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Al-Shifa Journal of Ophthalmology

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   2. Cochereau I, Goldschmidt P, Goepogui A, Afghani T, Delval L, Pouliquen P, Bourcier T, Robert PY. Efficacy and safety of short duration azithromycin eye drops versus azithromycin single oral dose for the treatment of trachoma in children - a randomised, controlled, double-masked clinical trial. Br J Ophthalmol. 2007;91:667-72.
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Al-Shifa Journal of Ophthalmology

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EDITORIAL

Monitoring Outcomes of Cataract Surgery

Ismatullah Chaudhry

Visual loss from cataract represents an estimated 50% or more of the global burden of blindness, and according to the last national blindness survey (2003-4) conducted in Pakistan, it counted to be 53% of all blindness. By its sheer magnitude, unoperated cataract has public health dimensions. As a result of population growth and ageing, the number of people blind as a result of cataract is estimated to double in the next 20 years if the cataract surgical services are not increased in parallel and improved.[1] Therefore, the efforts and intervention need to be planned and applied in a public health mode.

Time tested, safe and effective technologies are available that could restore near normal vision to a large majority of those operated for cataract. Despite this, the magnitude of the global burden of blindness from unoperated cataract continues to increase. The fact that nearly 20 million persons are currently blind from cataract reflects the lack of access to surgical services for a majority of these persons, even though the knowledge and skills required for applying the technology exist. That is the reason intervention against cataract blindness has received priority attention globally and in Pakistan as well.

Cataract surgery has been demonstrated to be one of the most cost effective health interventions.[1] The cost effectiveness is related to short operation time, the potential for high volume, high success rates, and the low cost of consumables. However, when success rates are low, cost effectiveness is reduced.[2]

For too long, emphasis has been placed on the quantity of surgical operations performed, rather than the outcome of such surgery, as an indicator of the performance of cataract surgical services. Fortunately, the paradigm is changing, with greater emphasis being focused on using the outcome of surgery as an indicator, in addition to the numbers of cataract surgeries performed.

It needs to be stressed that the objective of performing cataract surgery is not merely to restore visual function at the 'organ level'. More importantly, the goal should be to achieve restoration of visual function, as measured by visual acuity, contrast sensitivity and other parameters, on the one hand and, functional vision, as judged by such measures as activities of daily living (ADL), on the other.

Monitoring outcomes could, therefore, be applied in a clinical setting, where the visual outcome of cataract surgery (post-operative visual acuity) is primarily measured. In addition, studies based on ADL, patient well-being, quality of life and patient satisfaction may be instituted as a routine or, more commonly, on randomly selected post-operative patient groups.

The clinical monitoring of post-operative visual outcome falls within the realm of clinical audit. It should be considered mandatory (an absolute requirement), in any setting where cataract surgery is performed, that records are kept, among other clinical details, of the pre-operative and post-operative visual acuity of both eyes of the patient. Such recordings, carried out at appropriate post-operative timings, including a record of presenting and best corrected visual acuity, would provide invaluable information. In this context, the monitoring of the outcome of cataract

services in general, and cataract surgery in particular, has become imperative. An effective tool to measure the outcomes of cataract surgery is crucial to improving the quality – we simply cannot improve what we don't measure. Quality of surgery and resulting patient satisfaction are fundamental elements to a sustainable cataract service.

A global consortium of leading eye health organizations have launched a new smartphone app named *BOOST (Better Operative Outcomes Software Tool)*, a simple, free and easy-to-use application to help ophthalmic surgeons and community ophthalmologists monitor and improve cataract surgical outcomes, especially for people in developing countries (boostcataract.org).

Good surgical outcomes for patients depend on hospitals having the right equipment, well-trained surgeons, and strong quality assurance practices. However, many patients in developing countries do not return for follow up care because of lack of transport and other costs. Surgeons and eye care facilities need a simple tool to measure, benchmark and improve their results, even where few patients return.

Traditionally, surgeons measure outcomes 6-8 weeks after surgery – but if patients don't return for follow-up care, the surgeons cannot assess, and much less improve their own surgical results. BOOST solves this challenge by assessing results immediately after surgery and offering specific advice to improve outcomes.

BOOST not only helps individual practitioners, it also provides a platform for governments, NGOs, hospitals and training institutes to monitor cataract surgical outcomes in a simple manner. All data is anonymous, and users can opt out of sharing their data at any time. BOOST also automatically suggests tailored strategies to help further improve quality. By equipping surgeons and hospitals with the right tools, we can establish and support a practice of continuous quality improvement across the eye care sector.

References:

1. Foster A. Cataract—a global perspective: output, outcome and outlay. Eye 1999; 13:449–53.
   Limburg H, Foster A, Vaidyanathan K, *et al.* Monitoring visual outcome of cataract surgery in India. Bull World Health Organ 1999; 77:455–60.

ORIGINAL ARTICLE

A Comparison of Macular Edema Formation in Diabetic Patients Undergoing Phacoemulsification and Extracapsular Cataract Extraction

Faran bin Afzal[1], Saba Waqar Qureshi[2], Muhammad Usman Arshad[3], Hassan Massana[1], Masud ul Hassan[4], Muhammad Nawaz Cheema[5]

ABSTRACT:

Objective: To investigate the frequency of macular edema formation in diabetic patients undergoing cataract surgery and to compare the frequency of macular edema formation in diabetic patients undergoing phacoemulsification and extracapsular cataract extraction.

Study design: Descriptive case series

Materials and Methods: The study was conducted at Al Shifa Trust Eye Hospital from 25th March to 25th September 2018 and a total of 100 patients were selected. Central Subfield Mean Thickness (CSMT) was recorded through OCT one day before surgery and one-month post-surgery. One of the groups underwent Extra capsular cataract extraction (ECCE) (N=56) while the other underwent phacoemulsification (N=44). Statistical analyses were done. Frequency of macular edema between phacoemulsification and ECCE was compared through chi-square. $P<0.05$ was considered significant.

Results: There were insignificant differences in the formation of macular edema in ECCE and phacoemulsification groups and with respect to age and gender.

Conclusions: For diabetic patients undergoing cataract surgery, there is no significant difference between phacoemulsification and ECCE in terms of macular edema formation. *Al-Shifa Journal of Ophthalmology 2019; 15(1): 8-14.*

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Introduction:

Diabetes mellitus (DM) remains one of the most common systemic diseases responsible for causing reduced vision in both the developed and developing countries. According to the International Diabetes Federation (IDF), 382 million people suffer from diabetes worldwide. Out of these, 80% belong to low- and middle-income countries. In Pakistan, the prevalence of DM is 6.8%.[(1)]

Diabetic retinopathy, macular edema and early cataract are common causes for decreased visual acuity in diabetic patients. DM increases the chances of cataract formation through sorbitol accumulation leading to osmotic stress and swelling of the lens protein.[(2)] Although diabetic macular edema can exist even in early stages of

retinopathy, studies have shown that there is a strong correlation between cataract surgery and accelerated retinopathy or macular edema formation.[3]

Inflammatory mediators released from affected retina lead to macular edema and the effect is enhanced by surgical intervention. In a previous study, incidence of macular edema in diabetic patients after uncomplicated phacoemulsification was found to be 18%.[4] Macular edema and accelerated retinopathy were the main causes of reduced visual acuity after cataract surgery.[4]

Ocular Coherence Tomography (OCT) can detect macular edema both qualitatively and quantitatively in normal as well as diabetic patients. OCT is comparable to Fluorescein angiography (FA) in detecting macular edema. However OCT is superior to FA in detecting subtle changes in macular thickness.[4] [5]

Extra capsular cataract surgeries (ECCE) with manual expression and phacoemulsification (phaco) differ in terms of wound size, duration and method of cataract extraction. Phacoemulsification involves usage of ultrasound to break the cataract into small pieces and aspirate it.[6] Because of the nature of the two techniques, different sort of surgical trauma is exerted which in turn can lead to varying degrees of macular edema formation. Although modern phaco techniques allow ophthalmologists to operate on most cataracts, ECCE with manual expression may still be the better option for mature cataracts where phaco might result in increased surgical time and increased ultrasonic energy being delivered and therefore increase heat generation. This can lead to serious complications like wound burn, wound leak, corneal edema and corneal decompensation.[7]

Majority of the population of Pakistan resides in the rural areas. Access to tertiary medical care is restricted due to poverty and illiteracy. This includes decreased access to modern diabetic medication and tertiary ophthalmic care. This results in late presentations with ocular complications such as retinopathy and cataract as mentioned above. For a number of reasons comprising of limited financial and other hospital resources that eventually affect the availability of phaco machines and other necessary equipment, it's not possible that all these patients get to have a phacoemulsification cataract surgery. For them, ECCE with manual expression provides an affordable alternative.

Given the fact that cataract surgery can give rise to macular edema which leads to reduced vision, the aim of this study was to identify as to how the two surgeries differ in terms of macular edema formation in our diabetic population.

Material and Methods:

This descriptive case series was carried out at the Al- Shifa Trust Eye hospital. The duration of the study was six months. A total of 100 patients were included in this study. These included patients with type 2 diabetes between the ages of 35 to 70 years who had cataract in either eye. Patients who had undergone surgery previously in the same eye or who had concurrent ocular/systemic conditions that could lead to potential irreversible vision loss or patients with retinal/choroidal disease that could affect macular thickness were excluded from the study. A written informed consent was taken from these patients. OCT scans were done following a thorough clinical evaluation one day pre-operatively.

Cataract surgery was performed by senior residents and registrars. None of the patients were strictly pre-assigned to undergo any surgery. It was left to the choice of the operating surgeon to decide which patient underwent which type of cataract surgery (phaco vs ECCE). For phacoemulsification, a clear corneal

incision was made, and continuous curvilinear capsulorrhexis was performed. For extra capsular cataract extraction (ECCE), incision of about 5-7 mm was made and cataract was extracted. The wound was closed with 5 x 10-0 nylon sutures. For Phaco, phacoemulsification equipment (Oertli catarrhex, Bausch and Lomb-Millennium phaco vitrectomy system and Alcon Sovereign) was used. The nucleus was divided, and phacoemulsification and aspiration were performed and after cortical aspiration, an acrylic foldable IOL was inserted in the capsular bag. Follow ups were ensured by adequate patient counseling recording personnel or next of kin cell phone numbers. All the observations were noted on pre-designed structured proforma by the trainee researcher individually for each patient after ruling out exclusion criteria and was being confirmed by the supervisor. (Consultant Ophthalmologist).

Data was entered and analyzed using Statistical Package for Social Sciences (SPSS) version 20.0. Chi Square test was applied to all results (p value of ≤ 0.05 was considered significant). The frequency of macular edema formation after cataract surgery and its percentage was calculated. Frequency of macular edema formation between the Phaco and ECCE groups was compared by using the chi square test. Frequency distribution of macular edema was calculated for both the genders and compared using chi square test for both phaco and ECCE groups. Patients were divided into two broad age groups (35-55 years and 56-70 years) and the frequency of macular edema formation was then compared for both ECCE and phacoemulsification by using chi square test. Patients who had received treatment previously (either anti-VEGF injections and or photocoagulation) in both the groups were divided into further subgroups and the frequency of macular edema formation was then compared using chi square test.

Results:

A total of 100 patients were enrolled in this study. Among these, 44 patients underwent phaco and 56 patients underwent ECCE. There were 60 patients who were operated in their right eye and 40 patients who were operated in their left eye. All of them remained part of the study. There were no major complications reported in the cataract surgeries. Significant results have shown that within the limitations of this study; the effects of age, gender and type of surgical intervention were insignificant. Similarly, history of previous treatment didn't have a significant effect on macular edema formation. Furthermore, lesser macular edema formation was observed in patients with no history of previous treatment.

A total of 9 (20.5%) patients in the phacoemulsification group and 16 (28.6%) patients in the ECCE group developed macular edema. Chi square analysis (Table no. 1) showed that there was no significant difference in the frequency of macular edema formation in these two groups. In terms of age, 52% of the population belonged to the 35-55 years age group and 48% belonged to the 56- 70 years age group. 10 out of 52 patients, (19.23%) in the younger age group developed macular edema and 15 out of 48 patients, (31.25%) in the older age group developed macular edema. However, the differences between the Phaco and ECCE surgical subgroups in the two age groups were statistically insignificant for macular edema formation. Almost a quarter of all male patients and all female patients developed macular edema. The differences between the Phaco and ECCE groups were overall insignificant for the two genders.

Table no.3 illustrates the results of previous treatment factors and type of surgical procedure on macular edema formation. Least amount of macular edema was found in patients without any history of previous treatment. More than 50 percent of patients developed macular edema that had a history

of previous treatment including laser alone, anti VEGF alone or both laser and anti-VEGF. However, previous treatment history didn't have an overall effect on macular edema formation between the Phaco and ECCE groups.

Table 1: Comparison of macular edema formation in Phaco and ECCE groups.

Groups	Macular Edema Formation		Total////	*p* value
	YES	NO		
Phacoemulsification	9 (20.5%)	35 (79.5%)	44	.3173
ECCE	16 (28.6%)	40 (71.4%)	56	.3173
Total	25	75	100	.3173

Table 2: Stratification with respect to age of macular edema formation in phacoemulsification and ECCE groups.

AGE GROUPS	PHACO/ECCE	MACULAR EDEMA		TOTAL	*p* value
		YES	NO		
35- 55 YEARS	PHACO	2	19	21	**0.167**
	ECCE	8	23	31	
	TOTAL	10	42	52	
56-70 YEARS	PHACO	7	16	23	**1.000**
	ECCE	8	17	25	
	TOTAL	15	33	48	

Table 3: Stratification with respect to previous treatment on macular edema formation

PREVIOUS TREATMENT	PHACO/ECCE	MACULAR EDEMA		TOTAL	*p* value
		YES	NO		
NONE	PHACO	2	22	24	**0.208**
	ECCE	0	29	29	
	TOTAL	2	51	53	
LASER	PHACO	2	2	4	**0.406**
	ECCE	6	2	8	
	TOTAL	8	4	12	
ANTI-VEGF	PHACO	2	5	7	**0.167**
	ECCE	7	4	11	
	TOTAL	9	9	18	
LASER+ ANTI-VEGF	PHACO	3	6	9	**0.627**
	ECCE	3	5	8	
	TOTAL	6	11	17	

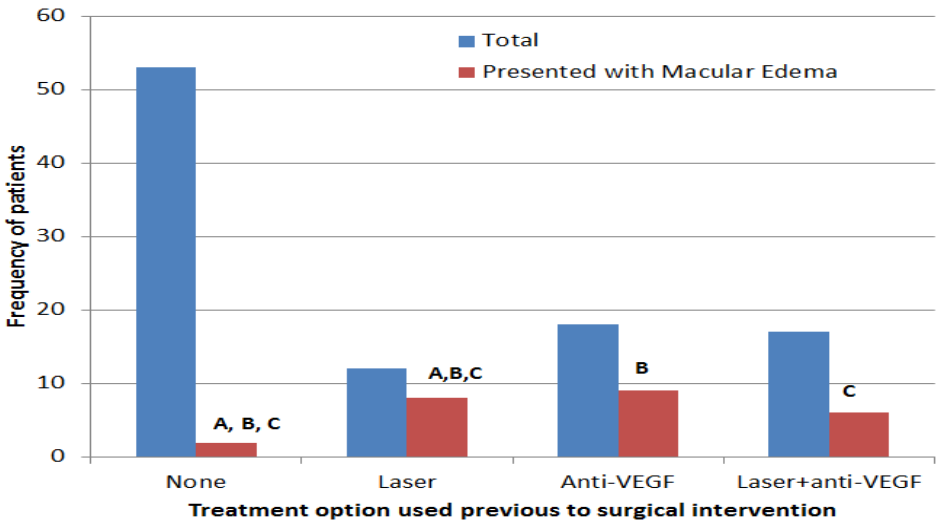

Figure 1: Differences between the frequencies of observed macular edema in patients with and without different previous treatments

Discussion:

The incidence and progression of cataract is elevated in patients with diabetes mellitus, [8] and they have higher complication rates from cataract surgery [9]. In addition to this, diabetes is one of the risk factors most frequently associated with Macular Edema[10]. Furthermore, macular edema is the most frequent cause of poor visual acuity after cataract surgery in patients with diabetes mellitus. Age has been proven by various studies as an important predictor for cataract formation but not directly for macular edema formation. However it may have an indirect link to it through prolonged uncontrolled blood glucose levels[11] which may lead to inflammatory cascade. In our study, there was no observed significant effect of age, as there was no significant difference between observations of macular edema in either age group (Table no. 2), but as the control of diabetes was not observed concomitantly, these results cannot be sufficient to represent the degenerative changes that may be observed with aging diabetic patients. Almost an equal number of male and female subjects were tested in this study. An insignificant difference was observed between these two population groups, as shown by table number 4. Similar results have been reported previously and it can be concluded that Macular edema formation is unaffected by gender. Diabetic patients having either ECCE or phacoemulsification may have progression of diabetic retinopathy post-operatively. Those having ECCE also have an increased rate of anterior segment complications including elevated levels of fibrin and the development of posterior synechia. Similarly; post-operative aqueous flare is greater in diabetic patients having phacoemulsification and occurs with increased intensity with advancing retinopathy [12, 13].

However, the insignificant difference found in this study between patients who underwent phacoemulsification vs. those who underwent extra capsular surgery does not support this idea. This may have one or two implications.

The results of the present study however contradict those of previous studies, in which lesser incidence of post-operative diabetic macular edema has been reported

after phacoemulsification. The factors contributing to this include smaller incision, lower rates of intraoperative complications such as vitreous loss and iris prolapse, shorter procedure time, and faster visual recovery. Although all these factors were in play in the present study too, there are many other confounding factors that have contributed to this study.

It may be possible that ignorance about the diabetic control of the patients behaved as a confounding factor. As the glucose profiling of the patients was not done as part of this study, this stands as a major limitation. In diabetics the blood-aqueous barrier is impaired leading to the development of a sight-threatening macular edema, a process that is exacerbated by cataract surgery.[14] Therefore, it is possible that rather than the type of surgery, the diabetes itself played a major role in macular edema formation in these patients, and any intervention exacerbated this process. This is in accordance with previously reported results which have elucidated that patients with diabetes mellitus have higher complication rates from cataract surgery in general. [9, 13]

It has been shown in previous research that there is a significant benefit of focal laser photocoagulation for eyes with clinically significant macular edema.[15] However an interesting finding of this study is that previous treatment administration can exacerbate the formation of macular edema as more than fifty percent patients who had undergone previous treatment developed macular edema.

One of the factors responsible for this result may also include the status of diabetes. The patients who underwent previous treatment are mostly those who had a comparatively more advanced stage of disease than the group who were not administered any previous treatment. This can be assumed from the fact that they required the treatment options tested herein due to the development of diabetic retinopathy. This development is a sequel of diabetes that occurs at later stages and derangement of blood glucose. Therefore, the more frequent development of macular edema may be a direct consequence of this diabetes rather than the administration of previous treatment.

Although all efforts were made to reduce the effects of bias, it was not a hundred percent bias-free data collection, as computer-operated randomization was not done. In addition, it would be recommended that a similar study be performed on a wider time scale, with a larger sample size with emphasis on blood sugar profiling and stratification of patients according to stage of disease may reveal more comprehensive results.

Conclusion:

Cataract surgery can lead to macular edema formation in patients suffering from Diabetes Mellitus. This process is independent of the type of surgery.

References:

1. Erie JC, Baratz KH, Hodge DO, Schleck CD, Burke JP. Incidence of cataract surgery from 1980 through 2004: 25-year population-based study. J Cataract Refract Surg. 2007;33:1273–1277.
2. Pannu RS. Intra-ocular lens. [Internet]. Vol. 93, Journal of the Indian Medical Association. 1995 [cited 2018 Sep 25]. p. 399.
3. Malik AR, Qazi ZA, Gilbert C. Visual outcome after high volume cataract surgery in Pakistan [Internet]. Vol. 87, British Journal of Ophthalmology. 2003 [cited 2018 Sep 25]. p. 937–40.
4. Bourne R, Dineen B, Jadoon Z, Lee PS, Khan A, Johnson GJ, et al. Outcomes of cataract surgery in Pakistan: Results from the Pakistan National Blindness and Visual Impairment Survey. Br J Ophthalmol [Internet]. 2007 Apr [cited 2018 Sep 25];91(4):420–6.

5. Chhipa SA, Junejo MK. Outcomes of cataract surgery at teaching hospital in Karachi. J Pak Med Assoc [Internet]. 2018 [cited 2018 Sep 25];68(1):76–80.
6. S.A. K, M. A, A.M. A, M. I. Comparison of the efficacy of subtenon with peribulbar Local anesthesia without hyaluronidase in patients undergoing cataract surgery. J Coll Physicians Surg Pakistan [Internet]. 2014 [cited 2018 Sep 25];24(5):331–4.
7. Baig R, Chaudhry TA, Kukreja S, Shakil S, Ahmad K. Patients' satisfaction and spectacle independence after cataract surgery with multifocal intraocular lens implantation in a tertiary care hospital. J Pak Med Assoc [Internet]. 2016 [cited 2018 Sep 25];66(6):745–7.
8. Latif K, Shakir M, Zafar S, Rizvi SF, Naz S. Outcomes of Congenital Cataract Surgery in a Tertiary Care Hospital. Pakistan J Ophthalmol Pak J Ophthalmol [Internet]. 2014 [cited 2018 Sep 25];30(1).
9. Khan MA, Malik AM, Niazi MK, Khan AG, Irum N. Efficacy of manual small incision cataract surgery for all types of cataracts. J Rawal Med Coll [Internet]. 2009 [cited 2018 Sep 25];13(2):70–2.
10. Bellucci R. An introduction to intraocular lenses: Material, optics, haptics, design and aberration. Cataract. 2013;3:38–55.
11. Özyol P, Özyol E, Karel F. Biocompatibility of intraocular lenses [Internet]. Vol. 47, Turk Oftalmoloiji Dergisi. Galenos Yayinevi; 2017 [cited 2018 Sep 26]. p. 221–5.
12. Goodall KL, Ghosh YK. Total Opacification of Intraocular Lens Implant After Uncomplicated Cataract Surgery: A Case Series. Arch Ophthalmol [Internet]. 2004 May 1 [cited 2018 Sep 26];122(5):782.
13. Ronge laura. Opacification of IOLs: A Problem Often Misdiagnosed? - American Academy of Ophthalmology [Internet]. [cited 2018 Sep 26].
14. Milauskas AT. Silicone Intraocular Lens Implant Discoloration in Humans [Internet]. Vol. 109, Archives of Ophthalmology. American Medical Association; 1991 [cited 2018 Oct 3]. p. 913.
15. Srinivasan S. Intraocular lens opacification: What have we learned so far [Internet]. Vol. 44, Journal of Cataract and Refractive Surgery. Elsevier; 2018 [cited 2018 Oct 3]. p. 1301–2.
16. Neuhann IM, Kleinmann G, Apple DJ. A New Classification of Calcification of Intraocular Lenses. Ophthalmology [Internet]. 2008 Jan 1 [cited 2018 Oct 3];115(1):73–9.
17. Giers BC, Tandogan T, Auffarth GU, Choi CY, Auerbach FN, Sel S, et al. Hydrophilic intraocular lens opacification after posterior lamellar keratoplasty - A material analysis with special reference to optical quality assessment. BMC Ophthalmol [Internet]. 2017 Dec 22 [cited 2018 Oct 3];17(1):150.

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ORIGINAL ARTICLE

Assessment of Binocular Visual Functions in Primary Open Angle Glaucoma

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ABSTRACT

Objectives: This study was conducted to find the impact of primary open angle glaucoma on the visual functions of the eye. It also aimed to find out the use of visual function tests as a screening tool for glaucoma.

Materials and Methods: Total 100 patients were included in the cross sectional study where binocular visual functions of the glaucomatous eyes were compared with those of healthy eyes from October 2017- January 2018 at Al-Shifa Trust Eye Hospital (ASTEH).After careful visual acuity testing and anterior segment ocular examination; all the subjects were checked for their contrast sensitivity, color vision and stereopsis followed by dilated fundus examination. Each patient was interviewed thereafter to fill the questionnaire and questions were explained to them in detail.

Results: Total 100 patients were included in this comparative cross-sectional study with 50 patients of glaucoma and 50 those with no ocular pathology. Males were 48% and that of females were 42% .Positive family history was found in 34% of the cases of glaucoma. Significant association of contrast sensitivity, stereopsis and color vision was found with the cases of primary open angle glaucoma. Each test performed had p value 0.04 , 0.01 ,0.01 respectively. No association was observed between gender and visual functions and age of patients with visual functions with p > 0.05. *Al-Shifa Journal of Ophthalmology 2019; 15(1): 15-20. © Al-Shifa Trust Eye Hospital, Rawalpindi, Pakistan.*

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Introduction:

Glaucoma is defined as progressive bilateral or unilateral optic neuropathy characterized by optic disc cupping with characteristic visual field defects with or without raised IOP.[1] The main types of glaucoma are open angle and closed angle, which maybe primary -that the cause is unknown or secondary - that it maybe secondary to some other ocular pathology. Long term use of corticosteroids and positive family history can also contribute to glaucoma. [2] glaucoma affecting approximately 1% of the general population over the age of 40 years.[3] Variety of medical surgical treatment options are available for the management of glaucoma but no work is being done about the early detection of optic nerve damage.[4] The raised intraocular pressure, measurement of visual acuity and visual field testing does not appear to be sensitive predictor of

development of optic nerve damage.[4] A number of other tests have been put forward to facilitate early detection of damage caused by glaucoma.

Few studies have done assessment of binocular functions in glaucoma patients.[4] Contrast sensitivity is the ability of the eyes to discriminate the luminance changes between the borders of the objects and its background.[5] Detection of low contrast objects is one of the important functions in daily routine of patient with the loss of peripheral vision due to glaucoma.[6] Stereopsis is a common term often used to denote depth perception. Glaucoma suspects show deficiency in depth perception. According to few studies the impaired stereoacuity in glaucoma patients suggests that binocular interactions can be disturbed in the presence of normal visual fields by standard automated perimetry.[7] Color vision deficiency is defined as the inability to distinguish few shades of color. In glaucoma disruption in the cones and the optic nerve fibres cause a disturbance in the color perception leading to an acquired color vision deficiency (dyschromatopsia).[8] The three tests described can have a role in determining the damage in glaucoma patients.

Materials and Methods:

A comparative cross-sectional study was conducted from October 2017 to January 2018 in Glaucoma Department and General OPD of Al-Shifa Trust Eye Hospital Rawalpindi. Visual acuity of all the patients was taken with Snellen acuity chart followed by best corrected refraction. Glaucoma patients were examined in glaucoma department by the specialized doctor. The questionnaire was based on questions related to demographics, glaucoma history, spectacle use history, best corrected visual acuity and visual functions.

Examination included measure of intraocular pressure and examination of disc and provision of respective medicine. Normal subjects with no ocular pathology were examined in OPD where detailed eye examination was done. After eye exam both the normal subjects and glaucoma patients were tested for their visual functions that is for contrast sensitivity, stereopsis and color vision. Pelli Robson contrast sensitivity test was used to measure contrast sensitivity, PV 16 and pseudoisochromatic plates were used to assess color vision and TNO test was used for the assessment of stereopsis. All the tests were performed at appropriate distances with adequate lightening conditions. The study was conducted after approval by Institutional Review Board , head of Glaucoma Department and also from the head of General OPD. All participants were included after taking verbal consent from their attendants and participants themselves.

Results:

Total 100 patients were included in this study. Males were 58% and females were 42%. Mean age of participants was 43.77 years (SD±14.086) ranging from 15-65 years.92% participants belonged from urban areas and 8% from rural areas with 42% belonging from working class and 58% from non-working class. Positive family history for glaucoma was found in 17 (34%) of the glaucoma patients.

Contrast senstivity:

Most of the glcaomatous patients (N=22,44%) had low contrast senstivity while majority of the normal individuals (N =38, 76%) showed higher contrast levels. A statistically significant association was seen between contrast sensitivity and respondent with $p<0.05$.The following chart shows the distribution of different levels of contrast sensitivity among participants.

Stereopsis:

Forty two glaucomatous subjects (84%) showed stereopsis within the range of qualitative-240 sec of arc while most of normal subjects [46 (92%)] had stereopsis within the range of 120-60 sec of arc.. The association found above had p value less

than 0.05 between respondent and stereopsis. Following graph shows the different ranges of stereopsis among participants.

Color vision:

About 32% of the cases showed mildly defective or defective color vision while all the controls had normal color vision. A relationship between respondent and color vision with p value significantly less than 0.05 was observed. The different percentages of color vision among participants is shown in the form of graph.

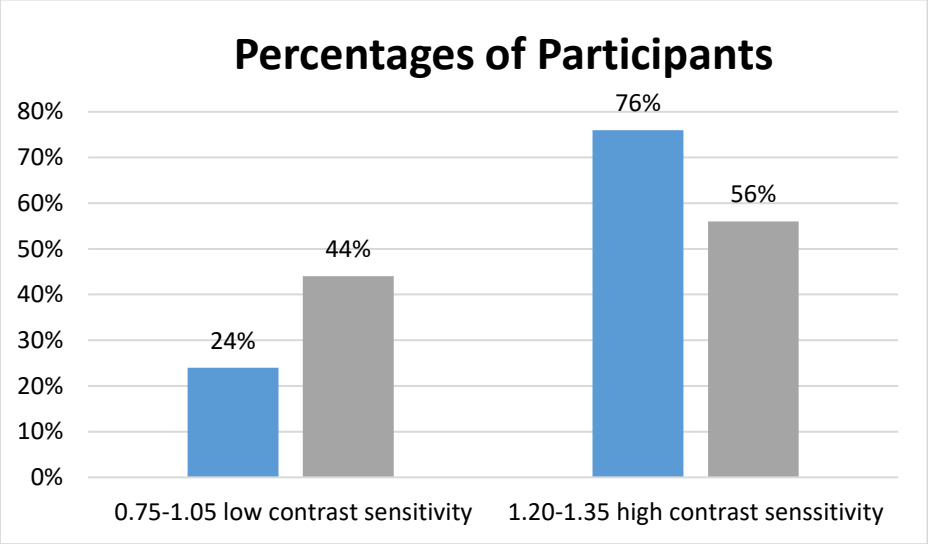

Fig:1 Percentages of different ranges of contrast sensitivity among normal and glaucomatous subjects

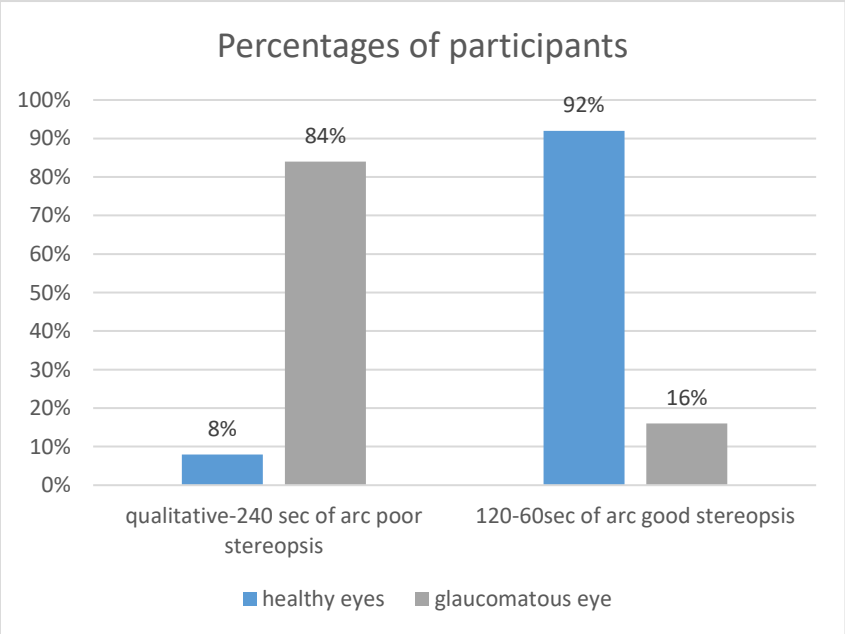

Fig.2 Percentages of range of stereopsis among normal and glaucomatous subjects

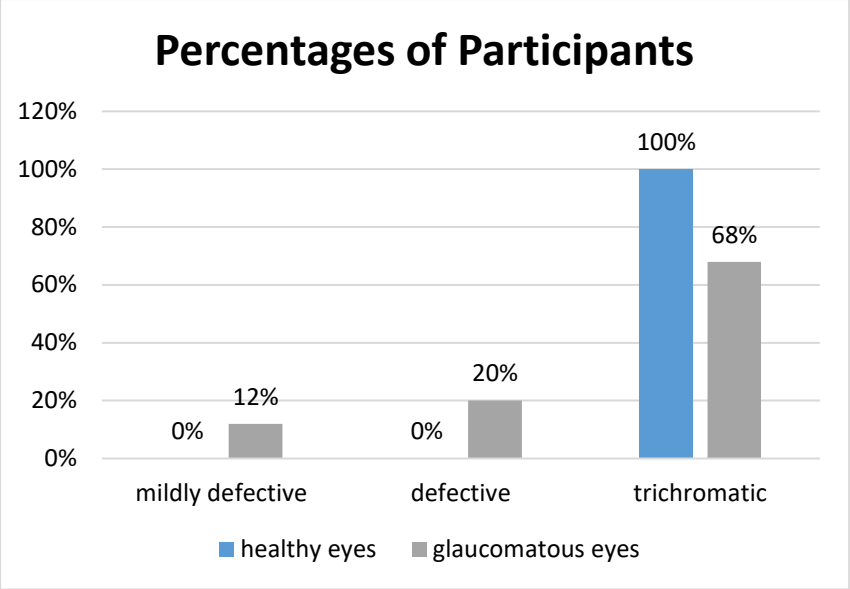

Fig.3 Percentages of binocular color vision among healthy and glaucomatous subjects

Table No.1 Association of binocular contrast, Color Vision and Stereopsis with subjects

Contrast	Glaucomatous subjects N (%)	Normal subjects N (%)	χ^2 (df)	p-value
0.75-1.05	22 (44)	28 (56)	4.456 (1)	0.048
1.20-1.35	12 (12)	38 (76)		
Stereopsis				
Gross-240	42 (84)	4 (8)	58.132 (1)	0.01
120-60	8 (16)	46 (92)		
Color Vision				
Defective	16 (32)	0 (0)	19.048 (1)	0.01
Trichromatic	34 (68)	50 (50)		

Discussion:

Glaucoma is the major cause of blindness worldwide and leads to progressive damage to the retinal ganglion cells affecting the visual functions of the eye. A case control study was conducted to determine the impact of POAG on the visual functions of the eye including contrast sensitivity, stereopsis and color vision.

Total 100 patients were included in the study with 50% cases of glaucoma and 50% those of normal patients. The number of males who participated in this study was 48% and that of females was 42%. Positive family history was found in 34% of the cases of glaucoma.

There was significant reduction in visual functions among glaucoma patients. Contrast sensitivity was reduced in all the glaucoma cases ($p < 0.05$) in comparison to

healthy age matched controls. Results were found to be in agreement with the results of other studies on measurement and comparison of grating and letter contrast sensitivity between glaucoma cases and healthy controls.[4] Similar results were found in the study conducted in Philadelphia and Israel.[9,10] The underlying cause include the changes at the level of retinal ganglion cells.[4] Result of association of glaucoma with positive family history was found to be in accordance with study conducted in Tasmania.[11]

Pseudoisochromatic plates and PV 16 was used for the evaluation of color vision among the participants. 32% of the cases had defective color vision and 68% were trichromatic, whereas 100% of controls had normal color vision. Chi square testing showed significant association with $p<0.05$. This was in accordance with research conducted to evaluate acquired color vision deficiencies in glaucoma patients which showed statistically significant results with reduction in color vision.[12] Poor yellow blue scores were observed in glaucoma patients. The study conducted to check the acquired color deficiency also indicated that this occurs due to disruption of specific physiological color mechanisms and chromatic pathways.[13] Another study over comparison of binocular visual impairment showed defective color vision among glaucoma patients.[4] The respondents with no ocular pathology had normal color vision as compared with glaucoma patients. TNO test was used for the assessment of stereopsis. 84% of the glaucoma patients showed stereopsis within the range of gross-240 seconds of arc. 92% of controls had good stereopsis within the range of 120-60 seconds of arc. The present study showed the congruency with the study conducted in the past where glaucoma patients and glaucoma suspects showed deficit in depth perception.[4] Conformity was found with the study held in USA where sufficient reduction of stereopsis was found both among patients with moderate damage and severe damage with a p value of 0.0001.[14] Similar results were found in a study done in USA by MiYoung kwon where loss of stereopsis was reported among the glaucoma patients.[15] The loss of stereopsis is reported due to changes at the level of retinal ganglion cells in glaucoma.[4]

Glaucoma being a silent killer of sight can hamper many daily activities of the patient. Based on the research done, it can be said that the three tests used for the assessment of visual function can have role to be used as screening tool in the optometric practice. It is a cheap, easy and quick method of evaluating visual functions affected earlier in glaucoma. Glaucoma patients who have otherwise normal or near normal visual acuity on standard visual acuity charts have reduced contrast sensitivity and stereopsis due to progressive damage because of glaucoma.

These tests are not done on regular basis on every eye in Pakistan. So, it should be a part of regular eye examination .These are neither complicated nor difficult tests to perform and conduct. This will help a lot in reducing disease burden and preserving sight**.**

References

1. Chan MP, Broadway DC, Khawaja AP, Yip JL, Garway-Health DF, Burr JM, Luben R, Hayat S, Dalzell N, Khwa KT, Foster PJ. Glaucoma and intraocular pressure in EPIC-Norfolk Eye Study: cross sectional study. Bmj. 2017 Sep 13; 358: j3889.
2. Kapetanakis VV, Chan MP, Foster PJ, Cook DG, Owen CG, Rudnicka AR. Global variations and time trends in the prevalence of primary open angle glaucoma (POAG): a systematic review and meta-analysis. British Journal of Ophthalmology. 2016 Jan 1; 100 (1):86-93.
3. Kanski JJ, Bowling B (Bradley), Niscgak KK, Pearson A. Clinical ophthalmology: a systematic approach

[Internet].Elsevier/ Saunders; 2011[cited 2017 Apr 15].
4. Bassi CJ, Galanis JC. Binocular visual impairment in glaucoma. Ophthalmology. Sep 1;98 (9):1406-11.
5. Pelli DG, Bex P. Measuring contrast sensitivity. Vision research. 2013 Sep 20; 90: 10-4.
6. Bambo MP, Ferrandez B, Güerri N, Fuertes I, Cameo B, Polo V, Larrosa JM, Garcia-Martin E. Evaluation of contrast sensitivity, chromatic vision, and reading ability in patients with primary open angle glaucoma. Journal of ophthalmology. 2016; 2016.
7. Gupta N, Krishnadev N, Hamstra SJ, Yücel YH. Depth perception deficits in glaucoma suspects. British journal of ophthalmology. 2006 Aug 1;90 (8):979-81.
8. Papaconstantinou D, Georgalas I, Kalantzis G, Karmiris E, Koutsandrea C, Diagourtas A, Ladas I, Georgopoulos G. Acquired color vision and visual field defects in patients with ocular hypertension and early glaucoma. Clinical ophthalmology (Auckland, NZ). 2009;3:251.
9. Richman J, Lorenzana LL, Lankaranian D, Dugar J, Mayer J, Wizov SS, Spaeth GL. Importance of visual acuity and contrast sensitivity in patients with glaucoma. Archives of Ophthalmology. 2010 Dec 1;128(12):1576-82.
10. Lahav K, Levkovitch-Verbin H, Belkin M, Glovinsky Y, Polat U. Reduced mesopic and photopic foveal contrast sensitivity in glaucoma. Archives of ophthalmology. 2011 Jan 10;129(1):16-22.
11. Green CM, Kearns LS, Wu J, Barbour JM, Wilkinson RM, Ring MA, Craig JE, Wong TL, Hewitt AW, Mackey DA. How significant is a family history of glaucoma? Experience from the Glaucoma Inheritance Study in Tasmania. Clinical & experimental ophthalmology. 2007 Dec;35(9):793-9.
12. Niwa Y, Muraki S, Naito F, Minamikawa T, Ohji M. Evaluation of acquired color vision deficiency in glaucoma using the Rabin cone contrast test. Investigative ophthalmology & visual science. 2014 Oct 1;55(10):6686-90.
13. Castelo-Branco M, Faria P, Forjaz V, Kozak LR, Azevedo H. Simultaneous comparison of relative damage to chromatic pathways in ocular hypertension and glaucoma: correlation with clinical measures. Investigative ophthalmology & visual science. 2004 Feb 1;45(2):499-505.
14. Lakshmanan Y, George RJ. Stereoacuity in mild, moderate and severe glaucoma. Ophthalmic and Physiological Optics. 2013 Mar 1;33(2):172-8.
15. Kwon M, Liu R, Patel BN, Girkin C. Slow Reading in Glaucoma: Is it due to the Shrinking Visual Span in Central Vision?. Investigative ophthalmology & visual sience. 2017 Nov 1; 58 (13): 5810-8.

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ORIGINAL ARTICLE

Impact of Early Non-Proliferative Diabetic Retinopathy on Contrast Sensitivity

Maryam Firdous[1], Halima Masud[2], Hina Sharif[3]

ABSTRACT

Purpose: This study was conducted to find out the impact of early Non-proliferative Diabetic Retinopathy (NPDR) on contrast sensitivity.

Material and Methods: A case-control study was conducted (including 32 cases and 64 controls) from August 2016-February 2017 at General OPD of Al-Shifa Trust Eye Hospital (ASTEH), Jhelum Road, Rawalpindi.

After careful visual acuity testing and anterior segment ocular examination; all the cases and controls were checked for their contrast sensitivity with Pelli-Robson contrast sensitivity test chart, followed by mydriatic fundus examination. Each patient was interviewed, thereafter, to fill the Questionnaire and questions were explained to them in detail.

Results: Results showed that mean letter contrast sensitivity of cases was 1.70 (SD=0.19) while that of controls was 2.01 (SD=0.12). Mean difference in contrast sensitivity values between cases and controls was 0.31. There was statistically significant difference between contrast sensitivity values of the two comparison groups (Eta^2=0.43 and p-value=0.001).

Conclusion: Contrast sensitivity in cases was well below that of controls, reason being DR. So, contrast sensitivity testing can be used as a potential screening tool in optometric practice for early Non-proliferative diabetic retinopathy; responsible for causing functional vision reduction while sparing visual acuity at early stages. *Al-Shifa Journal of Ophthalmology 2019; 15(1): 21-29.*

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Introduction:

Diabetic retinopathy (DR) or 'diabetic eye disease', is a condition related to long-standing and/or uncontrolled diabetes that damages the retina.[1] It is the major cause of visual impairment and blindness in people of middle and old age.[2] DR is ranked as fifth leading cause of visual impairment and blindness worldwide (4.8%)[3,4]; first four being Cataract (47.9%), Glaucoma (12.3%), Age-related macular degeneration (ARMD) (8.7%) and Corneal opacities.[3,4]

Globally, overall 93 million (34.6%) people with type-2 diabetes are affected by any type of DR, while prevalence of PDR, DME, and Vision threatening diabetic retinopathy (VTDR) is 17 million (6.96%), 21 million (6.81%) and 28 million (10.2%) respectively.[6] The major risk factors include prolonged and uncontrolled diabetes as well as hypertension.[4] Nearly

21% of the patients have DR when diagnosed with diabetes whereas above 60% of diabetics develop DR within twenty years of diagnosis, according to American Diabetes Association (ADA).[7] The risk of blindness in diabetics is 25 times greater as compared to non-diabetic individuals.[8] DR is highly prevalent in Pakistan. According to a study conducted in 2014, 56.9% of Pakistani population with type-2 diabetes is estimated to have DR.[7] According to a national survey on blindness in Pakistan (conducted in 2004-5), DR is considered as a fourth major cause of blindness, first three being Cataract, Corneal blindness and Cataract related causes like aphakia and PCO (Posterior Capsular Opacities).[9]

The risk of developing DR increases with increased duration of diabetes. Almost 40-45% of Americans have DR when diagnosed with diabetes, while majority of them are unaware of it(10). The cost associated with DR is estimated to be less in mild NPDR as compared to PDR(11). The overall average cost for screening and treating diabetic eye disease is $3190, reported in U.S.[12] Seventy two percent and forty two percent of the cases of Type-I diabetics eventually develop PDR and DME that require treatment and cost reaches up to $966 and $1118 respectively per person per year to save vision.[13-15]

Contrast sensitivity is the ability to differentiate minor changes in luminance between two regions, not separated by sharp boundaries.[16] It differs from visual acuity that is recognition of decreasing letter size on standard vision chart. DR is one of the ocular disorders that impacts contrast sensitivity, others being cataract, glaucoma, optic nerve & visual pathway defects. It may also be reduced due to uncorrected refractive errors, after refractive surgery and in some other common conditions like in dim lighting, fog or due to glare in bright sunlight. Reduced contrast can be improved and managed by prescribing special corrective lenses with a yellow filter.[17] Due to limitations in seeking early diagnosis and treatment of DR, there is a need for some quick, less technical and low-cost screening method to detect DR at an early stage to save sight and reduce disease burden. Contrast sensitivity testing can be used as a screening tool in optometric practice for DR.[18,24]

Contrast sensitivity testing can be used as a screening tool in optometric practice for diabetic retinopathy to ensure timely referral and treatment of disease so as to avoid complications and associated vision loss.[18,24] Secondly, low contrast sensitivity affects quality of life; people with abnormal contrast sensitivity find more difficulty in their daily tasks as compared to normal ones. Contrast sensitivity can be enhanced using yellow filters so as to improve the quality of life and enhance the performance of daily life activities in patients with reduced contrast.[17] The overall aim of the study was to find out the impact of early NPDR on contrast sensitivity, if any.

Materials and Methods:

A hospital-based case-control study was conducted from August 2016-February 2017 at General OPD of Al-Shifa Trust Eye Hospital (ASTEH), Jhelum Road, Rawalpindi, Pakistan. Sample size was calculated to be 31 by the following formula[25]

$$\frac{r+1}{r}\ \frac{SD^2\,(Z\beta+Z\alpha/2)^2}{d^2}$$

where; r = Ratio of control to cases ,r = 2),SD = Standard deviation i.e.; 0.21[19]

$Z\beta$ = Standard normal variate for power (For 80% power it is 0.84)

$Z\alpha/2$ = Standard normal variant for level of significance i.e.; 1.96 (constant)

d = Expected mean difference between cases and controls i.e.; 0.15

31 cases and double number of controls were taken for the study. Consecutive non-random sampling technique was used for cases while Purposive non-random sampling technique was used for controls. Cases are the patients with history of

diabetes (Type-I or Type-II Diabetes) for the duration of last 5 years having best-corrected visual acuity (BCVA) 6/9- 6/6. on slit-lamp fundus exam patients diagnosed with early Non-proliferative diabetic retinopathy changes and cataract of grade-I or less than grade- I according to LOCS system (Lens Opacification Classification System) were included. Controls are the non-diabetic patients having best-corrected visual acuity (BCVA) 6/6.On slit lamp examination having only cataract of grade-I or less than grade- I according to LOCS system (Lens Opacification Classification System) were considered as controls.

After taking complete history, visual acuity using Snellen visual acuity chart was tested by the researcher. Objective refraction was done by using autorefractometer and then completes subjective refraction with Snellen acuity chart using Trial frame and Trial box lenses was done along with pin-hole visual acuity in patients having visual acuity less than 6/6. All diabetics (with either type-1 or -2 diabetes) having best corrected visual acuity 6/9-6/6 were tested for their contrast sensitivity by performing Pelli-Robson contrast sensitivity test chart at one meter distance in well-illuminated room with patient wearing his/her best refractive correction and contrast sensitivity function was recorded. Slit-lamp biomicroscopy was conducted by an ophthalmologist to check anterior segment and then pupils of each patient were dilated by instilling Mydriacyl (Tropicamide 1%) eye drops, three drops; each at 10 minutes interval in both eyes. After 30 minutes, complete fundus examination of each patient was carried out on slit-lamp using 90-D lens. Those patients having non-proliferative diabetic retinopathy (NPDR) changes on slit-lamp exam were selected and informed consent was taken. In cases with bilateral NPDR, eye with more severe NPDR was included, while eye with NPDR was included if the other had PDR. Each patient was interviewed to fill the questionnaire; questions were explained in detail to all patients. Informed consent was taken from each participant. The study was conducted after the approval of Hospital's Ethical review board. Questionnaire was filled by asking questions to each control. To account for reduced contrast sensitivity due to cataract in cases included in the study, age-matched controls having same grade of cataract were included (i.e.; $\leq$ Grade-I according to Lens Opacification Classification System-LOCS). For cases without cataracts, same age-matched controls were included in the study.

Results:

A total of 32 cases and 64 controls were asked to participate in the study and all of them agreed. Response rate was 100%.

Socio demographic characteristics

The mean age of cases having diabetic retinopathy was 50.16 years (SD=8.97) ranging from 32 to 70 while mean age of controls was 49.78 years (SD=9.42) having a range of 32 to 72 years. There was no statistically significant difference between the age groups of the cases and controls (p-value=0.85) with calculated t-value of -0.190 (d.f.=64.91).

Medical and systemic history

All of the cases included in the study were diabetics with different time duration while controls were non-diabetics. The details of medical and systemic history of cases and controls are shown in table 1.

Ocular History

The common chief complaints of most of the patients included in the study were decreased vision either for near or distance(66% cases and 62% controls), itching, burning or watering of eyes (25% cases and 38% controls), diplopia, headache, photophobia, and lid swelling(4%, 6%, 4%, and 1% controls respectively). and referral from other hospital (25% cases). Some of the cases (7%) visited the hospital due to referral from either general physician or eye clinic for their poor eye condition. More details

of ocular history of the participants are given in table 2.

Visual Status

Visual acuity of both cases and controls without any spectacle correction was within a range of 6/6 to Finger counting (C.F.). After refractive correction, visual acuity of 6/6 to 6/9 was considered for the cases and 6/6 for the controls according to inclusion criteria of the study. Mean refractive correction of eye included in the study was -0.02 DS (Diopters Sphere) for the cases (SD=0.98) ranging from -3.00 to +1.50 DS, and for controls mean refractive correction was +0.18 DS (SD=0.87) ranging from -4.00 to +2.25 DS.

Ocular Examination

Ocular examination was carried out in each patient either case or control. In adnexal examination, more than half of the eyes included in the study were found to be normal whereas a small percentage was found to have abnormality of adnexa. There were no lenticular changes in 20.8 % of patients in both eyes examination, whereas lenticular changes of less than grade-1 and grade-1 were found in approximately 68 % and 12% of eyes respectively. Intraocular pressure (IOP) in almost all of the patients was found to be in normal range of 8-22mm of Hg (99%) except one eye of case in which IOP was above the normal range. In fundus examination, CDR (cup to disc ratio) of most of the patients was observed as normal i.e. 0.3 to 0.4 (in 96% of the patients) (Table 3). There was no significant finding in fundi of the controls, and all were found to be normal (100% of controls) while in cases; for right eye; 6.3% had normal fundi, 65.6% had non-proliferative diabetic retinopathy and for left eye; 81.3% had non-proliferative DR.

Contrast sensitivity

Mean letter contrast sensitivity values of eyes of controls included in the study was 2.01 (SD=0.12), whereas mean contrast sensitivity of the eyes of cases included in the study was 1.70 (SD=0.19).Mean difference in contrast sensitivity values between cases and controls was 0.4. Significantly large difference was observed between the contrast sensitivity values of the two groups, $p<0.005$. (Table4).

Table 1: Medical and Systemic History

Medical and Systemic History	**CASES**		**CONTROLS**		**TOTAL**	
	f n=32	**(%)**	**f n=64**	**(%)**	**F n=96**	**(%)**
Duration of diabetes						
5-15	27	84.4	---	---	27	28.1
16-25	5	15.6	---	---	5	5.2
Treatment for diabetes						
Yes	32	100	---	---	32	33.3
No	---	---	64	100	64	66.7
Type of treatment						
On medication	20	62.5	---	---	20	20.8
On insulin	12	37.5	---	---	12	12.5
Family history of diabetes						
Positive	25	78.1	16	25.0	41	42.7
Negative	7	21.9	48	75.0	55	57.3
Hypertension						
Positive	18	56.3	20	31.3	38	39.6
Negative	14	43.8	44	68.8	58	60.4
Systemic disease						
Positive	7	21.9	9	14.1	16	16.7
Negative	25	78.1	55	85.9	80	83.3

Table 2: Ocular History

Ocular History	CASES		CONTROLS		TOTAL	
	f n=32	(%)	F n=64	(%)	f n=96	(%)
Spectacle history						
Positive	18	56.3	36	56.3	54	56.3
Negative	14	43.8	28	43.8	42	43.8
Surgical history						
Positive	1	3.1	2	3.1	3	3.1
Negative	31	96.9	62	96.9	93	96.9
Reason for surgery						
Pterygium	---	---	1	1.6	1	1
Chalazion	---	---	1	1.6	1	1
Cataract	1	3.1%	---	---	1	1
Ocular trauma						
Positive	---	---	---	---	---	---
Negative	32	100	64	100	96	100

Table 3: Ocular Examination

Ocular Examination	CASES				CONTROLS			
	OD		OS		OD		OS	
	f n=32	(%)	f n=32	(%)	f n=64	(%)	f n=64	(%)
Adnexa								
Normal	26	81.3	26	81.3	42	65.6	43	67.2
Abnormal	6	18.8	6	18.8	24	34.4	21	32.8
Lens								
Clear	6	18.8	6	18.8	14	21.9	14	21.9
Cataract								
< Grade-1	21	65.6	23	71.9	44	688	44	68.8
Grade-1	5	15.6	1	3.1	6	9.4	6	9.4
IOP (mm of Hg)								
8-22	31	96.9	32	100	64	100	64	100
> 22	1	3.1	---	---	---	---	---	---
Disc (CDR)								
Up to 0.4	29	90.6	31	96.9	64	100	64	100
>0.4	---	---	---	---	---	---	---	---
No view	3	9.4	1	3.1	---	---	---	---

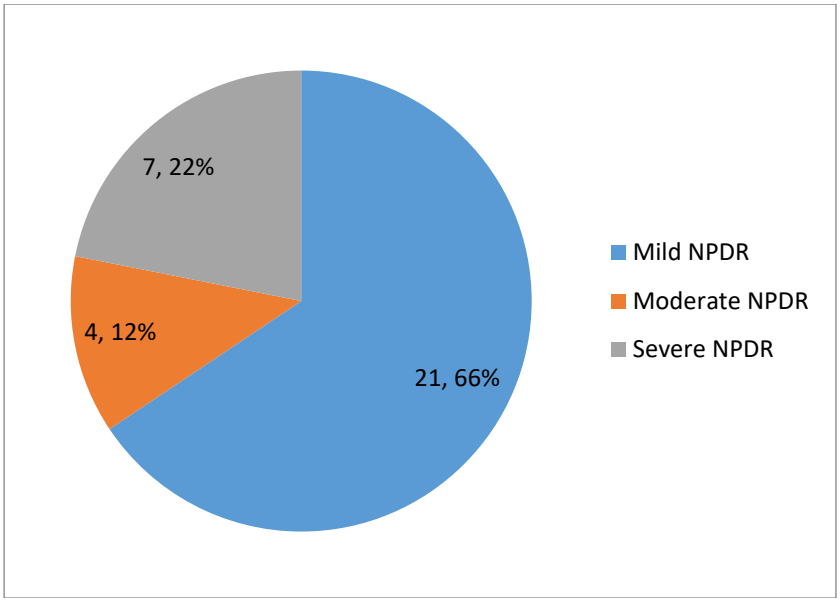

Figure 1: Non-proliferative diabetic retinopathy in cases

Table 4: Comparison of Contrast sensitivity between Cases and Controls

	N	Mean	SD	Mean difference	t-value	d.f.	p-value	Eta²
Cases	31	1.70	0.19	0.40	8.476	45.406	0.001	0.43
Controls	62	2.01	0.12					

Discussion:

Majority of the cases and controls landed the hospital either with symptoms of reduced vision related to cataract and/or refractive error (66% of cases and 63% of controls) or they had complaint of ocular redness, watering, itching and burning (25% of cases and 38% of controls), headache (0% of cases and 6% of controls). Almost 75% of the cases and controls were diagnosed with cataracts of variable density. Presentation with complaint of reduced vision associated to cataract is due to relatively high awareness of cataract-related loss of sight as compared to DR.[26]

DR due to its asymptomatic progressive nature needs to be diagnosed earlier. It is symptomatic only at later stages where most of the damage occurs to retina due to progression of disease. No doubt, cataract is a known cause of reduced visual acuity and it can be treated to restore normal or near-normal visual acuity.[27,28] In comparison, DR causes irreversible loss of sight if complicates or progresses to late stage.[3,4] Sight can be preserved only by diagnosing and treating the disease as early as possible. More importantly, diabetics should be made aware of the consequences of the disease through awareness programs. Only 25% of the cases were referred to the eye hospital to seek an eye exam or treatment for their eye disease by either General Physicians or Ophthalmologists. Many of these cases were having advanced complicated DR in their either eye, still they were unaware of their eye disease; the reason being lack of awareness.[10,29] This is an alarming situation and needs special attention. Contrast sensitivity was significantly reduced in all of the diabetic cases (p-value<0.005) in comparison to healthy age-matched controls where visual acuity was not significantly reduced. Results were found to be in agreement with the results of the other studies on measurement and comparison of grating or letter contrast sensitivity between diabetic

cases and healthy controls in developing countries like Iran.[19] which showed poor contrast sensitivity in cases with diabetes. Same was observed in developed countries like Glasgow[21], Canada,[22] England,[23] and Egypt[24]. DR is reported to be the underlying cause of reduced contrast sensitivity in diabetic cases. Mean difference of contrast sensitivity values between cases and controls was 0.31 which was approximately similar to the values (i.e; 0.21) of the study conducted in Australia.[18] In diabetic retinopathy, contrast sensitivity is reduced to abnormally low levels due to retinal ischemia (retinal non-perfusion) caused by retinal vascular occlusion. The retinal ischemia causes the changes at the level of retinal ganglion cell dendrites. Retinal ischemia causes loss of function at extremities (more distal portion) of the large dendritic branches of the ganglion cells before the damage to the smaller ones.[22] DR was found to have more adverse effect on contrast sensitivity than would be apparent from visual acuity levels in diabetic cases having early non-proliferative DR.[23,24]

Contrast sensitivity testing is not done regularly on each and every patient in eye hospitals, in Pakistan. So, it should be a part of regular protocol of eye examination. It is less time consuming and a simple test to perform; like visual acuity testing, it takes only a few minutes to test contrast sensitivity. Contrast sensitivity should also be evaluated in screening camps so as to refer any diseased cases to hospitals for proper eye check-up. It should also be corrected using appropriate yellow filters to enhance performance of daily life activities to improve task related quality of life. This will help enhancing much of the functions limited by reduced contrast sensitivity.[17] Although FFA (Fluorescein Fundus Angiography) is a gold standard method for diagnosing DR(30), still contrast sensitivity testing can be used as a screening tool for early DR in optometric practice.[18,24]

The study was carried out under strict eligibility criteria so there is a limitation in application of these findings on regular basis where these conditions are difficult to achieve. Like effect of other ocular conditions may also affect contrast as dense cataract and other retinal or optic nerve diseases. So, it is hard to get ideal conditions for using this instrument as a screening tool.

References:

1. Diabetic retinopathy. Diabetes.co.uk [Internet]. [cited 2016 Sep 6]; Available from: http://www.diabetes.co.uk/diabetes-complications/diabetic-retinopathy.html
2. Sivaprasad S, Gupta B, Crosby-Nwaobi R, Evans J. Prevalence of Diabetic Retinopathy in Various Ethnic Groups: A Worldwide Perspective. Surv Ophthalmol. 2012;57(4):347–70.
3. WHO Causes of blindness and visual impairment. WHO. 2010;
4. Lee R, Wong TY, Sabanayagam C. Epidemiology of diabetic retinopathy, diabetic macular edema and related vision loss. Eye Vis. 2015;2:17.
5. Roca JA. Table A6.1. 1995;(7):1995.
6. Yau JWY, Rogers SL, Kawasaki R, Lamoureux EL, Kowalski JW, Bek T, et al. Global Prevalence and Major Risk Factors of Diabetic Retinopathy. Diabetes Care. 2012;35(3):179–83.
7. P, C.R. AM, S. P. Prevalence of diabetic retinopathy among type 2 diabetes mellitus patients. Australas Med J. 2010;3(8):566–7.
8. Diabetic Retinopathy and Diabetes-Related Eye Problems [Videos] [Internet]. Available from: http://www.allaboutvision.com/conditions/diabetic.htm.
9. Jatoi S.M., Clinical Ophthalmology. 5th Edition. Pakistan: Paramount Book Publications; 2013
10. Facts About Diabetic Eye Disease | National Eye Institute. nei.nih.gov [Internet]. [cited 2016 Sep 6]; Available

from: https://nei.nih.gov/health/diabetic/retinopathy
11. Happich M, Reitberger U, Breitscheidel L, Ulbig M, Watkins J. The economic burden of diabetic retinopathy in Germany in 2002. Graefe's Arch Clin Exp Ophthalmol. 2007;246(1):151–9.
12. Javitt JC, Aiello LP, LJ M, GW B, GL K, M J, et al. Cost-Effectiveness of Detecting and Treating Diabetic Retinopathy. Ann Intern Med. 1996;124:164.
13. Javitt JC, Canner JK, Sommer A. Cost Effectiveness of Current Approaches to the Control of Retinopathy in Type I Diabetics. Ophthalmology. 1989;96(2):255–64.
14. Ibrahim OA, Foster A, Oluleye TS. Barriers to an effective diabetic retinopathy service in ibadan, nigeria (Sub -Saharan Africa) - a pilot qualitative study. Ann Ibadan Postgrad Med. 2015;13(1):36–43.
15. Yuan Z. Risk Factors and Barriers to Eye Care Services among Late Presenting Diabetic Retinopathy Patients in Shanxi Province in China. 2006;
16. Wikipedia. Contrast (Vision). Available from: https://en.wikipedia.org/wiki/Contrast_(vision)
17. Contrast Sensitivity Testing - AllAboutVision.com [Internet]. Available from: http://www.allaboutvision.com/eye-exam/contrast-sensitivity.htm
18. Stavrou EP, Wood JM. Letter contrast sensitivity changes in early diabetic retinopathy. Clin Exp Optom. 2003;86(3):152–6.
19. Abrishami M, Heravian J, Derakhshan A, Mousavi M, Banaee T, Daneshvar R, et al. Abnormal Cambridge low-contrast grating sensitivity results associated with diabetic retinopathy as a potential screening tool. East Mediterr Heal J. 2007;13(4):810–8.
20. Sokol S, Moskowitz A, Skarf B, Evans R, Molitch M, Senior B, et al. Contrast Sensitivity in Diabetics With and Without Background Retinopathy. Arch Ophthalmol. 1985;103(1):51–4.
21. Ghafour IM, Foulds WS, Allan D, McClure E. Contrast sensitivity in diabetic subjects with and without retinopathy. Br J Ophthalmol. 1982;66(8):492–5.
22. Regan D. Letter Charts. 1984;885–9.
23. Della Sala S, Bertoni G, Somazzi L, Stubbe F, Wilkins AJ. Impaired contrast sensitivity in diabetic patients with and without retinopathy: a new technique for rapid assessment. Br J Ophthalmol. 1985;69(2):136–42.
24. Sherif S. Karawya NMS. Impact of morphological changes of foveal avascular zone on contrast sensitivity in diabetic patients. Rawal Med J. 2009;34(1):51–3.
25. Charan J, Biswas T. How to calculate sample size for different study designs in medical research? Indian J Psychol Med. 2013;35(2):121–6.
26. Zhou JB, Guan HJ, Qu J, Yang XJ, Peng D, Gu HY. A study on the awareness of cataract disease and treatment options in patients who need surgery in a rural area of Eastern China. Europian H Ophthalmol. 2008 Jul;18(4):544-50.
27. What does Cataract treatment involve? How is cataract surgery done? [Internet]. [cited 2017 Feb 27]. Available from: http://www.rivercityvisioncenter.com/your-eye-health/eye-diseases/cataracts/cataract-surgery-treatment/
28. Facts About Cataract | National Eye Institute [Internet]. [cited 2017 Feb 27]. Available from: https://nei.nih.gov/health/cataract/cataract_facts
29. Murugesan N, Snehalatha C, Shobhana R, Roglic G, Ramachandran A. Awareness about diabetes and its complications in the general and diabetic population in a city in southern

India. Diabetes research and clinical practice. 2007 ;77(3):433-7.
30. Salz DA, Witkin AJ. Imaging in diabetic retinopathy. Middle East Afr J Ophthalmol. 2015;22(2):145–50.

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ORIGINAL ARTICLE

Microbial Etiology of Neonatal Conjunctivitis in A General Hospital in Saudi Arabia

Ali Faraz[1a], Muhammad Imran Ali[1b], Muhammad Asad Farhan[1c], Salahuddin Balooch[2]

ABSTRACT

Objective: To determine the microbial etiology of ophthalmia neonatorum in our setup.

Study design: Observational/cross sectional study

Place and duration of study: King Khalid hospital, Majmaah, Saudi Arabia from January 2016 to January 2018

Materials and Methods: Conjunctival swab specimens were collected from Neonates with suspected conjunctival inflammation and sent to the microbiology laboratory. The specimens were subjected to routine identification procedures in the laboratory.

Results: Of the 134 specimens, 81 were positive for microbial growth. The most commonly isolated microorganism were gram negative enterobacteriaceae, (n=26,32 %); *Enterobacter cloacae* was the most common gram negative enterobacteria (n=13, 16%) followed by *Escherichia coli* (n=11,13.5%).Coagulase negative *staphylococci* were the next most common etiological agent of acute neonatal conjunctivitis (n=18, 22%). 53 samples yielded no growth despite showing clinical signs and symptoms.

Conclusion: Ophthalmia neonatorum is a common infection occurring in the newborn; various gram negative enterobacteriaceae as well as gram positive cocci are common etiological agents. *Al-Shifa Journal of Ophthalmology 2019; 15(1): 30-34. © Al-Shifa Trust Eye Hospital, Rawalpindi, Pakistan.*

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Introduction:

Neonatal conjunctivitis or ophthalmia neonatorum refers to conjunctivitis occurring during first month of life. It is a common infection among the neonates[1]. In majority of the cases, bacteria are the causative agents; however, viruses as well as chemical agents like topical silver nitrate may also lead to this condition[2]. The usual clinical sign is the red watery eyes [3].The conjunctival discharge is mucoid or purulent depending upon the etiology. Although it usually runs a benign self-limited course, complications such as, systemic progression and corneal perforation may lead to blindness if left untreated.[4]

In medieval England, there were facilities devoted entirely to the eye lavage of babies with ophthalmia neonatorum. Crede developed topical 2 % silver nitrate in 1881, as prophylaxis to prevent this

infection in neonates. Routine use of topical drops coupled with delivery of intrapartum antibiotic treatment to mothers with positive high vaginal swabs has led to significant reduction in the incidence of this once very common infection in neonates.[5]
However, Conjunctivitis among neonates still remains a common infection in third world countries and places with no effective antenatal screening and topical prophylaxis. The prevalence rate worldwide ranges between 1-24% with high infection rate in Africa where about 4000 infants become blind annually due to conjunctivitis.[6]
The bacteria most commonly implicated are the *Neisseria gonorrhea* and *chlamydia trachomatis* however many gram positive bacteria (e.g. *Streptococcus pneumonia, Staphylococcus aureus* and coagulase negative *staphylococcus*)and gram negative bacteria (e.g. *Escherichia coli, Klebsiella pneumoniae, Enterobacter cloacae, Heamophilus influenzae* and *Hemophilus para influenzae*) can cause this condition.[7]
In neonatal conjunctivitis the reservoir of infection is usually the mother and the route of acquisition in most cases is the passage through the birth canal ,however some may be acquired after birth.[8] While Sexually transmitted infections in mothers like Neisseria gonorrhea and chlamydia trachomatis are leading sources of neonatal conjunctivitis worldwide, in conservative countries like Saudi Arabia various gram negative enterobacteriaceae and gram positive cocci like *Staphylococcus aureus* , *Streptococcus pneumoniae* and coagulase negative *Staphylococcus* are the leading cause.[9]

The precise determination of microbiology of neonatal conjunctivitis cases will help improve empirical treatment, thereby controlling and preventing it. This study was carried out to identify microbial causes of conjunctivitis in neonates relative to our setup.

Materials and Methods:

This study was conducted at King Khalid hospital Majmaah, Saudi Arabia. The study period is 2 years (from January 2016 to January 2018). Cases were diagnosed based on clinical presentation. Name, age, sex and hospital numbers were recorded. Ethical approval was obtained .Specimens were collected by experienced staff from each neonate using sterile swabs and sent to the microbiology laboratory.

King Khalid hospital Majmaah received 134 specimens of conjunctival samples, Saudi Arabia during the study period. In the microbiology laboratory the conjunctival swabs were inoculated on the Blood, Chocolate and MacConkey's agars right away and incubated at 37 ^{o}C overnight. The isolated microorganisms were identified using routine procedures mainly utilizing the Microscan walk away automated system of bacterial identification as well as conventional methodology of gram stain and biochemical identification. Facilities for isolation and detection of *Chlamydia trachomatis* are not available in the laboratory so this organism was not considered in this study.

Results:

The microbiology laboratory of King Khalid hospital Majmaah received 134 specimens of conjunctival samples during the study period. The majority of the samples were received from the nursery ward, but some were also received from the maternity and eye ward. Bacterial cultures were positive in 81 patients (60 %), and negative in 53 (40%) patients despite showing clinical signs and symptoms of conjunctivitis (table 1)

Of the culture positive cases, bilateral involvement was seen in 20 % cases whereas unilateral involvement was observed in 79 % cases, 41(51 %) were male, and 40 (49%) were female (table 1).
The most common organisms recognized on cultures were gram negative

enterobacteriaceae (n=26,32.09 %) as shown in Figure 1. Out of these 26 gram negative enterobacteriaceae, there were 13 samples that grew *Enterobacter cloacae* (50%) and 11 samples that grew *Escherichia coli* (41%). *Klebsiella pneumonia, pontoea agglomerans and morganella moganii* were isolated from 1 sample each (3% each) (figure 2)

Coagulase negative *staphylococci* were the next most common etiological agent of acute neonatal conjunctivitis (n=18, 22%).Among other gram-positive cocci, the organisms isolated were *Staphylococcus aureus* (n=8, 9.87%), *streptococcus pneumoniae* (n=7,8.64%) and *Streptococcus viridans* (n=3,3.7%) and *Streptococcus agalactiae* (n=1,1.23%) (figure1). Other micro-organisms isolated include *Hemophilus influenzae* (n=7,8.64%), *Hemophilus parainfluenzae* (n=3,3.37%), *Acinetobacter species* (n=5,6.17%), *candida species* (n=3,3.37%) and *Stenotrophomonas maltophilia* (n=1,1.23%)(figure 1).

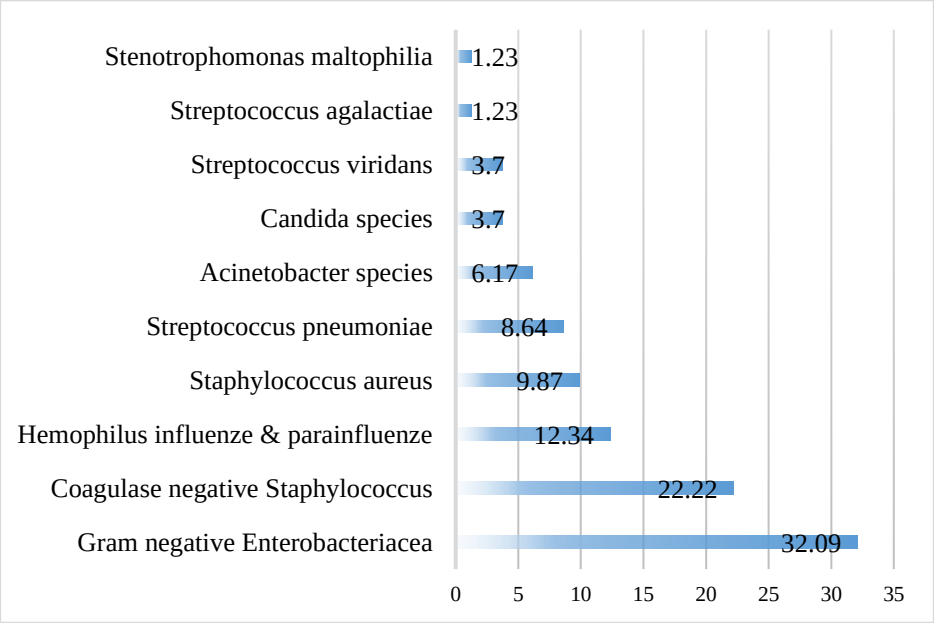

FIGURE 1. MICROBIAL ETIOLOGY OF CULTURE POSITIVE CONJUNCTIVAL SWABS (PERCENTAGE)

Table I: Demographic details of patients

Patient characters	**n(%age)**
Bacterial culture (n=134)	
Positive	81(60)
Negative	53(40)
Sex(culture positive)(n=81)	
Male	41(51%)
Female	40(49%)
Eye involvement (culture positive only)(n=81)	
Unilateral	n=64 (79%)
Bilateral	n=17 (20%)

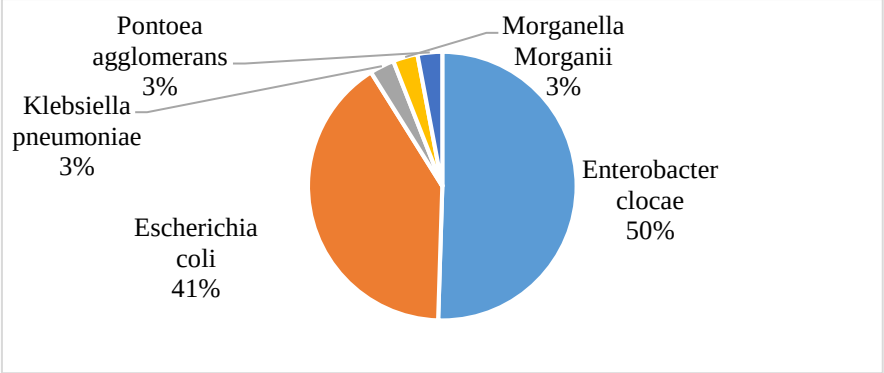

Figure 2: Percentage representation of Gram negative enterobacteriaceae

Discussion:

Neonatal conjunctivitis is a common infection worldwide. The global incidence of neonatal conjunctivitis varies with as low as 1% of newborns in developed countries to as high as 24% in developing countries particularly Africa.[10] The common source is the acquisition of infection from the vaginal canal. In the current study the incidence of neonatal conjunctivitis was 12 % which correlates with the global range of 1-24% of neonatal births.

Culture positive cases in our study were 60% of the patients with clinical signs & symptoms of conjunctivitis. In a report by Afjeiee et al., in which 80% of neonates with clinical conjunctivitis-were culture positive.[5] In another regional study done by Chowdhury MNH, in Saudi Arabia the culture positivity was 26%(n=107) out of total 400 samples sent from neonates with clinical conjunctivitis.[9]

In our study, the leading microorganisms producing neonatal conjunctivitis were gram negative enterobacteriaceae and coagulase negative *Staphylococcus*, respectively. In a report by Mallika P et al., *Chlamydia* and *gonococci* are the most common microbial organisms in the developed countries like USA and Europe.[11] A study conducted in 2 hospitals in Tehran by Afjeiee et al, the most commonly observed organisms were coagulase negative staphylococcus (53 %) and gram negative enterobacteriaceae(6.6%).[5] In a similar research by Chowdhury MNH in Saudi Arabia using a large sample size , the micro-organisms causing neonatal conjunctivitis were gram negative enterobacteriaceae, (48%), staphylococcus aureus (8.5%) and streptococcus pneumonia (3.5%), in order of decreasing frequency.[9] The result of these regional studies correlates well with our findings.

Enterobacter clocae and *Escherichea coli* were the most common enterobacteriaceae cultured from neonates with acute conjunctivitis. In the study conducted by Chowdhury MNH in Saudi Arabia, *Enterobacter species* and *klebsiella pneumonaie* were the commonest enterobacteriacea.[9] While S. epidermidis is a frequently isolated bacterial species in this study as well as many other studies,[5,12] the precise role of this particular organism in the etiology of neonatal conjunctivitis in newborn babies is not clear and many clinicians disregard it as normal flora .
Even though. Gonococcal ophthalmia neonatorum has a high incidence worldwide (US and Africa and Europe,[6,13] in our study no case of gonococcal infection were recognized using standard methods of isolation. This is also supported by Badeeb O et al's esearch[14] on neonatal conjunctivitis in Saudi Arabia, whereby no case of gonococcal ophthalmia was detected and only one case was isolated in a study by Chowdhury MNH. [9]

Facilities for isolation and detection of *Chlamydia trachomatous* are not available

in the laboratory so this organism was not considered in this study. Again, this organism has a high association with neonatal conjunctivitis worldwide, but a large study conducted in Saudi Arabia concluded that pregnant Saudi women have low prevalence rate of *Chlamydia trachomatis* IgG antibodies(8.7%) and lower prevalence for *Chlamydia trachomatis* IgM(1.5%).[15]

Conclusion:

The outcomes of this study clearly manifested considerably high frequency of neonatal conjunctivitis (12 %) in our set up with gram negative enterobacteriaceae being the leading cause.

References:

1. Wadhwani M, D'souza P, Jain R, Dutta R, Saili A, Singh A. Conjunctivitis in the newborn-a comparative study. Indian J Pathol. Microbiol. 2011;54(2):254–7.
2. Matejcek A, Goldman RD. Treatment and prevention of ophthalmia neonatorum. Can fam physician. 2013;59(11):1187-90.
3. Mansoor N, Mansoor T, Ahmed M. Eye pathologies in neonates. Int J Ophthalmol. 2016;9(12):1832-38
4. Moore DL, MacDonald NE, Canadian Paediatric Society, Infectious Diseases and Immunization Committee. Preventing ophthalmia neonatorum. Can J Infect Dis Med Microbiol. 2015;26(3):122-5.
5. Afjeiee SA, Tabatabaei SR, Fallah F, et al. A microbiological study of neonatal conjunctivitis in two hospitals in Tehran, Iran. Asian Pac J of Trop Dis. 2013;3(6):429-33.
6. Abdulsalam M, Ibrahim M, Asani M O. An overview of neonatal conjunctivitis. Sudan Med Monit 2015;10(3):91-8.
7. Epling J, Smucny J. Bacterial conjunctivitis. *Clin Evid*. 2005;2(14):756-61.
8. Silverstein BE, Morris TW, Gearinger LS, DeCory HH, Comstock TL. Besifloxacin ophthalmic suspension 0.6% in the treatment of bacterial conjunctivitis patients with *Pseudomonas aeruginosa* infections. Clin Ophthalmol. 2012; 6:1987–96.
9. MNH Chowdhury. Conjunctivitis in Riyadh, Saudi Arabia: A Bacteriological Study. Ann Saudi Med. 1987; 7(2): 111-4.
10. Klauss V. Newborn ophthalmia. Community Eye Health 1988;2:2-4
11. Mallika P, Asok T, Faisal H, Aziz S, Tan A, Intan G. Neonatal Conjunctivitis – a Review. Malays fam physician. 2008;3(2):77-81.
12. Mohile M, Deorari Ashok K, Satpathy G, Sharma A, Singh M. Microbiological study of neonatal conjunctivitis with special reference to *Chlamydia trachomatis*. Indian J Ophthalmol. 2002;50(4):295–99.
13. Darville T. Neisseria gonorhoeae. In: Behrman RE, Kliegman RM, Jenson HB, editors. Nelson textbook of pediatrics. Philadelphia: WB Saunders; 2008. pp. 1169–73.
14. Badeeb O, Basher E, Gazzaz F. Neonatal Conjunctivitis: A Pilot Study Bahrain medical bullet . 1993;15(3)-90-93.
15. Ghazi HO, Daghestani MH, Mohamed MF. Seropositivity of *chlamydia trachomatis* among saudi pregnant women in makkah. J Fam Community Med. 2006;13(2):61-64.

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ORIGINAL ARTICLE

Central Corneal Thickness and Pattern Standard Deviation in Patients with Primary Open Angle Glaucoma

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ABSTRACT

Purpose: To determine the frequency of thick and thin cornea in patients with primary open angle glaucoma (POAG) and correlate them with the visual field (VF) parameter, mean pattern standard deviation (PSD) in Pakistani adult patients.

Materials and Methods: A total of 97 eyes with POAG were consecutively recruited. Patients were labeled as glaucomatous based on VF and optic nerve head damage. All patients underwent Goldman applanation tonometry, Humphrey perimetry and measurement of CCT with non-contact pachymetry. Based on CCT value, the sample was divided in two groups (group 1, ≤ 535 mm, n=49; group 2 ≥ 535 mm, n=48).

Results: In the cohort, 51 (52.6%) males, 46 (47.4%) females, 50 (51.54%) right eyes and 47 (48.45%) left eyes were included. In Group 1, 49 (50.51%) eyes and in Group 2, 48 (49.48%) eyes were studied. PSD in group 1 and group 2 was 6.96 ± 1.49 dB and 4.60 ± 1.41 dB respectively. The mean CCT in Group 1 and Group 2 was 509.25 ± 13.05 µm and 571.3 ± 14.82 µm respectively. The difference in PSD between the two groups was statistically significant, $p \leq 0.05$.

Conclusion: A thinner CCT leads to an underestimation of intraocular pressure. POAG patients with thin corneas have significantly poorer VFs as compared to the ones with thick corneas. Evaluation of CCT is imperative in all patients with POAG so that thin corneas can be treated aggressively. *Al-Shifa Journal of Ophthalmology 2019; 15(1): 35-41. © Al-Shifa Trust Eye Hospital, Rawalpindi, Pakistan.*

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Introduction:

Glaucoma is the second leading cause of blindness worldwide. By the year 2020, it is anticipated that 80 million people will be living with glaucoma. [1] Primary open-angle glaucoma (POAG) is characterized by chronic, slowly progressive, optic neuropathy with characteristic patterns of optic nerve damage and visual field (VF) loss in the absence of ocular disease or congenital anomalies. *Quigley* estimated that out of 80 million persons living with glaucoma in 2020, 74% will have POAG. [3] An elevated intraocular pressure (IOP) is the focal risk factor associated with POAG; multiple other risk factors have been identified. [2] One of these factors, central corneal thickness (CCT) measurements has become of particular interest in glaucoma care after the ocular hypertension treatment study (OHTS). The aim of glaucoma

therapy is to stop or retard the progression of VF defects. The effect of glaucomatous neuropathy on visual function is assessed by the degree of damage shown on automated VF testing. An important parameter on VF test is humphrey global indices, particularly pattern standard deviation (PSD). [4]

Goldmann applanation tonometry (GAT) remains the gold standard for recording IOP in clinical practice.[5] CCT has a significant effect on IOP measured by applanation tonometry; thick cornea overestimate IOP and a thin one underestimates it. [6,7] Studies have shown that eyes with thinner CCTs tend to have more advanced visual field loss. This study aims to determine the relationship of CCT with mean PSD on Humphrey VF in POAG and endorse; GAT readings should be complimented with CCT measurements.

Materials and Methods:

The cross-sectional comparative study was conducted after approval by the hospital ethical committee. The study was conducted from June 2013 to March 2014 in the Ophthalmology Department of Al-Shifa Trust Eye Hospital, Rawalpindi. An informed written consent was taken from all the subjects.

In this descriptive case series 97 consecutive eyes with POAG using IOP lowering medications for at least 5 years were studied. All subjects had a normal cornea on slit-lamp exam and were phakic. VFs were assessed by a Humphrey Field Analyzer (HFA, USA), 24-2 SITA (Swedish Interactive Threshold Algorithm) standard program full threshold. We considered the Humphrey global indices PSD in this study. CCT was measured using an automated non-contact specular microscope (SP 2000P, TOPCON, USA). Each patient underwent a biomicroscopic examination, VF perimetry, CCT and IOP measurements.

Patients were classified as having POAG, when a characteristic abnormal optic nerve head (ONH) or typical damage on VF perimetry and open angle on gonioscopy were present. The Shaffer's gonioscopy grading system was used. The findings necessary to classify patients as having abnormal OHN were the optic rim notch, diffuse generalized loss of neuro-retinal rim, vertical cup/disc ratio (CDR) of > 0.7 and an asymmetry of > 0.3 and/or disc hemorrhage.

Glaucomatous VF defect was defined as two or more adjacent test locations demonstrating a threshold sensitivity loss on the pattern deviation plot with $P < 0.01$, three or more such adjacent test locations with $P < 0.05$ with at least one of the points depressed to $P < 0.01$ or a 10 dB difference across the nasal horizontal midline at two or more test locations. Patients were excluded when either VF testing was considered unreliable (false-negative and false positive responses ≥ 30% and fixation losses ≥ 20%). Patients with keratopathy, pseudoexfoliation, uveitis, pigment dispersion syndrome, retinal disease, previous refractive or intraocular surgeries, systemic diseases such as diabetes mellitus and hypertension and any grade of nuclear sclerosis as defined by Lens Opacities Classification System (LOCS) III were excluded.

All patients had former experience of VF examination and all fields were performed by the same perimetrist. The refractive error ranged from + 3 to - 3 diopters. Pachymetry values were obtained by an observer masked to the perimetry data. IOP was evaluated with GAT (Haag-Streit, Switzerland). All IOP measurements were taken whilst sitting with a topical anesthetic drop with fluorescein instilled in both eyes. Patients were asked not to move or blink their eyes and to continue breathing normally during measurements.

The entire cohort of eyes was divided in two groups based on CCT values. In group

1, the CCT value was ≤ 535 mm whereas in group 2 CCT value was ≥ 535 mm. All information was entered and analyzed in Statistical Package for Social Sciences (SPSS) version 19.0. Frequency and percentages were calculated for qualitative variables while mean, and standard deviation were calculated for quantitative variables. Independent sample t-test was used to compare PSD in both groups. P < 0.05 was considered as statistically significant.

Results:

Of the total 97 patients selected, 52.6% were males while females were 47.4%. The minimum age was 37 years and the maximum age was 60 years with a mean age of 52.03 ± 5.931 years. Right eye studied were 51.54 % and left eye were 48.45 %.

Out of the 97 eyes, 49 eyes had corneal thickness less than 535µm, 48 eyes had corneal thickness greater 535 µm. The percentages of thin and thick corneas were 50.51% and 49.48 % respectively. The minimum CCT in the entire sample was 480.97 µm and the maximum CCT was 598.13 µm with a mean CCT of 593.97 ± 34.15 µm. The mean CCT of Group 1 was 509.25 ± 13.05 µm and the mean CCT of Group 2 was 571.3 ± 14.82 µm.

The minimum PSD in the entire sample was 2.01 dB and the maximum PSD was 9.97 dB with a mean PSD of 5.79 ± 1.86 dB. The mean PSD of Group 1 was 6.96 ± 1.49 dB and of Group 2 was 4.60 ± 1.41 dB. The mean PSD amongst males was 5.44 ± 1.84 dB while the mean PSD among females was 6.18 ± 1.83 db.

Independent sample t-test was used to compare PSD in both groups. The calculated P-value of ≤ 0.05 revealed that there was significant difference between the two groups. PSD amongst males and females was not significant (P-value ≥ 0.05). Patients of POAG having thin corneas had worse VF damage as compared to the patients with thick corneas, while there was no statistically significant difference in PSD in terms of gender.

Table 1: CCT among groups

Group	N	Mean	Std. Deviation
CCT Group 1 Thin	49	509.25	13.05
CCT Group 2 Thick	48	571.34	14.82

Table 2: PSD among the sample

	N	Minimum	Maximum	Mean	Std. Deviation
Pattern Standard Deviation	97	2.01	9.97	5.79	1.86

Table 3: Pattern standard deviation in two groups

Pattern Standard Deviation among groups	N	Mean	Std. Deviation	Std. Error Mean	P-value
Group 1 Thin	49	6.96	1.49	0.21	<0.05
Group 2 Thick	48	4.60	1.41	0.20	

Table 4: Pattern standard deviation among gender

Pattern Standard Deviation among gender	N	Mean	Std. Deviation	Std. Error Mean	P-value
Male	51	5.44	1.84	0.25	>0.05
Female	46	6.18	1.83	0.27	

Discussion:

Glaucoma is a multifactorial disease principally caused by deterioration of axons in the optic nerve (ON) and the death of retinal ganglion cells (RGC). Optic neuropathy occurs as a result of damage to the ONH and it manifests as changes in VFs. It is unclear whether risk attributed to CCT is the product of inaccuracy in the measured IOP or reflects pathology in the posterior sclera and lamina cribrosa. The pressure gradient across the lamina sustained by the axon depends significantly on laminar thickness, IOP and retro-laminar pressure. If CCT, scleral and laminar thickness are related; a measurement of CCT can possibly provide a substitute guide of laminar thickness and a prospective useful measure in the overall risk assessment for glaucoma. The aim of this study was to assert the importance of CCT by relating it to mean PSD in POAG. Optical low coherence reflectometry represents the most accurate and objective pachymetry technique currently available with precision in order of 1 µm. The additional advantage is its non-contact feature. *Graefe* demonstrated that different CCT caused different readings of IOP both with GAT and non-contact tonometry (NCT). [8] *Whitacare* also found errors in IOP readings with varying CCT. [9] CCT readings also differ in various subgroups of glaucoma; people classified as having ocular hypertension have thicker corneas than controls whereas those with normal tension glaucoma have thinner corneas. [10]

The only modifiable risk factor in glaucoma is IOP. Lowering the IOP retards RGC apoptosis and glaucoma progression thus decreasing the risk of glaucomatous VF defects. OHTS was the first study to document prospectively that a thinner CCT measurement may predict the development of POAG. Subjects with ocular hypertension and thin corneas had a significantly higher risk of developing glaucoma; a decrease in CCT of 40 µm added a 70% increase in the risk. CCT was also a strong predictive factor for the development of POAG even after adjusting for the effects of baseline age, IOP, vertical CDRs and VF indices. [11] *Medeiros* reported that patients with thinner CCT have early glaucomatous VF damage even on frequency doubling perimetry. [12]

Jonas found that the amount of glaucomatous ON damage correlated significantly with a thin CCT however progression of glaucomatous optic neuropathy was independent of CCT. They suggested that patients with thin corneas are at a higher risk for delayed detection of glaucoma; however, they are not at a higher risk for progression of glaucoma. [13] Conversely *Kim* concluded that VF damage in patients with thinner CCT were more likely to progress than those with thicker CCT and CCT was the only risk factor identified to be significantly associated with VF progression. [14] We also found that patients with thin CCT had severe VF damage however we didn't prospectively review the patients to establish its implication on progression. In this study mean CCT in Group 1 and Group 2 was 509.25 ± 13.05 µm and 571.3 ± 14.82 µm respectively. PSD in Group 1 and 2 was 6.96 ± 1.49 dB and 4.60 ± 1.41 dB respectively ($P \leq 0.05$). Eyes with thin

corneas had significantly more VF damage than the eyes with thick corneas. *Papadia* also divided his cohort into 2 groups based on CCT (group 1 < 535 µm; group 2 > 535 µm). Results showed that patients with thinner corneas had worse PSD. [15] In contrast studies have found no statistically significant relationship between CCT and PSD. *Cao* studied 100 patients and found no relationship between CCT and VF loss in treated patients with POAG. [16] However this study found a statistically significant difference between patients having thick and thin cornea in terms of PSD.

Mokbel also concluded that thin CCT is associated with more deterioration of VF as compared to thick CCT in patients with POAG. [17] The mean PSD in his thick cornea group was 4.87 ± 1.28 dB while in our study it was 4.60±1.41 dB. The mean PSD in his thin cornea group was 5.65±0.39 dB while in our study it was 6.96±1.49 dB. *Wangsupadilok* found a significant correlation between CCT and PSD ($r = -0.288$, $p = 0.05$) in a recent study. [18] A study done on Hispanic population revealed that patients with POAG showed a statistically significant inverse correlation between CCT and the severity of the VF damage. They concluded that thinner corneas can be considered as a risk factor for the severity of VF loss in POAG. They also found no significant difference between males and females in terms of PSD. [19]

Herndon found that lower CCT was associated with worsened VFs, increased vertical & horizontal CDRs and increased number of glaucoma medications. They concluded that measuring CCT aids ophthalmologist in identifying glaucoma patients at high risk for damage and progression. [20] *Shah* evaluated 121 eyes over a period of 4 years and found a significant negative relationship between CCT and PSD (correlation coefficient: -0.02, $p < 0.05$). [21] *Rogers* also found that worse VF changes occur in patients having thin corneas ($p = 0.0019$). [22]

Kniestedt revealed that patients with thin CCT are more likely to be diagnosed at advanced stages of the disease; inferring that underestimating IOP by GAT could be one contributing factors. [23] *Rodriguez* concluded that patients classified as having advanced damage in their VF have significantly lower CCT measurements than patients classified as having initial or moderate damage. [24] *Meirelles* revealed that patients with severe VF loss showed lower CCT compared with patients with mild and moderate VF loss. [25] Since the nature of our study was cross sectional therefore, we couldn't directly asses the influence of CCT on the progression of glaucoma. Moreover, inclusion criteria narrowed the patient sample with conclusive glaucomatous damage, not assessing glaucoma suspects or patients diagnosed with ocular hypertension.

To sum up, thin corneas have got significant association with advanced VF damage. CCT should be measured in every patient with POAG, as underestimation can cause significant damage to VF in spite of treated IOP being within normal limits. CCT should be taken into account while setting the target IOPs. We stress the need of aggressive treatment and frequent follow-up of POAG patients having thin central corneas.

References:

1. Cedrone C, Mancino R, Cerulli A, Cesareo M, Nucci C. Epidemiology of primary glaucoma: prevalence, incidence, and blinding effects. Prog Brain Res 2008;173:3-14.
2. Leske MC. Open-angle glaucoma - an epidemiologic overview (review). Ophthalmic Epidemiol 2007;14:166-172.
3. Quigley HA, Broman AT. The number of people with glaucoma worldwide in 2010 and 2020. Br J Opthalmol. 2006:90:262-7.
4. Yaqub M. Visual fields interpretation in glaucoma: a focus on static automated

perimetry. Community Eye Health 2012;25:1-8.
5. Kotecha A, White ET, Shewry JM, Garway-Heath DF. The relative effects of corneal thickness and age on Goldmann applanation tonometry and dynamic contour tonometry. Br J Ophthalmol 2005;89:1572-75.
6. Gunvant P, Baskaran M, Vijaya L, Joseph IS, Watkins RJ, Nallapothula M, et al. Effect of corneal parameters on measurements using the pulsatile ocular blood flow tonograph and Goldmann applanation tonometer. Br J Ophthalmol 2004;88:518-22.
7. Kitsos G, Gartzios C, Asproudis I, Bagli E. Central corneal thickness in subjects with glaucoma and in normal individuals (with or without pseudoexfoliation syndrome). Clin Ophthalmol 2009;3:537-42.
8. Graef M. Zur Bedeutung der Hornhautdicke bei der non-contact-tonometrie. Kli Mbl Augenheilk 1991;199:183-86.
9. Whitacre MM, Stein R. Sources of error with Goldmann-type tonometres. Surv Ophthalmol 1993;38:1-30.
10. Copt R-P, Thomas R, Mermoud A. Corneal thickness in ocular hypertension, primary open angle glaucoma and normal tension glaucoma. Arch Ophthalmol 1999;117:14-16.
11. Brandt JD, Beiser JA, Gordon MO, Kass MA. The ocular hypertension treatment study (OHTS) group. Central corneal thickness and measured IOP response to topical ocular hypotensive medication in the ocular hypertension study. Am J Ophthalmol 2004; 138:717-22.
12. Medeiros FA, Sample PA, Weinreb RN. Corneal thickness measurements and frequency doubling technology perimetry abnormalities in ocular hypertensive eyes. Ophthalmology. 2003;110:1903-8.
13. Jonas JB, Stroux A, Velten I, Juenemann A, Martus P, Budde WM. Central corneal thickness correlated with glaucoma damage and rate of progression. Invest Ophthalmol Vis Sci 2005;46:1269-74.
14. Kim WJ, Chen PP. Central corneal pachymetry and visual field progression in patients with open-angle glaucoma. Ophthalmology 2004;111:2126-32.
15. Papadia M, Sofianos C, Iester M, Bricola G, Mete M, Traverso CE. Corneal thickness and visual field damage in glaucoma patients. Eye (Lond) 2007;21:943-7.
16. Cao KY, Kapasi M, Betchkal JA, Birt CM. Relationship between central corneal thickness and progression of visual field loss in patients with open-angle glaucoma. Can J Ophthalmol. 2012;47:155-8.
17. Mokbel TH, Ghanem AA. Correlation of central corneal thickness and optic nerve head topography in patients with primary open-angle glaucoma. Oman J Ophthalmol. 2010;3:75-80
18. Wangsupadilok B, Orapiriyakul L. Correlation between central corneal thickness and visual field defect, cup to disc ratio and retinal nerve fiber layer thickness in primary open-angle glaucoma patients. J Med Assoc Thai 2014;97:751-7.
19. Fernandez-Bahamonde JL, Roman-Rodriguez C, Fernandez-Ruiz MC. Central Corneal Thickness as a Predictor of Visual Field Loss in Primary Open Angle Glaucoma for a Hispanic Population. Semin Ophthalmol. 2011;26:28-32.
20. Herndon LW, Weizer JS, Stinnett SS. Central corneal thickness as a risk factor for advanced glaucoma damage. Arch Ophthalmol. 2004;122:17-21.
21. Shah H, Kniestedt C, Bostrom A, Stam per R, Lin S. Role of central corneal thickness on baseline parameters and progression of visual fields in open angle glaucoma. Eur J Ophthalmol 2007;4:545-9.
22. Rogers DL, Cantor RN, Catoira Y, Cantor LB, Dunn DW. Central corneal

thickness and visual field loss in fellow eyes of patients with open-angle glaucoma. Am J Ophthalmol 2007;143:159-61.
23. Kniestedt C, Lin S, Choe J, Nee M, Bostrom A, Sturmer J, et al. Correlation between intraocular pressure, central corneal thickness, stage of glaucoma, and demographic patient data: Prospective analysis of biophysical parameters in tertiary glaucoma. J Glaucoma 2006;15:91-97.
24. Jimenez-Rodriguez E, Lopez-De-Cobos M, Luque-Aranda R, Lopez-Egea-Bueno MA, Vazquez-Salvi AI, Garcia-Campos JM. Relationship between central corneal thickness, intraocular pressure and severity of glaucomatous visual field loss. Arch Soc Esp Oftalmol 2009;84:139-44.
25. Meirelles SH. Relationship between corneal thickness and severity of visual field loss in primary open-angle glaucoma. Arq Bras Oftalmol 2006;69:313-7.

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ORIGINAL ARTICLE

Dry Eye Disease in Postmenopausal Women

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ABSTRACT:

Objective: To determine the prevalence of dry eyes in post-menopausal women presenting to tertiary care Hospital in Peshawar.

Materials and Methods: Total of 200 post-menopausal women with menopause as cessation of menstruation from last one year with symptoms of dry eyes were included in the study. In all patients Schirmer test was performed using specialised litmus paper strip, placed in the lower eyelid for 5 minutes and the patient is asked to close the eyelids. After removal from the eye, the wetting of litmus paper was measured immediately. Value of more than 15 mm was considered normal, from 9–15 mild dryness,5 – 8 mm moderate dryness and <5 mm severe dryness.

Results:A total of 200 post-menopausal women were included in the study with mean age of 53.42 ±3.54 years and with a mean menopausal starting age of 42.56 ±4.37 years. Schirmer's test result showed the prevalence of dry eyes in 48% of the patients with mild dryness in 24%, moderate in 19.5% and severe dryness in 4.5% of the patients. Thirty two percent of the patients were having symptoms and some patients had more than one symptom for dry eyes with ocular discomfort as the most common symptom accounting for 23.5%.

Conclusion: Dry eye is a common disease in post-menopausal women therefore it is important for gynecologists and for primary care physicians to recognize symptoms and refer patients to ophthalmologists where applicable for proper management. *Al-Shifa Journal of Ophthalmology 2019; 15(1): 42-46. © Al-Shifa Trust Eye Hospital, Rawalpindi, Pakistan.*

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Introduction:

Dry eye disease is a common problem and is characterised by ocular discomfort, foreign body sensation, tear film instability, photophobia and decrease vision due to ocular surface damage[1].In addition to that there are chances of corneal ulceration, opacification and permanent visual loss.[2]

Postmenopausal women are more commonly affected by dry eye disease. It is a debilitating condition and greatly affects day to day activity and working capacity of the patient and is responsible for frequent visits of these patients to ophthalmologists[3]. Studies have shown that the prevalence of dry eyes over the age of 50 years is more common in women than men ranging from 7% to 4% respectively.[3]

The tear film has three major components – the aqueous layer secreted by the lacrimal gland, the lipid layer secreted by the meibomian glands, and mucin secreted by the conjunctival goblet cells. Both androgens and estrogen have known effects on the synthesis and components of the tear film. Sex steroid receptors are present on the meibomian glands, which are the sebaceous glands on the eyelids responsible for producing the oil component of tears that prevents evaporation.[4] Androgen binding results in synthesis and secretion of lipids from these glands, while estrogens actually cause a decrease in lipid production.[5] For this reason, increased levels of estradiol are believed to be a risk factor for dry eye.

Various Studies have shown the presence of oestrogen and progesterone receptor mRNA in the lacrimal and meibomian glands, conjunctiva, cornea and the lids. The presence of these hormones in ocular structures and the prevalence of dry eyes in post-menopausal women suggest that the tear film function is under the influence of complex hormonal system.[3] A reduction in these hormones production in post-menopausal women results in inflammation of lacrimal glands and subsequently affecting tear production.[5]

Purpose of our study is to know about the prevalence of dry eyes in post-menopausal women in our community so as to make a coalition between gynaecologists and ophthalmologists to diagnose and manage dry eyes in this age group as early diagnosis and treatment of this condition is important for improving life style of the patient.

Materials and Methods:

It was a cross sectional study conducted at outpatient department of Obstetrics and gynaecology, Hayatabad Medical Complex, Peshawar from March 2018 to February 2019. Total of 200 post-menopausal women were included in the study by convenient sampling methods. All post-menopausal women with menopause as cessation of menstruation from last one year with symptoms of dry eyes were included in the study.

Patients with other co morbidities like hypertension, diabetes, hypothyroidism, autoimmune diseases, local ocular conditions causing dry eye, ocular infections with corneal or conjunctival pathology, contact lens users, patients who underwent ocular surgery in the last 6 months before presentation were excluded from the study to avoid bias in the study results.

In all the patients Schirmer test was performed using specialised litmus paper strip, placed in the lower eyelid for 5 minutes and the patient is asked to close the eyelids. After removal from the eye, the wetting of litmus paper was measured immediately. To check the basal secretion of tears and to avoid the reflex secretion of tears a local anaesthetic drop is used 5 minutes before the procedure. Value of more than 15 mm was considered normal, from 9–15 mild dryness, 5 – 8 mm moderate dryness and <5 mm severe dryness.

All the analysis was done in SPSS version 20.0. For categorical variables like gender, frequencies and percentages were calculated. For numeric variables like age, mean ± standard deviation was calculated. All the results were presented in the form of tables.

Results:

A total of 200 post-menopausal women were included in the study with mean age of 53.42 $\pm$3.54 years and with a mean menopausal starting age of 42.56 $\pm$4.37 years. Age distribution is shown in table 1. Post-menopausal symptoms of patients with which they presented to the gynaecologists included hot flushes, night sweats, insomnia, urinary urgency and weight gain. Most patients had more than

one symptom. Frequency of symptoms is shown in table 2.

Ocular symptoms of dry eyes were present in majority of patients and included foreign body sensation, ocular discomfort, irritation of eyes, blurred vision and ocular fatigue. Some patients had more than one symptom with ocular discomfort being the most common symptom. Frequency of different symptoms of dry eyes is shown in table 3.

Schirmer's test result showed the prevalence of dry eyes in 98 (48%) of the patients while 102 (52%) of patients had normal result with value >15mm. Mild dryness with Schirmer value 9-15 mm was found in 23 (24%) of patients, moderate dryness with value 5-8 mm in 39 (19.5%) of patients and severe dryness with value <5mm in 9 (4.5%) of the patients as shown in table 4.

Table 1: Distribution of study population according to age

Age group	Number of patients	Percentage of patients
41 – 45 years	23	11.50%
46 – 50 years	47	23.50%
51 – 55 years	43	21.50%
56 – 60 years	34	17.00%
> 60 years	53	26.50%
Total	200	100

Table 2: Postmenopausal symptoms

Hot flushes	87	43.5%
Night sweats	42	21%
Insomnia	47	23.5%
Urinary urgency	35	17.5%
Weight gain	31	15.5%

Table 3: Ocular symptoms of dryness

Symptoms	Number of patients	Percentage
Foreign body sensation	38	19.00%
Ocular discomfort	47	23.50%
Irritation of eyes	34	17.00%
Blurring of vision	38	19.00%
Ocular fatigue	42	21.00%

Table 4: Schirmer test result

Schirmer test result	Dryness of eyes	Number of patients	Percentage
>15 mm	No dryness	104	52.00%
9 – 15 mm	Mild dryness	48	24.00%
5 – 8 mm	Moderate dryness	39	19.50%
<5 mm	Severe dryness	09	4.50%
Total		200	100

Discussion:

Dry eyes is a very debilitating condition mostly affecting post-menopausal women.Sex hormones i.e. estrogens and androgens are involved in the production of different components of the tear film including aqueous layer, mucin layer and lipid layer.[6] Various mechanisms play their role for manifestation of dry eyes in post-menopausal women including decrease in

hormonal levels, alteration in feedback mechanisms, and changes in receptor response.[7]

Several studies have proved that dry eyes in postmenopausal women are due to imbalance of estrogen and progesterone at this age. Versura P[4] proposed that reduce level of estrogen and progesterone were responsible for severe dry eyes in post-menopausal age. GolebiowskiB et[8] al in their study showed that serum oestrogen appears to be a key factor in meibomian gland dysfunction in post-menopausal women. They proposed that estrogen plays a role through its effect on meibum secretion and hence results in dry eye symptoms in non-Sjögren's dry eye in postmenopausal women.

Peck T et al[5] showed in their study that post-menopausal patients presented with symptoms of burning, dryness, tearing, blurred vision, and photophobia. They also suggested that medical history, surgical history, concurrent medications, environmental exposures to allergens, smoking, etc., need to be evaluated to identify potential contributing factors. Matossian C[9] et al also showed that dry eyes is the major disease in post-menopausal women for ocular discomfort, pain, and visual disturbance.In our study the presenting complaints of patients were foreign body sensation, ocular discomfort, irritation of eyes, blurring of vision and ocular fatigue. Majority of patients have multiple complaints with foreign body sensation being the most common symptom.

Our study results revealed the presence of eye dryness in 48% of post-menopausal patients and severe dryness in 4.5% of the patients. Studies have reported the prevalence of dryness from 14.4% to 73.5% by Moss[10] et al and Unicho M et al[11]. It is found in our study that the symptoms of dry eyes increase with increasing post-menopausal age. This direct relationship of dry eyes symptoms with increasing post-menopausal age is consistent with the study results of Adam et al[12], who reported that there is a significant relationship between dry eyes and increasing age.

In our study 32% of the post-menopausal women were having symptoms of dry eyes, similar results were also derived by Lin PY in his study who reported the prevalence of dry eyes symptoms in 33.7% of the patients[13]. Most of these patients were having more than one symptom of dry eye disease.

It is suggested that hormone replacement therapy in post-menopausal women improves the ocular complaints by increasing tear production. Jenson et al reported that post-menopausal women taking hormone replacement therapy for more than five years have significantly lower ocular complaints and increased tear production than those taking these medicine for less than five years showing a definitive role of hormonal imbalance in the pathogenesis of dry eye at this age[14].

Conclusion:

Dry eye is a common disease in post-menopausal women therefore it is important for gynaecologists and for primary care physicians to recognize symptoms and refer patients to ophthalmologists where applicable for proper management. Early diagnosis and management could significantly improve a woman's quality of life.

References:

1. Dorennavar L, Maurya RP, Singh VP, Singh MK, Sharma K. The role of Rebamipide ophthalmic suspension in management of dry eye disease. Indian J ClinExpOphthalmol. 2015;1(4):191-6.
2. Kumar GV, Praneetha G, Pandharpurkar A, Prasand B, Pavani G, Vasavi T et al. A study on prevalence of dry eyes among menopausal women

attending a tertiary care centre in Hyderabad Telangana. Int J Community Med Public Health. 2019;6(1):423-7.
3. Pujari MR, Salagar K, Bagare SN. Prevalence of dry eyes in post-menopausal women. J of evolution of Med and Dent Sci. 2015:75(4):13005-10.
4. Versura P, Giannaccare G, Campos EC. Sex-steroid imbalance in females and dry eye. Curr Eye Res. 2015; 40:162–75.
5. Peck T, Olsakovsky L, Aggarrwal S. Dry eye syndrome in menopause and perimenopausal age group. J Mid-life Health 2017; 8:51-4.
6. Milner MS, Beckman KA, Luchs JI, Allen QB, Awdeh RM, Berdahl J, et al. Dysfunctional tear syndrome: Dry eye disease and associated tear film disorders – New strategies for diagnosis and treatment. CurrOpinOphthalmol. 2017;27(Suppl 1):3–47.
7. Milner MS, Beckman KA, Luchs JI, Allen QB, Awdeh RM, Berdahl J. Dysfunctional tear syndrome: Dry eye disease and associated tear film disorders – New strategies for diagnosis and treatment. CurrOpinOphthalmol. 2017; 27:3–47.
8. Golebiowski B, Badarudin N, Eden J, You J, Hampel U, Stapleton F.Does endogenous serum oestrogen play a role in meibomian gland dysfunction in postmenopausal women with dry eye? Br J Ophthalmol. 2017;101:218-222.
9. Matossian C, McDonald M, Donaldson KE, Nichols KK, MacIver S, Gupta PK. Dry Eye Disease: Consideration for Women's Health. J Womens Health. 2019;28(4):502-514.
10. Moss SE, Klein R, Klein BEK. Prevalence of and Risk Factors for Dry Eye Syndrome. Arch Ophthalmol. 2000;118(9):1264-8.
11. Uchino M, Dogru M, Yagi Y. The features of dry eye disease in a Japanese elderly population. Optom Vis Sci. 2006; 83:797–802.
12. Adam J, Paulsen JC, Mary EF, Guan-Hua H, Barbara EK, Klein R et al. Dry Eye in the Beaver Dam Offspring Study: Prevalence, Risk Factors, and Health-Related Quality of Life. Am J Ophthalmol. 2014:157(4);799-806.
13. Lin PY, Tsai SY, Cheng CY, Liu JH, Chou P, Hsu WM. Prevalence of dry eye among an elderly Chinese population in Taiwan: the Shihpai Eye Study. Ophthalmology. 2003;110(6):1096-101.
14. Jensen AA, Higginbotham EJ, Guzinski GM, Davis IL, Ellish NJ. A survey of ocular complaints in postmenopausal women. J Assoc Acad Minor Phys. 2000; 11:44–9.

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CASE REPORT

Pseudocalcification: A Rare type of Intraocular lens Opacification

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Abstract

Intraocular lens (IOL) opacification is a rare complication of IOL implantation after cataract surgery. It can occur because of inherent disparities in biochemical properties of eye and IOL. Current report is of a case of pseudocalcification which is a rare form of IOL opacification. A 65-year-old male with pseudophakic bullous keratopathy underwent penetrating keratoplasty. After keratoplasty sub optimal improvement was observed in visual status which was attributed to distinctive opacification of the IOL. The opacified IOL was replaced with another IOL which resulted in the significant improvement in visual status. *Al-Shifa Journal of Ophthalmology 2019; 15(1): 47-50. © Al-Shifa Trust Eye Hospital, Rawalpindi, Pakistan.*

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Introduction:

Intraocular lens (IOL) implantation is one of the most indispensable parts of cataract surgery, the most commonly performed ophthalmologic surgery of modern times.[1] Since the accidental discoveries of its benefits for visual functions.[2] It has practically become the biggest weapon used against cataract related blindness worldwide. Over the years, cataract surgery has evolved in Pakistan with improving outcomes.[3-5] Currently, many advanced techniques and variations of cataract extraction surgeries are being practiced in Pakistan to improve visual functions, prognosis and quality of life of patients.[6-9] Initially the IOLs were manufactured through hard synthetic materials like PMMA. Later with the increasing variety of biosynthetic materials based foldable IOLs were introduced which revolutionized the world of cataract surgery. Currently we have IOLs available in both hydrophilic and hydrophobic materials, in meniscus and non-meniscus designs, monofocal and multifocal variety and variable insertion position within eye.[10]

Interestingly after so much development we are still unable to find the perfect IOL. At one hand Hydrophilic materials have poor capsular compatibility and on the other

hand hydrophilic materials show poor uveal biocompatibility.[11] Silicon decreases these issues but is difficult to implant through microincisions because of the high rigidity.[10] Intraocular lens opacification is an IOL based problem which is often misdiagnosed and treated as Posterior capsular opacification. It is usually caused because of some inherent biochemical disparities between IOL composition and intraocular environment.[12, 13]Current report is a case with late IOL opacification which according to the best of our knowledge is the first of its sort from Pakistan.

Case Report:

A 65 years old male presented with complaint of reduced vision and pain in right eye (RE) for more than a year. He had a cataract extraction with IOL implantation in RE. He was not using any ocular medication at that time. He had a positive history of hypertension and was taking treatment for it. The patient had a corrected visual acuity of not more than finger counting at 2 meters. On examination he had decompensated cornea with epithelial bullae. There was no clear view posterior to cornea. B-Scan Ultrasonography was done which showed normal posterior segment and flat retina. The patient was advised Penetrating -Keratoplasty (PKP) and was operated afterwards. Unexpectedly, his visual acuity improved only up to 6/36 in subsequent follow ups and a peculiar haze was seen in IOL which was different from the usual posterior capsular opacification. Six months after the Keratoplasty IOL removal and exchange was carried out. Consequently, corrected visual acuity improved to the level of 6/18 one month after IOL exchange. The secondary IOL is clear and is functioning optimally.

Chemical analysis was conducted for determining chemical properties of the deposits and IOL. Laboratory testing was unable to specify the material found in deposits. However, IOL showed significant hydrophilic properties inherently.

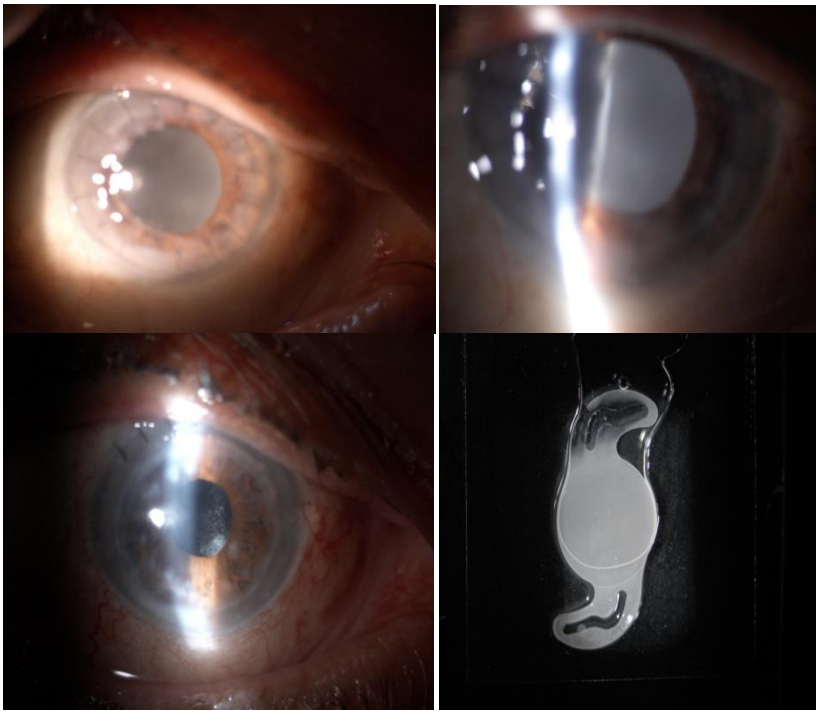

Figure 1a and 1b: IOL opacification seen after PKP
Figure 1c: Clear IOL after IOL replacement
Figure 1d: Explanted opacified IOL

Discussion:

One of the very first report of IOL discoloration was reported in early 90s in silicon based IOLs. The report described central haze with brown discoloration.[14] Since then multiple authors have reported opacification of IOL in lenses of different composition.[12, 13, 15] Usually these cases have been linked with calcification of lenses either on the surface or within the lens. In fact, a classification was presented to help determine the possible pathogenesis of opacified IOL. Based on this classification pseudocalcification was seen in cases that have been diagnosed clinically as opacified because of calcium but chemical testing proved otherwise.[16]. This was also seen in our case as biochemical testing proved no signs of calcium within lens deposits.

Opacification of IOL has also been associated with keratoplasties involving intracameral air injection. The combined mechanism includes an inflammatory reaction of anterior chamber, dehydration of IOL surface and disturbance of blood aqueous barrier due to underlying corneal pathology (Endothelial dysfunction). [17]
In the current case we were able to observe the opacification only after keratoplasty, so it is difficult to determine any temporal relationship between IOL opacification and keratoplasty. Nevertheless, if it is assumed that the opacification had some relationship with PKP, this becomes a relatively new scenario and needs to be further explored as none of studies according to the best of our knowledge have reported any cases in which IOL opacification has been reported in association with PKP.

It is concluded that IOL opacification is a rare phenomenon but should be considered in non-conventional cases of posterior capsular opacification. It should also be considered in certain ocular procedures involving intracameral air injection into the anterior segment especially lamellar corneal grafting procedures.

The authors recommend further studies in understanding biochemical composition of pseudocalcification based IOL opacification to determine risk factors or predictors associated with IOL composition and internal environment of eye as it will help in assessing all the cases pre operatively to avoid such complications making IOL implantation an ever-safe procedure for everyone, achieving the dream of "Right to Sight".

References:

1. Erie JC, Baratz KH, Hodge DO, Schleck CD, Burke JP. Incidence of cataract surgery from 1980 through 2004: 25-year population-based study. J Cataract Refract Surg. 2007;33:1273–1277.
2. Pannu RS. Intra-ocular lens. [Internet]. Vol. 93, Journal of the Indian Medical Association. 1995 [cited 2018 Sep 25]. p. 399.
3. Malik AR, Qazi ZA, Gilbert C. Visual outcome after high volume cataract surgery in Pakistan [Internet]. Vol. 87, British Journal of Ophthalmology. 2003 [cited 2018 Sep 25]. p. 937–40.
4. Bourne R, Dineen B, Jadoon Z, Lee PS, Khan A, Johnson GJ, et al. Outcomes of cataract surgery in Pakistan: Results from the Pakistan National Blindness and Visual Impairment Survey. Br J Ophthalmol [Internet]. 2007 Apr [cited 2018 Sep 25];91(4):420–6.
5. Chhipa SA, Junejo MK. Outcomes of cataract surgery at teaching hospital in Karachi. J Pak Med Assoc [Internet]. 2018 [cited 2018 Sep 25];68(1):76–80.
6. S.A. K, M. A, A.M. A, M. I. Comparison of the efficacy of subtenon with peribulbar Local anesthesia without hyaluronidase in patients undergoing cataract surgery. J Coll Physicians Surg Pakistan [Internet]. 2014 [cited 2018 Sep 25];24(5):331–4.
7. Baig R, Chaudhry TA, Kukreja S, Shakil S, Ahmad K. Patients' satisfaction and spectacle independence after cataract surgery with multifocal

intraocular lens implantation in a tertiary care hospital. J Pak Med Assoc [Internet]. 2016 [cited 2018 Sep 25];66(6):745–7.
8. Latif K, Shakir M, Zafar S, Rizvi SF, Naz S. Outcomes of Congenital Cataract Surgery in a Tertiary Care Hospital. Pakistan J Ophthalmol Pak J Ophthalmol [Internet]. 2014 [cited 2018 Sep 25];30(1).
9. Khan MA, Malik AM, Niazi MK, Khan AG, Irum N. Efficacy of manual small incision cataract surgery for all types of cataracts. J Rawal Med Coll [Internet]. 2009 [cited 2018 Sep 25];13(2):70–2.
10. Bellucci R. An introduction to intraocular lenses: Material, optics, haptics, design and aberration. Cataract. 2013;3:38–55.
11. Özyol P, Özyol E, Karel F. Biocompatibility of intraocular lenses [Internet]. Vol. 47, Turk Oftalmoloiji Dergisi. Galenos Yayinevi; 2017 [cited 2018 Sep 26]. p. 221–5.
12. Goodall KL, Ghosh YK. Total Opacification of Intraocular Lens Implant After Uncomplicated Cataract Surgery: A Case Series. Arch Ophthalmol [Internet]. 2004 May 1 [cited 2018 Sep 26];122(5):782.
13. Ronge laura. Opacification of IOLs: A Problem Often Misdiagnosed? - American Academy of Ophthalmology [Internet]. [cited 2018 Sep 26].
14. Milauskas AT. Silicone Intraocular Lens Implant Discoloration in Humans [Internet]. Vol. 109, Archives of Ophthalmology. American Medical Association; 1991 [cited 2018 Oct 3]. p. 913.
15. Srinivasan S. Intraocular lens opacification: What have we learned so far [Internet]. Vol. 44, Journal of Cataract and Refractive Surgery. Elsevier; 2018 [cited 2018 Oct 3]. p. 1301–2.
16. Neuhann IM, Kleinmann G, Apple DJ. A New Classification of Calcification of Intraocular Lenses. Ophthalmology [Internet]. 2008 Jan 1 [cited 2018 Oct 3];115(1):73–9.
17. Giers BC, Tandogan T, Auffarth GU, Choi CY, Auerbach FN, Sel S, et al. Hydrophilic intraocular lens opacification after posterior lamellar keratoplasty - A material analysis with special reference to optical quality assessment. BMC Ophthalmol [Internet]. 2017 Dec 22 [cited 2018 Oct 3];17(1):150.

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