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ASSOCIATION BETWEEN DYSLIPIDEMIA AND SEVERITY OF MEIBOMIAN GLAND DYSFUNCTION: A CROSS-SECTIONAL OBSERVATIONAL ANALYSIS

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Abstract

Purpose: Meibomian Gland Dysfunction (MGD) is a leading cause of evaporative dry eye, and emerging evidence suggests that systemic metabolic disturbances such as dyslipidemia may influence its pathogenesis by altering the biochemical composition of meibum. This study aimed to evaluate the association between comprehensive serum lipid abnormalities and the severity of MGD.

Materials and Methods: A cross-sectional observational study was conducted on 40 patients aged 18–54 years diagnosed with MGD at a tertiary care center over three months. Clinical evaluation included meibomian gland expressibility assessment, tear breakup time (TBUT), measured as the interval between a blink and tear film disruption, and Schirmer's test. Fasting lipid profiles were analysed, including total cholesterol (TC), low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides (TG). The primary outcome was the association between dyslipidemia and MGD severity. Data were analysed using appropriate parametric and non-parametric statistical tests, with $p < 0.05$ considered significant.

Results: The majority were 41–50 years (35%), and females constituted 52.5%. Stage II MGD was most common (42.5%), followed by Stage I (22.5%), Stage III (20%), and Stage IV (15%). TBUT reduced progressively from 12.4 ± 1.4 seconds in Stage I to 6 ± 1 seconds in Stage IV, and Schirmer's values declined from 11.5 ± 1.3 mm/5min to 5.8 ± 1 mm/5min ($p < 0.001$). Dyslipidemia prevalence increased with disease severity, from 28% in Stage I to 67% in Stage IV ($p = 0.009$). LDL ≥130 mg/dL ($p < 0.001$), low HDL ($p=0.004$), and TG >150 mg/dL ($p = 0.028$) were significantly associated with advanced MGD.

Conclusion: Dyslipidemia is significantly associated with increasing severity of MGD. Early lipid screening may support targeted management and slow disease progression.

Keywords: Meibomian Gland Dysfunction; Dyslipidemias; Dry Eye Syndromes; Tear Film; Low-Density Lipoproteins; High-Density Lipoproteins; Triglycerides

Introduction

Meibomian Gland Dysfunction is characterized by a chronic diffuse abnormality of the meibomian glands, which consists of blockage of the terminal duct and/or changes in quality/quantity of secretion.[1] Some studies have shown that dyslipidemia can play a significant role in the development and evolution of Meibomian Gland Dysfunction.[2] The meibomian glands, a particular type of sebaceous glands found on the tarsal plates of both upper and lower lids, produce meibum, a complex lipid mixture that constitutes the lipid layer of the precorneal tear film. The lipid layer performs various physiological roles such as the reduction of evaporative loss of aqueous tears, stabilization of the tear film, formation of a smooth optical refractive surface, and preventing excessive tearing.[3] The development of MGD is marked by a series of events starting with hyperkeratinization of the epithelial lining of the duct and elevated melting point of meibum, causing lipid secretions to stagnate, resulting in duct obstruction, inflammation, and atrophy.[4] This results in tear film instability, increased evaporation rates, compensatory reflex tearing, and ocular surface inflammation collectively manifesting as evaporative dry eye disease. MGD has been known as the leading cause of evaporative dry eye. Studies

have reported a wide variation in the prevalence of MGD across different populations, depending on demographic factors and diagnostic criteria used.[5,6]

The systemic associations of MGD remain a subject of intense clinical investigation. One related metabolic condition is dyslipidemia, an abnormality in serum lipid levels, typically characterized by elevated triglycerides (TG), low-density lipoprotein (LDL), total cholesterol, and/or decreased high-density lipoprotein (HDL). Alterations in circulating lipid profiles may influence lipid composition of meibum, thereby altering its physical properties (e.g., increased viscosity), predisposing to duct obstruction and exacerbating MGD.[7,8]

Previous clinical studies support this hypothesis; they identified a significantly higher prevalence of dyslipidemia among individuals with MGD compared to those without MGD.[9] A more recent cross-sectional study findings, reporting that changes in lipid profiles, especially elevated total and LDL cholesterol, reduced HDL, and high triglycerides were commonly associated with MGD, and may contribute to MGD development and progression.[10] The findings indicate that the presence of MGD and its severity as well as the health of the ocular surface may be associated with systemic lipid abnormalities. Although there is growing evidence, the association is still not fully understood, particularly in populations from developing countries where the lipid profile may be different at full fasting, as it is influenced by genetic, dietary and lifestyle variations. The present study therefore aimed to determine the prevalence of dyslipidemia in patients with diagnosis of MGD and to evaluate the association between serum lipid profile (TC, LDL, HDL, TG) and clinical stages of disease.

Materials and Methods

The Department of Ophthalmology at SRM Medical College Hospital and Research Centre conducted this cross-sectional observational study during a period of three months and subjects were recruited after taking informed consent. The Institutional Ethics Committee granted ethical approval prior to the start of the investigation.

Inclusion criteria

Patients aged 18–54 years presenting with dry eye symptoms and diagnosed with MGD, as well as asymptomatic individuals identified with MGD during routine screening, were included. A consecutive sampling technique was used for participant recruitment.

Exclusion criteria

Patients had active lid infections or ocular inflammation, recent ocular surgery, prior topical steroid use within four weeks, altered lacrimal drainage anatomy, were using anti-glaucoma medications or systemic drugs affecting tear secretion (including antihypertensives, oral contraceptive pills, cholinergic, or isotretinoin), had keratoconjunctivitis, were pregnant, diagnosed with rosacea, Sjögren's syndrome, cholestatic liver disease, or Parkinsonism, or were currently on lipid-lowering therapy such as statins, fibrates, bile acid sequestrants, or ezetimibe.

Sample size

The following formula was used to calculate the sample size:

$$n \geq (z_{1-\frac{\alpha}{2}} \times \frac{\sigma}{d})^2$$

Using values of z = 1.96, standard deviation (σ = 16.2) and allowable error (d = 5), the minimum required sample size was calculated as 40. A total of 40 patients were included. Sample size was calculated assuming 95% confidence level, allowable error of 5, and σ =16.2 derived from previous literature.[11]

Methods

Structured evaluation was performed and demographic data including age and gender were recorded. Slit lamp biomicroscopy was used for detailed anterior segment examination, particularly the meibum quality, lid margin telangiectasia, irregularity of the lid margin, and blockage of the meibomian gland orifice. Meibomian Gland Expressibility was evaluated by gently expressing the middle portion of the lower lid of the eyes with a cotton tip applicator or with the fingers. Expressed meibum was analyzed for quality (clear or cloudy, thick, or toothpaste consistency) and ease of expression. Schirmer's test and tear breakup time (TBUT) were used to evaluate the tear film function. TBUT was determined by the time taken from full closure of the eyelids to the first detectable disruption of the precorneal tear film by fluorescein instillation or first detectable dry spot. Each eye was measured three times, and the average result was noted. TBUT <10 seconds was considered abnormal. Schirmer's test values of <10 mm after five minutes were considered indicative of reduced tear secretion. For the purpose of analysing lipid profiles, including total cholesterol, LDL, HDL, and TG, fasting blood samples were obtained. LDL ≥130 mg/dL, HDL <40 mg/dL, triglycerides >150 mg/dL, or total cholesterol >200 mg/dL were all considered indicators of dyslipidemia.

MGD severity was graded using the standardized staging system established in the International Workshop on MGD (2011), based on symptoms and observable clinical signs.[12] All tear film assessments were performed by the same examiner between 10:00 AM and 1:00 PM to minimize diurnal variation.

The primary outcome was the association between dyslipidemia and MGD severity. Secondary outcomes included changes in TBUT and Schirmer scores across different stages and correlation between individual lipid parameters and MGD severity.

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Statistical analysis

SPSS version 25 was used for the analysis. ANOVA was used to compare continuous variables, which were given as mean ± standard deviation, while the Chi-square test was used to present categorical variables as frequencies and percentages. To assess relationships between lipid markers and the severity of MGD, Pearson correlation analysis was used. Statistical significance was defined as a p-value of less than 0.05.

Results

Baseline Characteristics

Forty patients in all met the requirements for inclusion; no participants withdrew or were excluded after enrolment. Most participants were between 41–50 years of age (35%), and females represented a slightly higher proportion (52.5%) than males. The majority of patients had Stage II MGD (42.5%), followed by Stage I (22.5%), Stage III (20%), and Stage IV (15%) [Table 1].

Table 1: Demographic and clinical characteristics

Variable	Category	N (%)
Age group (years)	18–30	13 (32.5%)
	31–40	8 (20%)
	41–50	14 (35%)
	51–54	5 (12.5%)
Gender	Male	19 (47.5%)
	Female	21 (52.5%)
MGD stage	Stage I	9 (22.5%)
	Stage II	17 (42.5%)
	Stage III	8 (20%)
	Stage IV	6 (15%)

MGD – Meibomian gland dysfunction.

Tear Film Parameters Across MGD Severity Stages

TBUT decreased from 12.4 ± 1.4 in Stage I MGD to 6 ± 1 in Stage IV, while Schirmer values decreased from 11.5 ± 1.3 in Stage I to 5.8 ± 1 in Stage IV. TBUT and Schirmer score values differed significantly across MGD severity stages ($p < 0.001$) [Table 2].

Table 2: Comparison of TBUT and Schirmer values across MGD stages

MGD stage	TBUT (seconds)	Schirmer (mm/5 min)
Stage I	12.4 ± 1.4	11.5 ± 1.3
Stage II	10.2 ± 1.2	9.5 ± 1
Stage III	8 ± 1.1	7.8 ± 1.2
Stage IV	6 ± 1	5.8 ± 1
P value	< 0.001	< 0.001

MGD – Meibomian gland dysfunction, TBUT – Tear breakup time.

The number of patients with dyslipidemia increased with MGD severity, from 28% in Stage I to 67% in Stage IV. LDL ≥130 mg/dL was most frequent in Stage III (41%), whereas low HDL was highest in Stage II (42%) and Stage IV (48%). Overall, lipid abnormalities were significantly associated with advancing MGD stage ($p < 0.05$) [Table 3].

Table 3: Distribution of lipid abnormalities across MGD stages

Lipid abnormality	MGD stage				P value
	Stage I	Stage II	Stage III	Stage IV	
TC >200 mg/dL	1 (12%)	6 (38%)	3 (27%)	2 (25%)	0.012
LDL ≥130 mg/dL	0	5 (30%)	5 (41%)	2 (28%)	< 0.001
HDL <40 mg/dL	0	7 (42%)	1 (7%)	3 (48%)	0.004
TG >150 mg/dL	1 (15%)	3 (21%)	4 (44%)	1 (18%)	0.028
Any dyslipidemia	3 (28%)	10 (56%)	5 (62%)	4 (67%)	0.009

HDL: high-density lipoprotein; LDL: low-density lipoprotein; TG: triglyceride; TC: total cholesterol.

Discussion

Our study demonstrated that MGD severity was linked with both reduced tear film stability and a higher prevalence of dyslipidemia, supporting the hypothesis that systemic lipid imbalances may contribute to ocular surface pathology. Most participants were middle aged with a smaller proportion in the younger age groups and a slight predominance of females. Stage II MGD was the most frequently observed severity level, followed by earlier and more advanced stages, indicating that MGD is common in middle-aged adults and it becomes increasingly prevalent with advancing age. Afroz Irfan *et al*. reported a mean age of 48.86 years, with MGD severity increasing significantly with age ($p = 0.002$), and an almost equal gender distribution (60 males, 56 females).[13] Ha *et al*. included 89 patients with a mean age of 57.55 ± 13.19 years, where 33.68% were above 65 years and nearly half were aged 45–64 years, showing a strong female predominance (84.21% vs. 15.79%).[14] Babber *et al*. found a mean age of 46.75 ± 10.93 years with no significant age difference from controls ($p = 0.620$) and a gender distribution of 45% males and 55% females without a significant association ($p = 0.263$).[15] Shakya *et al*. observed most cases in the 61–70-year age group with a slight female predominance and a clear age-related increase in severity.[16] This suggests that MGD is more prevalent in older age and more common among females, supporting our demographic profile.

In our study, tear film stability progressively decreased with increasing MGD severity, with both TBUT and Schirmer scores decreasing from early to advanced stages, indicating tear quality and volume. This study showed an association between disease progression and impaired tear film function. Ha *et al*. reported a much lower mean TBUT in patients with MGD (2.95 ± 2.63 seconds), indicating substantial tear film instability; however, their study did not demonstrate stage-wise variation in TBUT across severity levels. Additional parameters such as corneal staining (1.40 ± 0.77) and OSDI (43.65 ± 20.86) confirmed clinical deterioration in symptomatic patients.[14] The higher TBUT values in our study compared to Ha *et al*. may be attributed to younger participant age and lower baseline MGD severity, since tear film quality declines progressively with age-related gland atrophy.[14] Selimoğlu *et al*. found significantly lower tear film parameters in MGD patients, with TBUT markedly reduced (9.6 ± 2.4 seconds in the MGD group) and Schirmer scores significantly lower than controls (9.91 ± 2.31 mm vs. 10.85 ± 1.56 mm, $p = 0.001$), supporting tear film instability in obstructive MGD.[17] Evidence consistently suggests that worsening MGD severity is associated with measurable tear film impairment, reinforcing its role as a key driver of ocular surface disease.

In our study, dyslipidemia was more common with higher MGD severity, with lipid abnormalities more frequently observed in moderate and advanced stages, suggesting a metabolic role in disease progression. This is consistent with the conclusions of Afroz *et al*., who reported significantly elevated total cholesterol ($p = 0.0001$), triglyceride ($p = 0.0001$), and LDL ($p = 0.015$) levels with increasing severity. HDL showed no significant association ($p = 0.114$), showing that worsening lipid imbalance may similarly drive gland dysfunction.[13] Babber *et al*. confirmed significantly higher serum cholesterol ($p = 0.022$), LDL, and triglycerides with higher severity ($p < 0.001$), with HDL still insignificant.[13] Variation in lipid abnormality patterns across studies may show differences in regional dietary habits, metabolic health, and inclusion criteria, particularly the exclusion of patients already on lipid-lowering therapy in our sample. Li *et al*. reported dyslipidemia increased dry eye disease risk (OR = 1.53), with cholesterol (OR = 1.57), LDL (OR = 1.13), and HDL (OR = 1.06) significantly associated, and noted that prior lipid-lowering therapy increased risk independently (OR = 1.41), implying that chronic metabolic disturbance rather than isolated lipid levels may influence ocular disease.[18]

Shakya *et al*. also observed a significant association between elevated cholesterol, LDL, triglycerides, and increasing MGD severity.[16] Tulsyan *et al*. further linked higher LDL levels with severe MGD alongside systemic metabolic markers.[19] Also, supported by Aldaas *et al*. increased odds of dry eye disease with elevated cholesterol (OR = 1.66), LDL (OR = 1.55), low HDL (OR = 1.45), and high triglycerides (OR = 1.43), a strong association between dyslipidemia and MGD severity.[20] In this way, dyslipidaemia is a part of the underlying mechanism of MGD and helps in metabolic monitoring and systemic treatment of affected patients.

TBUT and Schirmer tests were used for tear film evaluation in MGD and clinical staging was used as an aid to grading the severity of the disease. Further follow-up is needed to see changes over time. Future studies should evaluate the gland structure through assessment such as meibography or tear lipid analysis, to guide treatment.

Limitations

Conclusions of the present study can only be generalized because of the single-center setting and low sample size. In terms of the cross-sectional design of the current research, it is not possible to determine the causality of the relationship between dyslipidemia and MGD. The sample may not be representative of the disease cases in the general population as patients were recruited from a referral hospital. In addition, there was no effort to consider the sex-specific HDL cutoff values, and no analysis for the VLDL component was performed. Thus, further research involving large populations should be conducted.

Conclusion

It is evident from the results that there is a substantial association between dyslipidemia and the severity of MGD. Higher levels of LDL and blood triglycerides and lower HDL have been found to have a correlation with more advanced stages of disease. It can therefore be stated from the current results that lipids have a significant influence on the development and progression of MGD. Hence early lipid screening may support targeted management and slow disease progression.

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