma -Glaucoma is a potentially devastating complication of uveitis and remains a therapeutic challenge despite availability of new modalities of treatment in both the conditions.^[12]

Inflammation can produce secondary angle closure glaucoma with pupillary block mechanism due to posterior synechiae formation or without pupillary block due to inflammatory peripheral iris swelling, exudates in the angle contracting to form PAS or due to forward rotation of the ciliary body. PAS form easily in eyes with shallow anterior chamber and in eyes with chronic inflammatory processes.

Inflammatory glaucoma can occur after trauma, surgery, idiopathic inflammatory condition or due to specific uveitic entities.

1.7 Aqueous Misdirection Syndrome -The aqueous misdirection syndrome is a form of secondary angle closure glaucoma occurring postsurgery with raised intraocular pressure (IOP), shallow or flat anterior chamber (AC) in the presence of a patent peripheral iridectomy (PI).^[13] It is unresponsive to miotic or filtering surgery. It can occur after filtering surgery ^[14], cataract ^[15] /combined surgery, surgical peripheral iridectomy, following suturelysis ^[16], glaucoma drainage device implantation ^[17] or even after laser iridotomy ^[18]. The predisposing factors are pre-existing angle closure glaucoma, shallow anterior chamber due to wound leak or overfilteration. The pathophysiology is not completely understood, but is believed that the primary mechanism is a blockage of anterior aqueous flow at the level of the ciliary body combined with an inherent impermeability defect in the anterior hyaloid.

1.8 Iridocorneal Endothelial Syndromes -Iridocorneal endothelial (ICE) syndrome is a spectrum of ocular diseases characterized by corneal endothelial abnormalities, unilateral glaucoma and iris stromal abnormalities. ^[19] They include progressive iris atrophy, Chandler syndrome and Cogan-Reese syndrome. They may be regarded as different manifestations of the same disease process. It is caused by an abnormal corneal endothelium which forms a membrane (ICE membrane) over the anterior surface of the iris and the angle structures, which on contraction distorts the iris, forms peripheral anterior synechiae and closes the angle leading to glaucoma. ^[20] Half of all the patients of ICE syndrome develop glaucoma. ^[21]

1.9 Glaucoma following Scleral Buckling Procedures -Here angle closure glaucoma is produced by swelling of the ciliary body due to impaired venous drainage from the vortex veins by the scleral buckle. The incidence of angle closure glaucoma after scleral buckling procedures range from 1.4 to 4.4%. ^[22] The risk factors are pre-existing narrow angles, use of an encircling band, placement of the band anterior to the equator and high myopia.

1.10 Glaucoma after Silicone Oil Injection -Silicone oil is used as a vitreous substitute for retinal tamponade. It can produce glaucoma by pupillary block, inflammation, synechial closure, neovascularization, migration of oil into anterior chamber or by open angle mechanism. Secondary glaucoma after silicone oil injection has been reported to be in 6 to 30% of eyes.^[23]

II. Method And Material

A total of 137 Patients who were diagnosed as a case of secondary angle closure glaucoma, included in this analytical study conducted in the Department of Ophthalmology, Maharani Laxmi Bai Medical College, Jhansi, Uttar Pradesh, India over a period of 15 months from March 2016 to May 2017. The procedures followed were in accordance with the ethical standard committee on human experimentation (institutional or regional) and with the Helsinki Declaration of 1975, as revised in 2000. The necessary permission from the Ethical and Research Committee was obtained for the study.

2.1 Inclusion criteria

Patients recently diagnosed with secondary angle closure glaucoma. 2. Both male and female patients were included in the study. 3. There was no age limit in the study.

2.2 Exclusion criteria

Patients with secondary angle closure glaucoma already on medication 2. History of any ocular surgery related to glaucoma, were excluded from the study 3. Patients with open angle, primary angle closure glaucoma, were excluded from the study. 4. Patients with neurological disorders were not included in this study. An assessment of present complaints, detailed clinical history (present and past), and detailed history of dietary habits, any other medication and any ocular surgery/trauma were taken. Age, sex, occupation, socio-economic status, and personal history were recorded. Ophthalmological check up as external examination of the eves, visual acuity, torch light examination, slit lamp examination, colour vision, refraction (pre and post), direct ophthalmoscopy, tonometry, provocation test for glaucoma, Gonioscopy, Perimetry (HFA), Pachymetry, and OCT were done. B-scan, CT and MRI were done in special cases. Close Follow-up was done for complications. At each visit vision was noted and final visual outcome at 3 months was noted and analyzed.

Table -	1: Sex and etiolo	ogy wise di		. Results	secondary a	ngle closu	ire glaucoma (n=137)
	Types of	Male	Percentage	Female	Percentage	Total	Percentage	
	glaucoma							
	Phacomorphic	26	18.98%	33	24.09%	59	43.07%	
	glaucoma							
	Uveitic	20	14.59%	14	10.22%	34	24.82%	
	Traumatic	17	12.41%	04	2.92%	21	15.33%	
	Neovascular	07	5.11%	04	2.92%	11	8.03%	
	Postsurgical	05	3.65%	02	1.46%	07	5.11%	
	Drug-induced	00	0%	00	0%	00	0%	
	Others	04	2.92%	01	0.73%	05	3.65%	
	Total	79	57.66%	58	42.34%	137	100%]

Table 2: Age V	Wise Distribution	Of Patients And	d Most Common	Causes
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Age group (In years)	No. of patients	Percentage	Most common cause
1-15	01	0.73%	Congenital
16-30	07	5.11%	Uveitic
31-45	23	16.79%	Traumatic and uveitic
46-60	35	25.55%	Phacomorphic
61-75	54	39.42%	Phacomorphic
Above 75	17	12.41%	Phacomorphic
Total	137	100%	

Table 3: Vis	sual assessmen	t of the	patients (Pre)
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I	Table 3. Visual assessment of the patients (11c)		
BCVA	No. of patients	Percentage	
6/9-6/12	06	4.38%	
6/18-6/24	12	8.76%	
6/36-6/60	23	16.79%	
5/60-1/60	16	11.68%	
FC to HM	13	9.49%	
PL (+)	62	45.26%	
PL (-)	05	3.65%	
Total	137	100%	

BCVA: Best corrected visual acuity, FC: Finger count, HM: Hand movement, PL: Perception of light

	The second second and second				
BCVA	No. of patients	Percentage			
6/9-6/12	20	14.6%			
6/18-6/24	41	29.93%			
6/36-6/60	33	24.09%			
5/60-1/60	17	12.41%			
FC to HM	12	8.76%			
PL (+)	09	6.57%			
PL (-)	05	3.65%			
Total	137	100%			

Table 4: Final visual outcome after 3 months (After conservative or surgical management)

BCVA: Best corrected visual acuity, FC: Finger count, HM: Hand movement, PL: Perception of light

IV. Discussion

In developing country like India Secondary closed angle glaucomas are an important cause of ocular morbidity and vision loss. 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. 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 million^[24]. Common causes of secondary glaucoma reported by Gadiaet al 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%).^[25]This analytical study mainly focused on secondary angle closure glaucoma in which the male female ratio was1.36:1. Most of the cases of secondary angle closure glaucoma had inflammatory pathology. The most common secondary angle closure glaucoma was Phacomorphic glaucoma 43.07% (59 patients) followed by uveitic 24.82 (34 patients), traumatic 15.33% (21 patients), but in this study no any single cases of Drug-induced glaucoma was found. Most common age group suffering from secondary angle closure glaucoma was 61-75 years (39.42%) followed by 46-60 years (25.55%) and most common glaucoma among these age group was Phacomorphic glaucoma followed by traumatic and uveitic glaucoma. Least common affected age group was 1-15 years (0.73%). In our study 62 patients (45.26%) patients were PL (+) followed by 23 patients had 6/36 to 6/60 BCVA, while 5 patients (3.655) were PL (-). Final visual outcome (BCVA) after 3 months of conservative or surgical management, VA 6/18-6/24 in 29.93%, followed by 6/36-6/60 VA in 24.09% patients. There were drastic changes in BCVA in patients with PL (+) visual acuity in case of Phacomorphic glaucoma. In this study there is higher incidence of Phacomorphic glaucoma due to lack of awareness, poor socio-economic status, and lack of tertiary care facility in Bundelkhand region.

V. Conclusion

A careful history, clinical examination, and when necessary anterior segment imaging such as slit lamp examination, Gonioscopy aid in recognizing the etiology for secondary angle closure. Identifying the cause early and timely institution of appropriate therapy helps in improving the visual outcome and reducing the ocular morbidity. Some patients in the poorest parts of the India with cataract wait for self resolution and free eye camp surgery in the vicinity of their homes rather than visit a distant hospital for treatment for reasons both social and economic. Poor transportation and bad roads make travelling difficult; two thirds of the patients do not have access to an escort to the hospital and consequently do not reach the hospital on time. A similar percentage does not reach the health care practitioners due to economic constraints. As is true for all other cases of glaucoma, the importance of long-term follow-up to assess IOP control and field loss cannot be over emphasized. Public health education programs to increase awareness and community support, upgradation of eye care delivery services in rural areas for early referral and treatment of cataract cases can help in primary prevention of this condition. Also improving the peripheral health care infrastructure for postoperative care as well as management of the fellow eye can prevent the incidence of visual loss, and the consequent economic burden on the community at large. 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.

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