Study of Developing Dry Eye After Cataract Surgery in MGD Patients

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Abstract

Introduction: Many patients have complained of dry eye and symptoms of irritations after cataract surgery, and it has been shown that both the incidence and severity of dry eye use increase. In particular, the reduction in tear break-up time (TBUT) and squamous metaplasia in conjunctival impression cytology were documented after phacoemulsification.

Objectives: To investigate the dry eye symptoms after cataract surgery in MGD patients and their relationships.

Methods: This prospective observational study was conducted at the Regional Institute of Ophthalmology, Sitapur. Demographic data of 50 enrolled patients will be collected after obtaining their written informed consent. All patients underwentuncomplicated cataract surgery with age-related cataracts. In the study, patients were divided into two groups according to MGD diagnostic criteria: Group A (MGD group) and group B (control group). Shirmer 1 test (ST1), tear break up time (TBUT), and control fluoresce staining (CSF) were performed preoperatively and at 3,7, 14, and 30 days postoperatively also measured eyelid meibomian gland morphology, meibomian gland expression, and meibomian character scores before and after cataract surgery.

Keywords: Cataract surgery, Dry eye disease, Prevalence, Risk factors, Systematic review, Meibomian gland dysfunction.

INTRODUCTION

This systematic review and meta-analysis comprehensively present DED prevalence after cataract surgery. We observed that 37.4% (95% CI 22.6–52.3; 206/775) of patients without preexisting DED developed DED postoperatively, highlighting the importance of perioperative DED management in addressing postoperative patient dissatisfaction and decreased QOV. The global DED prevalence is 5 to 50%. The inclusion of cataract surgery-related DED may expand it to half of the global population. Thus, a thorough and effective DED assessment during the perioperative period of cataract surgery is warranted.

The major mechanisms underlying DED pathogenesis include tear-film instability and ocular surface inflammation. We observed that DED after cataract surgery was weakly and strongly associated with tear secretion (Schirmer's I test) and TFBUT, respectively. Additionally, postoperative upregulation of inflammatory mediators contributes to DED development, which simultaneously alters subjective perception and sensitivity of the ocular surface nerve plexuses.¹ This might be attributed to the surgical procedure and/or preservatives in postsurgical eye drops, which contribute to inflammation

	O THE LACE OF SOLUTION	UP JOURNAL OF OPHTHALMOLOGY		
		An Official Journal of Uttar Pradesh State Ophthalmological Society, UPSOS (Northern Ophthalmological Society, NOS)		
Ì	p-ISSN: 2319-2062		DOI: 10.56692/upjo.2024120103	

and tear-film instability. Larger corneal wounds,² longer microscopic exposure times,^{3,4} and greater phacoemulsification energy increase the likelihood of DED after cataract surgery, including the persistent decrease of conjunctival goblet cells despite uncomplicated phacoemulsification.

MGD is closely related to DED pathology,⁵ and Han *et al.*^{6,7} reported persistent MGD after cataract surgery without accompanying structural changes, even in patients without preexisting MGD. A Japanese study⁸ focused on TFBUT patterns in patients with DED after cataract surgery, observing a random break pattern predominant during the postoperative period, a common feature in evaporative DED and is often associated with MGD. Therefore, owing to potential

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How to cite this article: Singh S, Siddiqui RA, Saroj SC, Singh S, Yadav A. Study of Developing Dry Eye After Cataract Surgery in MGD Patients. UP Journal of Ophthalmology. 2024;12(1): 7-10. Received: 10-04-2024, Accepted: 15-06-2024, Published: 30-08-2024 comorbidity, MGD should be preoperatively evaluated in all patients regardless of preexisting DED.

Additionally, we investigated the risk factors for DED after cataract surgery, which were consistent with the characteristics reported by epidemiological studies on DED in the elderly. Patients with traditional DED risk factors⁹⁻¹¹ may present with DED after cataract surgery; therefore, preventative measures should be considered. Other non-ocular risk factors include systemic diseases, medications, and psychiatric conditions. Notably, diabetes mellitus is among the systemic risk factors for sDED, likely affected by tear hyperosmolarity and tear-film instability resulting from dysfunction of lacrimal functional units and the ocular surface. The effects of hyperglycemia on the lacrimal gland functional unit components are systematically transmitted via neural connections, resulting in abnormal tear production and composition, both of which contribute to DED.9 Ultrasonic energy produced during phacoemulsification can cause free radical formation,² which may compound the damage. Although the exact underlying mechanisms remain unclear, other systemic diseases and treatments damage the conjunctiva, lacrimal glands, goblet cells, and peripheral nerve innervations, predisposing the affected population to DED after cataract surgery.

Recent consensus is on neurogenic stress and ocular surface inflammation's concomitant role in DED pathogenesis.¹⁸ Additionally, DED is associated with other chronic pain conditions and may alter the genetic susceptibility for depression.^{12,13} Patients with DED usually present with chronic pain syndromes, which are associated with increased severity of subjective DED symptoms, even with comparable objective ocular surface signs. Anxiety disorders and usage of anxiolytics, antidepressants, or sleep medication are also associated with DED after cataract surgery.¹⁴ After extrapolating the psychogenic effects on ocular sensations, it is possible that only the subjective indicators are elevated for specific populations, although further studies are required to validate the hypothesis. The relationship between surgical techniques and DED after cataract surgery was also analyzed. Studies have reported a correlation of DED examination values with microscopic light exposure time^{15,16} and phacoemulsification energy used. Surgeons should strategize to minimize both factors in patients with preoperative DED or risk factors.

Pathological changes and inflammatory kinetics preceding DED after cataract surgery are crucial when determining postoperative treatment and management. DED severity after cataract surgery tends to peak around one week postoperatively.¹⁷⁻²⁰ Notably, numerous studies reported postoperative follow-ups scheduled after one week and one month, and the true peak of DED symptoms most likely lies within this time period. Kasetsuwan et al.7 proposed that the timeline of corneal neuron regeneration and DED symptom exacerbation within one postoperative month may be correlated. As new neurite cells emerge, there is a stark increase in neural growth factors at 25 days postoperatively, indicative of sub-epithelial corneal axon regeneration. Khanal et al. reported that alterations in corneal neuronal plexuses and a postoperative decrease in the blink rate reduced corneal sensitivity and feedback. Moreover, the authors reported recovery of tear functions within one postoperative month, supporting the existence of a DED peak within one week and one month postoperatively. Notably, this result also coincides with the observed increased tear evaporation up to one month postoperatively associated with usage of benzalkonium chloride preservatives.

RESULTS

Postoperatively, in group A tear break-up time decreased and control fluorescein staining scores increased significantly. Schirmer test 1(ST1) increased in the early postoperative course but decreased later. The eyelid margin morphology scores and meibomian gland expression score of group A significantly increased after cataract surgery. Thus, patients with MGD may have a greater chance of developing dry eye

			GROUP A (MO	GD GROUP)				
Parameters	Preoperative value	1 day	1 month	2 months	Overall	Preop. Vs 1 day	Preop. vs 1 month	Preop. vs 2 months
TBUT (sec)	4.2 ± 0.4	3.1 ± 0.3	3.7 ± 0.5	4.1 ± 0.4	<0.001	<0.001	<0.001	<0.001
Schirmer test (mm)	5.2 ± 0.5	4.3 ± 0.5	4.5 ± 0.8	5.2 ± 0.6	0.894	0.816	0.924	0.557
Corneal sensitivity threshold (mm)	57.9 ± 1.7	55.1 ± 1.8	57.7 ± 1.5	57.9 ± 1.7	0.016	0.024	0.021	0.019
Corneal staining score	1.3 ± 0.4	2.1 ± 0.8	1.2 ± 0.7	0.7 ± 0.3	0.024	0.041	0.031	0.035
GROUP B (CONTROL GROUP) Shirmer 1 test (ST1)								
TBUT (sec)	12.9 ± 2.5	11.4 ± 2.3	13.4 ± 2.7	14.1 ± 3.1	<0.001	<0.001	<0.001	<0.001
Schirmer test (mm)	17.4 ± 5.9	15.7 ± 4.8	16.2 ± 5.1	16.9 ± 5.4	0.829	0.529	0.241	0.268
Corneal sensitivity threshold (mm)	55.9 ± 1.4	51.2 ± 0.9	54.1 ± 1.2	56.8 ± 1.3	0.295	0.262	0.418	0.350
Corneal staining score	0.4 ± 0.1	0.7 ± 0.2	0.5 ± 0.1	0.3 ± 0.1	0.031	0.035	0.029	0.027

Table 1: Mean value of ocular surface parameters measured preoperatively, at 1 day, 1 and 2 months after cataract surgery

Table 2: Correlation between cytokines in the lacrimal tear samples
and dry eye parameters at 1 month postoperatively

	TBUT	Schirmer I test	Corneal staining score
IL-1β	-0.34	-0.41	0.38
	0.04*	NS	0.03*
IL-6	-0.35	-0.31	0.39
	<0.01*	0.03*	<0.01*
IL-8	-0.29	-0.25	0.26
	0.01*	NS	0.02*
MCP-1	-0.28	-0.25	0.31
	0.021*	NS	0.034*
IFN-γ	-0.25	-0.23	0.33
	0.04*	NS	0.03*
TNF-α	-0.14	-0.11	0.16
	0.032*	NS	0.027*

Table 3: Correlation between ocular symptom score and MGD	I
parameters at two months postoperatively	

Ocular symptom score		p-value
Lid margin abnormality	0.37	0.002*
Meibum expressibility	0.21	0.031*
Meibum quality	0.19	0.029*
Meibo-score	0.16	0.067

Numbers are Spearman correlation coefficients and *p-value* (NS: non-specific)

Significant correlations are marked as*

disease after cataract surgery. Cataract surgery may aggravate the sign of MGD, and the severity of MGD may positively correlate with TBUS, CFS and corneal lesions after surgery.

Mean value of ocular surface parameters measured preoperatively, at 1 day, 1 and 2 months after cataract surgery as shown in Table 1.

Correlation between cytokines in the lacrimal tear samples and dry eye parameters at 1 month postoperatively as shown in Table 2.

Correlation between ocular symptom score and MGD parameters at two months postoperatively as shown in Table 3.

CONCLUSION

The characteristics of dry eye after cataract surgery in patients with MGD differs from common cataract patients. Changes in the early postoperative phase to the ocular surface were caused by surgical factors and the damage to epithelial function in the later postoperative phase were mainly associated with inflammation of the meibomian gland and eyelid

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