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# Original Research Article

# Study of Correlation between Dyslipidemia with Meibomian Gland Dysfunction and its Severity

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**ABSTRACT:**

**Purpose:** This study aimed to determine correlation between Dyslipidemia with meibomian gland dysfunction (MGD) and its severity. The dyslipidemia of cases were compared with the age and sex matched non-MGD individuals.

**Materials and methods:** This hospital based cross-sectional study was conducted in the ophthalmology department of a tertiary hospital consisting of 188 patients (94 patients with MGD and 94 controls without MGD). All the patients underwent comprehensive ocular examination including visual acuity, slit lamp examination, intraocular pressure. Slit lamp examination was performed to look for lid margin signs. Meibomian gland functionality was assessed by fluorescein staining. The disease severity was assessed according to International Workshop on Meibomian Gland Dysfunction and management 2011. MGD is divided into four stages taking both symptoms and signs into consideration and eye with more grading of MGD was included in study. Controls were age and sex matched non-MGD patient with refractive error. Dyslipidemia was defined by TC ≥200 mg/dl, TGs ≥150 mg/dl, LDL-C ≥130 mg/dl and/or HDL-C <40 mg/dl. Lipid parameters were assessed for both cases and controls. Data analysis was done by Statistical Package for Social Sciences (SPSS) version 17.0. Unpaired t test, Mann-Whitney U test, Chi-square test, Fisher's exact test and logistic regression model were used appropriately.

**Results:** Patients with MGD showed higher mean OSDI, TGs, TC, LDL-C, HDL-C as compared to controls. Dyslipidemia and high lipid parameters were associated with increasing severity of disease. Increased OSDI, LDL-C ≥130 mg/dl and HDL-C <40 mg/dl were significantly correlated with MGD in logistic regression model. In other multivariate logistic regression model , increasing age and MGD were significantly correlated with dyslipidemia.

**Conclusion:** Our study showed that symptomatic MGD patients were significantly associated with dyslipidemia and deranged lipid profile.

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**Keywords:** Symptomatic MGD, Dyslipidemia, OSDI, Lipid parameters.

## 1. INTRODUCTION

The term "Meibomian gland dysfunction" (MGD) was first used by Korb and Henriquez in 1980. Meibomian gland dysfunction (MGD) is chronic, diffuse abnormality of the meibomian glands, commonly characterized by terminal duct obstruction and /or qualitative/quantitative changes in glandular secretion.[1] MGD is a common chronic condition, affecting millions worldwide, and is one of the most frequent pathologies observed on a daily basis in the ophthalmology clinic. In the last decade, MGD has become recognized as the major cause of evaporative dry eye.[2] Wide variations in the prevalence of MGD exist, ranging from as low as 3.5% to close to 70% in hospital and population-based studies.[3] Differences in the diagnostic criteria used to define MGD account for much of this variation. MGD is markedly more prevalent in Asian populations.[4]

Meibomian glands are large sebaceous glands present in eyelids which secrete lipids that form that layer of tear film which is superficial to protect evaporation of the aqueous component. Meibomian glands are densely innervated, and their function is regulated by androgens, progestins, estrogens, retinoic acid, growth factors, and possibly by neurotransmitters. The polar and non-polar lipids were produced by the glands through a complex and incompletely understood process.[5] These lipids are secreted into the ducts that open in the lid margin secreting lipids to form tear film. Over 100 major individual complex mixture of lipids, over 90 proteins, electrolytes are present in meibum that contribute to the stability of tear film in health and disease.[6] Aging, diet, sex hormones, usage of antibiotics and the dysfunction of meibomian glands alters the lipid composition and proteins in meibum resulting in altered tear film stability and function.[2]

MGD exists in two broad categories: as low delivery and high delivery forms.[7] Low delivery MGD is further divided into hyposecretory and obstructive types, where reduced volume of meibum secretion results from glandular dropout and/ or blockage of MG orifices, respectively.[4] Hypersecretory or higher delivery forms of MGD are characterized by a large volume of meibum at the lid margin on gentle application of pressure on tarsus.[3]

The main source of lipids for the human tear film are the meibomian glands. The meibomian gland secretions consist of a complex mixture of various polar and non-polar lipids containing cholesterol and wax esters, diesters, triacylglycerol, free cholesterol, free fatty acids and phospholipids. [5] Considering

that meibum is primarily composed of lipid, disorders of lipid metabolism or production may also lead to MGD. Meibum of MGD patients has different components and proportions of cholesterol compared to the meibum of controls.[8]
Dyslipidemia has[9-12] been linked to the development of MGD, but direct evidence supporting this relationship is lacking. Dyslipidemia is a disorder of systemic lipid metabolism which is characterized

by increased levels of total cholesterol (TC), triglycerides (TGs), low-density lipoprotein cholesterol (LDL-C) and/or a reduction in high-density lipoprotein cholesterol (HDL-C).

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Dyslipidemia is a major risk factor for heart disease, but its exact relationship with MGD is unknown. [13,14] Plasma lipids have been suggested to affect the meibomian gland as well, but the evidence is largely circumstantial. Moreover there were reports stating that there is no relationship between the status of plasma lipids and MG lipid composition. [7,9-12,13-15] The idea that plasma lipids can affect the MG is anecdotal and likely based on the fact that it is a lipid synthesizing organ and alterations in systemic[9-11,14,15] lipid metabolism could affect its structure and function. Whether pathologic levels of systemic lipids contribute to MGD remains speculative.[7,16] It is thus important to determine if any correlation exists between lipid parameters/dyslipidemia and MGD and its severity. Hence, to screen cardiovascular disease at an iceberg level , it is important to determine whether MGD either asymptomatic or symptomatic can be the presentation of dyslipidemia/deranged lipid profile.

## 2. MATERIALS AND METHODS

This was a hospital-based cross-sectional study conducted from September 2019 to May 2020 at a tertiary care centre. Ninety four meibomian gland dysfunction (MGD) patients and ninety four controls visiting the ophthalmology outpatient department (OPD) with refractive error were assessed and those fulfilling the inclusion and exclusion criteria were included in the study.

### INCLUSION CRITERIA FOR CASES

Patient aged from 18-55 years with MGD were included in the study. Diagnostic criteria for MGD was taken as MGD was clinically defined as meibomian gland obstruction (meibomian gland orifice plugging) and/or gland dropout and abnormal gland secretions.[8]

### INCLUSION CRITERIA FOR CONTROLS

The control group included age and sex matched non-MGD patients visiting the eye OPD for correction of refractive error.

### EXCLUSION CRITERIA FOR BOTH CASES AND CONTROLS

Patients with infectious keratoconjunctivitis, inflammatory ocular surface disorders unrelated to MGD, recent ocular surgery, concomitant topical medications for glaucoma, topical ophthalmic steroids taken four weeks before study, treatment with drugs affecting tearing or lipid levels (antihypertensives/cholinergics/OCPs/isotretinion/statins), history of pregnancy, presence of Sjögren's syndrome, rosacea, Parkinson's disease.

Age and sex were the basic demographic features of cases and controls which were noted. Baseline assessment was done and symptoms were assessed on the basis of Ocular Surface disease Index (OSDI) questionarrie.[17] OSDI questionarrie was explained to all the patients in local language. All the patients underwent fasting blood lipid profile levels estimation.

### OPHTHALMOLOGICAL EXAMINATION

All patients underwent comprehensive ocular examination including visual acuity, slit lamp examination, intraocular pressure. Slit lamp examination was performed to look for lid margin signs for posterior lid margin erythema/hyperemia, lid margin thickening/irregularity, meibomian gland orifice plugging, turbidity of meibomian gland secretions, lid margin telangiectasia, and meibomian gland plugging. Meibomian gland functionality was assessed by the expressibility and secretion quality which was determined by giving moderate digital

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pressure over the central (±nasal) third of the lower/upper lids and was used to express the meibum. For meibum quality it was assessed in each of eight glands of the central third of the lower eyelid, graded as 0 to 3 for each gland in which 0= clear meibum,1= cloudy meibum, 2= cloudy with debris,3= thick like toothpaste. Range = 0-24.

In the central third of the lower eyelid expressibility was assessed on a scale of 0 to 3 in five glands, according to the number of glands expressible: 0= all glands expressible 1= 3-4 glands expressible

2= 1-2 glands expressible 3= no glands expressible. The surface damage to the exposed eye was assessed by fluorescein staining. Staining was represented by punctate dots on a series of panels. After instillation of the dye, the eye was examined with the slit-lamp biomicroscope using ×16 magnification with ×10 oculars using the Haag-Streit slit-lamp in cobalt blue exciter filter with a complementary yellow barrier filter. Staining scores were obtained by summing the scores of the exposed cornea and conjunctiva (OXFORD SCHEME GRADING).[18] The disease severity was assessed according to International Workshop on Meibomian Gland Dysfunction and Management in 2011, MGD is divided into four stages, taking both the symptoms and clinical signs into consideration. The eye with more grading of MGD was included in the study (Figure 2). Dyslipidemia, defined by TC ≥200 mg/dl, TG ≥150 mg/dl, LDL-C ≥130 mg/dl and /or HDL-C <40 mg/dl.

Institutional ethical clearance was taken for the study.

Written informed consent was taken from all the patients included in the study and during study period and we adhered to the tenets of the Declaration of Helsinki.

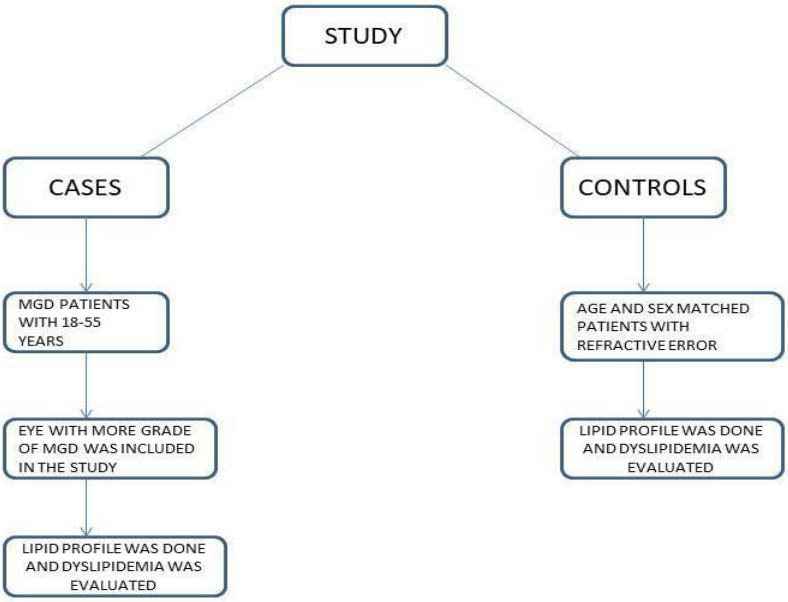


**FIGURE 1: Flow chart depicting the study**

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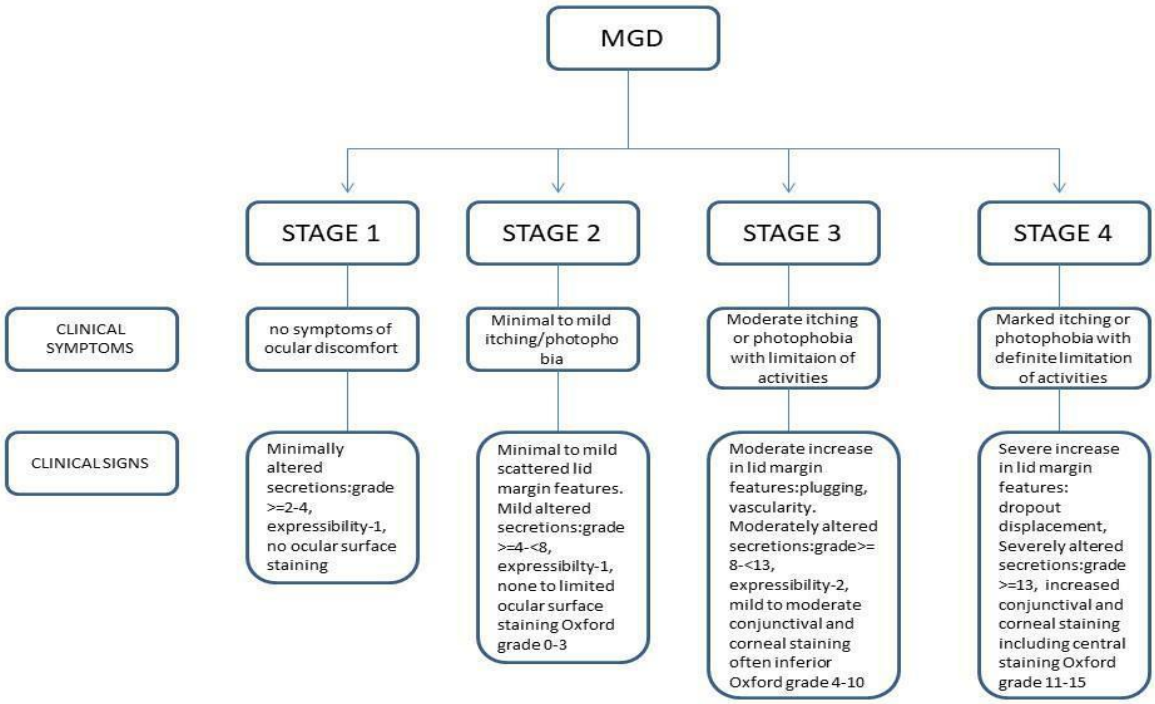


**FIGURE 2: Clinical description of the stages of meibomian gland dysfunction**

## STATISTICAL ANALYSIS

Statistical analysis was performed by the Statistical Package for Social Sciences (SPSS) program for Windows, version 17.0 (SPSS, Chicago, Illinois). Continuous variables were presented as mean ± SD, and categorical variables were presented as absolute numbers and percentage. Data was checked for normality before statistical analysis. Normally distributed continuous variables were compared using the unpaired t test, whereas the Mann-Whitney U test was used for those variables that were not normally distributed. The categorical variables were analyzed using either chi square test or Fisher's exact test. To identify potential factors associated with dyslipidemia univariate analyses was performed. To identify independent risk factors for cases and dyslipidemia multivariate logistic regression model was used. A stepwise approach was used to enter new terms into the model, with a limit of $p<0.05$ to enter the terms.

## 3. RESULTS

Ninety four MGD patients and ninety four controls without MGD were taken for the study from the ophthalmology OPD of a tertiary hospital.

## COMPARISON BETWEEN CASES AND CONTROLS

The demography and lipid parameters results are shown and compared in table 1. Both the cases and

controls were matched for age, sex ($p$=0.697 and $p$=0.559). The mean OSDI ($p$ <0.001), mean TG

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($p$=0.004), mean LDL-C ($p$=0.002), mean HDL-C ($p$=0.026), and mean TC ($p$=0.025) was significantly higher in the cases as compared to controls.

**FACTORS SIGNIFICANTLY CORRELATED WITH MGD AS A DEPENDENT VARIABLE**

Taking MGD as a dependent variable, in the logistic regression model, the Odds Ratio and 95% CI calculated for the factors associated with cases with model Nagelkerke $R^2$=0.817. This model revealed that greater OSDI had thirty times the odds of having MGD than those with lesser OSDI ($p$ <0.001). Compared to cases with LDL-C <130 mg/dl, the cases with LDL-C ≥130 mg/dl had 0.05 times greater odds of having MGD ($p$=0.025) whereas the cases with HDL-C <40 mg/dl had about six times greater odds of having MGD compared with cases with HDL-C ≥40 mg/dl ($p$=0.010). The model did not show significant association between raised TGs, TC and MGD.

**DYSLIPIDEMIAWITH SEVERITYOFMGD**

On taking dyslipidemia and severity of MGD in account there was no dyslipidemia in stage 1, the frequency of dyslipidemia in stage 2 was 3 (13.0%), the frequency of dyslipidemia in stage 3 was 9 (39.1%) and the frequency of dyslipidemia in stage 4 was 11 (47.8%). The maximum number of dyslipidemia was found in the MGD cases with stage 4. Dyslipidemia was found with the increasing severity of MGD and association between increasing severity of MGD and dyslipidemia was statistically significant i.e. $p$<0.001. The distribution and association of dyslipidemia in stages of MGD is shown in table 3.

**STUDYOF MGD SEVERITYAND LIPID COMPONENTS**

It was observed that as the stage of MGD increased the number of patient with TG ≥150 mg/dl increased. The frequency of TG ≥ 150 mg/dl in stage 1 was 2 (6.2%), in stage 2 was 7 (21.9%), and in stage 3 and 4 was 11 (34.4%) and 12 (37.5%) respectively. The maximum number of patients with raised TG were in stage 4 MGD and the $p$ value was <0.001 which was significant and hence, there was direct association of increasing severity of MGD and raised TGs.

The frequency of cases with TC ≥200 mg/dl in stage 1 was 2 (6.1%), in stage 2 was 6 (18.2%), and in stage 3 and 4 was 14 (42.4%) and 11 (33.3%) respectively. The maximum frequency of raised TC was in stage 3 MGD. As the $p$ value was found to be significant i.e. $p$ <0.001, there was direct association of increasing severity of MGD and abnormal TC.

The frequency of cases with LDL-C ≥130 mg/dl in stage 1 was 1 (2.7%), in stage 2 was 7 (18.9%), and in stage 3 and 4 was 17 (45.9%) and 12 (32.4%) respectively. The maximum frequency of raised LDL-C was in stage 3 MGD. As the $p$ value was found to be significant i.e. $p$ <0.001, there was direct association of increasing severity of MGD and abnormal LDL-C.

The frequency of cases with HDL-C ≥40 mg/dl in stage 1 was 23 (54.8%), in stage 2 was 18 (42.9%), and in stage 3 and 4 was 1 (2.4%) and 0 respectively. The maximum frequency of HDL-C ≥40 mg/dl was in stage 3 MGD. The HDL-C ≥40 mg/dl level showed a direct association with staging of MGD as the severity of MGD increased , number of patients with HDL ≥40 mg/dl also increased and number of patients with HDL-C <40 mg/dl reduced as the severity of MGD increased. The distribution is shown in table 4

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## FACTORS SIGNIFICANTLY CORRELATED WITH DYSLIPIDEMIAAS A DEPENDENT VARIABLE

With dyslipidemia as a dependent variable ,in the logistic regression model, odds ratio and 95% CI calculated for the factors associated with dyslipidemia with model Nagelkerke $R^2$=0.436 showed that only covariates which significantly affected the fit of model was age ≥40 years and MGD shown in table 5. This regression model showed that patients with age ≥40 years had twenty seven times increased odds of having dyslipidemia ($p$ <0.001) and patients with MGD had five times increased odds of having dyslipidemia ($p$=0.001)

## TABLES:

**Table 1: Demography and lipid parameters in cases and controls**

| S No | PARAMETERS | CASES (mean± standard deviation) | CONTROLS (mean± standard deviation) | P VALUE |
|---|---|---|---|---|
| 1 | Age ( years) | 34.54±9.16 | 35.07±9.56 | 0.697 |
| 2 | Gender – M/F | 46/48 | 42/52 | 0.559 |
| 3 | OSDI | 28.91±23.66 | 10.57±0.38 | <0.001 |
| 4 | Triglycerides (mg/dL) | 131.03±54.94 | 110.51±40.83 | 0.004 |
| 5 | LDL-C (mg/dL) | 129.22±45.81 | 110.55±40.83 | 0.002 |
| 6 | HDL-C (mg/dL) | 42.86±8.29 | 40.37±6.80 | 0.026 |
| 7 | TC (mg/dL) | 171.76±58.78 | 155.31±39.07 | 0.025 |

**Table 2: Table showing factors correlated significantly with MGD as a dependent variable**

| **PARAMETERS** | **B** | **S.E.** | **Wald** | **Df** | **p value** | **Odds Ratio** | **95.0% C.I.** | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | **Lower** | **Upper** |
| OSDI | 3.425 | 0.645 | 28.233 | 1 | <0.001 | 30.714 | 8.684 | 108.634 |
| TG≥150 | -0.418 | 1.308 | 0.102 | 1 | 0.749 | 0.658 | 0.051 | 8.555 |
| LDL≥130 | -3.068 | 1.369 | 5.019 | 1 | 0.025 | 0.047 | 0.003 | 0.681 |

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| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| HDL<40 | 1.742 | 0.678 | 6.593 | 1 | 0.010 | 5.709 | 1.51 | 21.58 |
| TC≥200 | -0.057 | 1.717 | 0.001 | 1 | 0.973 | 0.945 | 0.033 | 27.334 |
| Constant | -38.787 | 7.108 | 29.777 | 1 | <0.001 | 0 | | |

**Table 3: Distribution and association of dyslipidemia in stages of MGD**

| Stage of MGD | Dyslipidemia absent | Dyslipidemia present | Total | P value |
|---|---|---|---|---|
| Stage 1 | 32 (45.1%) | 0 | 32 | <0.001 |
| Stage 2 | 30 (42.3%) | 3 (13.0%) | 33 | |
| Stage 3 | 8 (11.3%) | 9 (39.1%) | 17 | |
| Stage 4 | 1 (1.4%) | 11 (47.8%) | 12 | |
| Total | 71 | 23 | 94 | |

**Table 4: Distribution of MGD severity and lipid components**

| Stage of MGD | Triglyceride | | | | Total cholesterol | | | | LDL cholesterol | | | | HDL cholesterol | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | TG <150 | TG ≥150 | TOTAL | p value | TC <200 | TC ≥200 | TOTAL | p value | LDL-C <130 | LDL-C ≥130 | TOTAL | p valve | HDL ≥40 | HDL< 40 | TOTAL | p value |
| STAGE 1 | **30** (48.4%) | **2** (6.2%) | **32** (34%) | <0.001 | **30** (49.2%) | **2**(6.1%) | **32** (34%) | <0.001 | **31** (54.4%) | **1** (2.7%) | **32** (34%) | <0.001 | **9** (17.3%) | **23** (54.8%) | **32** (34%) | <0.001 |
| STAGE 2 | **26** (41.9%) | **7** (21.9%) | **33** (35.1%) | | **27** (44.3%) | **6** (18.2%) | **33** (35.1%) | | **26** (45.6%) | **7** (18.9%) | **33** (35.1%) | | **15** (28.8%) | **18** (42.9%) | **33** (35.1%) | |

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| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| STAGE 3 | **6** (9.7%) | **11** (34.4%) | **17** (18.1%) | | **3** (4.9%) | **14** (42.4%) | **17** (18.1%) | | **0** | **17** (45.9%) | **17** (18.1%) | | **16** (30.8%) | **1** (2.4%) | **17** (18.1%) |
| STAGE 4 | **0** | **12** (37.5) | **12** (12.8%) | | **1** (1.6%) | **11** (33.3%) | **12** (12.8%) | | **0** | **12** (32.4%) | **12** (12.8%) | | **12** (23.1%) | **0** | **12** (12.8%) |

**Table 5: Table showing dyslipidemia a dependent variable and factors significantly correlated with dyslipidemia**

| | **B** | **S.E.** | **Wald** | **df** | **p value** | **OR** | **95.0% C.I.** | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | **Lower** | **Upper** |
| Age ≥40 yrs | 3.307 | 0.568 | 33.952 | 1 | <0.001 | 27.302 | 8.976 | 83.039 |
| Sex (Male) | -0.311 | 0.481 | 0.419 | 1 | 0.518 | 0.733 | 0.286 | 1.879 |
| MGD | 1.685 | 0.509 | 10.948 | 1 | 0.001 | 5.392 | 1.987 | 14.628 |
| Constant | -7.519 | 1.134 | 43.991 | 1 | <0.001 | 0.001 | | |

## 4. DISCUSSION

This study was conducted to study the correlation between lipid parameters and dyslipidemia with meibomian gland dysfunction and its severity.

In our study the mean age of cases was 34.74 ± 9.51 years and there were 51.1% females and 48.9% males with female to male ratio of 1.04:1. This is consistent with the study by Kaur *et al.*[21] Previous study by Villani *et al.*[22] and Banait *et al.*[23] had suggested old age as a risk factor for MGD.

The OSDI questionnaire was a subjective measurement tool used for assessing the symptoms of MGD. In the present study, mean OSDI was significantly greater in MGD patients as compared with the mean OSDI in controls without MGD in which OSDI was normal ($p$ <0.001). Study done by Guilani *et al.*[14] had also used OSDI score as the baseline assessment of MGD symptoms.

Our study found that patients with MGD had significantly higher mean values for TGs, TC, LDL-C and HDL-C versus individuals of similar age and gender without MGD ($p$ <0.05). The greatest variation in mean values was observed in TGs and LDL-C levels where the absolute differences in the mean values were 20.52 and 18.67 mg/dl,

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respectively. This is consistent with previous study by Pinna *et al.*[15] and Braich *et al.*[9] who found significantly higher mean values for TGs, TC, LDL-C and HDL -C. The greatest variation in mean values was observed in LDL-C and TC levels in study by Braich *et al.*[9]

In our study patients with MGD more often had serum TGs ≥150 mg/dl, TC ≥200 mg/dl, and LDL-C ≥130 mg/dl. However when considering patients with serum HDL-C <40 mg/dl there was no significant difference between those with 'or' without MGD. Previous study done by Dao *et al.*[11] which showed that MGD was associated with raised TG, hypercholesterolemia, raised HDL-C. A study by Pinna *et al.*[15] showed that MGD was associated with hypercholestereolemia, high HDL-C, high LDL-C and the study by Braich *et al.*[9] which showed that MGD was associated with high TG, high LDL-C, high TC. A study by Banait *et al.*[23] showed positive correlation between MGD and increased LDL-C, TGs and TC.

In our study there was significant association between dyslipidemia and increased severity of MGD as maximum number of patients 11 (47.8%) with dyslipidemia belonged to stage 4. This finding is consistent with that of Guilani *et al.*[14] which showed that there was positive association between increased severity of MGD and all lipid profile components.

The number of MGD patients with TC <200 mg/dl and TC ≥200 mg/dl were 61 (64.9%) and 33 (35.1%) respectively. Maximum number of the patients with TC <200 mg/dl belonged to stage 1 of MGD while maximum number of patients with TC ≥200 mg/dl belonged to stage 3 of MGD. As the p value was <0.001, it indicated association was significant between hypercholesterolemia (TC ≥200 mg/dl) and increased severity of MGD. This is consistent with the findings obtained in the studies conducted by Bukhari *et al.*[10], Dao *et al.*[11] and Guilani *et al.*[14]

The number of MGD patients with TG <150 mg/dl and TG ≥150 mg/dl were 62 (66%) and 32 (34%) respectively. Maximum number of the patients with TG <150 mg/dl belonged to stage 1 of MGD while maximum number of the patients with TG ≥150 mg/dl belonged to stage 4 of MGD. As the *p* value was < 0.001, it indicated a strong association between TG ≥150 mg/dl and increased severity of MGD. This is consistent with the findings obtained in the studies conducted by Guilani *et al.*[14] All the other studies found increased TGs in moderate and severe MGD cases, but could not reach a statistical significance.

The number of MGD patients with LDL-C <130 mg/dl and LDL-C ≥130 mg/dl were 57 (60.63%) and 37 (39.36%) respectively. Maximum number of the patients belonged to stage 2 of MGD, whereas stage 4 of MGD had least number of patients. Maximum number of the patients with LDL-C <130 mg/dl in our study belonged to stage 1, while maximum number of the patients with LDL-C ≥ 130 mg/dl belonged to stage 3. Because the *p* value was <0.001, it indicated a strong association between increased LDL-C (level ≥130 mg/dl) and increased severity of MGD. This observation is consistent with the findings of Bukhari *et al.*[10] and Guilani *et al.*[14]

The number of MGD patients with HDL-C ≥40 mg/dl and HDL-C <40 mg/dl were 52 (55.3%) and 42 (44.7%) respectively. The maximum number of patients with HDL-C <40 mg/dl belonged to stage 1 of MGD, while maximum number of patients with HDL-C ≥ 40 mg/dl belonged to stage 3 of MGD. As the *p* value was <0.001, it indicated a strong association between increased HDL-C increased severity of MGD. This observation is in

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accordance with the study conducted by Dao *et al.*[11], Pinna *et al.*[15] and Guliani *et al.*[14] Their study was concluded with the observation that the component which contributed most to the hypercholesterolemia found in moderate to severe MGD patients was increased HDL-C levels. In our study even increasing LDL-C level was found to be associated with the increasing severity of MGD which is consistent with the study conducted by Guliani *et al.*[14]

In the first logistic regression model (table 2), the model revealed that greater OSDI had thirty times the odds of having MGD than those with lesser OSDI ($p$ <0.001). As compared to cases with LDL-C <130 mg/dl, the cases with LDL-C ≥130 mg/dl had 0.05 times greater odds of having MGD ($p$=0.025) whereas the cases with HDL-C <40 mg/dl had about six times greater odds of having MGD compared with cases with HDL-C ≥40 mg/dl ($p$=0.010). The model did not show significant association between raised TGs, TC and MGD. This is not consistent with the previous study by Braich *et al.*[9], in which the model showed an age >65, abnormally high LDL-C, TC and TGs which had raised the odds of having MGD.

In our study, using multivariate logistic regression analysis, age ≥40 years and MGD were independent predictors associated with dyslipidemia and were significantly associated with the increased odds of having dyslipidemia. This is consistent with the finding of Braich *et al.*[9] in which elderly subjects and MGD were associated with increased odds of having dyslipidemia.

There are few limitations of our study like it was a cross-sectional study and hence cause and effect could not be established between MGD and dyslipidemia. Since genetics characteristics and dietary regional practices have been shown to alter an individual's lipid profile, the outcome and recommendations of the study cannot be generalized as the sample was a homogenous one consisting of Indian patients only. Whether treatment of dyslipidemia has any effect on MGD was not studied by us due to time constraint.

## 5. CONCLUSION

We found a strong association of dyslipidemia and abnormal lipid components with MGD. We found a strong association of severity of MGD with dyslipidemia. Hence, MGD patients should be routinely evaluated for dyslipidemia. MGD is associated with elevated levels of LDL-C, TGs, TC, which are known risk factors for cardiovascular events. Hence all MGD patients should be screened for cardiovascular diseases.

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