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Comparison of insulin resistance and lipid profile in clinically significant macular oedema versus non-clinically significant macular oedema in patients with type 2 diabetes mellitus

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Abstract. Lifestyle-related disorders, particularly diabetes, pose a significant global health challenge. Diabetic macular oedema, a microvascular complication, highlights the importance of managing insulin resistance and hyperlipidaemia for optimal clinical outcomes. Understanding the interplay between these factors is crucial for optimising therapeutic strategies and improving patient care. This cross-sectional study aimed to compare insulin resistance and lipid profiles between patients with clinically significant macular oedema and those with non-clinically significant macular oedema, both diagnosed with type 2 diabetes mellitus. This research can aid in the earlier identification and classification of macular oedema, enabling more timely and specific interventions. In general, 86 patients with type 2 diabetes

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mellitus and macular oedema were divided into two groups: clinically significant macular oedema and non-clinically significant macular oedema. Comprehensive demographic data, medical histories, and current medication regimens were recorded. Glycaemic control and lipid profiles were assessed, while ophthalmological evaluations included visual acuity measurements and intraocular pressure assessments. Significant differences were observed between the two groups, particularly in diabetes duration, body mass index, blood glucose levels, and lipid profiles. Patients with clinically significant macular oedema had a longer duration of diabetes, a higher body mass index, and elevated blood glucose levels. Triglyceride levels were significantly higher, while high density lipoprotein levels were lower in the clinically significant macular oedema group. Multivariate analysis revealed significant associations between the odds of developing clinically significant macular oedema and diabetes duration, visual acuity, and high-density lipoprotein levels, suggesting their potential as risk factors for this condition

Keywords: diabetic macular oedema; dyslipidaemia; retinal diseases; diabetic neuropathies; retinal vein occlusion

Introduction

Diabetes, a leading lifestyle disorder, poses a significant global health challenge. Diabetes, a widespread chronic ailment caused by insufficient production or utilisation of insulin, impacts an estimated global adult population of approximately 537 million individuals. This figure is anticipated to increase to 643 million by 2030, and further to 783 million by 2045 [1, 2]. In South-East Asia (SEA), specifically, the prevalence of diabetes has exceeded all previous projections. Diabetic macular oedema (DMO) exacerbates the complications associated with diabetes mellitus (DM) by inducing visual impairment, frequently occurring in conjunction with diabetic retinopathy (DR) [3]. DMO, which impacts around 10% of diabetic patients, is identified as the predominant cause of vision-threatening DR in primary care settings. Furthermore, those who have diabetes are at an increased risk of developing cataracts during their youth, which further compromises the vision of individuals with diabetes mellitus [4].

Lipids and insulin are both pivotal factors in the pathogenesis and advancement of DMO and DR. An increased risk is correlated with elevated levels of total cholesterol and triglycerides, specifically low-density lipoprotein (LDL) cholesterol [5]. Conversely, high-density lipoprotein (HDL) cholesterol demonstrates a protective effect. Insulin resistance, a defining feature of type 2 diabetes, contributes to neurodegeneration and retinal vascular abnormalities by impeding glucose absorption and increasing inflammation and oxidative stress. The importance of dyslipidaemia and insulin resistance in the pathophysiology of DR and DMO is underscored by their interaction, which highlights potential therapeutic targets that could alleviate vision loss in diabetic patients [6].

Y.X. Xu *et al.* [7] recently conducted a cross-sectional study to investigate the relationship between diabetic retinopathy (DR) and various measures of insulin resistance (IR) in a cohort of 2,211 patients with type 2 diabetes. The study demonstrated a significant association between the estimated glucose disposal rate (eGDR) and both the presence and severity of diabetic retinopathy among the indicators analysed. The results suggest that eGDR may be a reliable indicator of DR, potentially surpassing other IR indices [8].

In a study by R. Behera *et al.* [9], the correlation between lipid profile and the occurrence and severity of DR, including clinically significant macular oedema (CSMO), was confirmed. With the exception of high triglyceride levels, elevated levels of total cholesterol, LDL, and triglycerides (TG), as well as decreased HDL levels, were found to increase the risk and severity of DR and CSMO. The findings highlight the complex relationship between metabolic factors, such as dyslipidaemia and insulin resistance, and the onset and progression of diabetic ocular complications. This underscores the potential for targeted interventions and risk-reduction strategies.

Understanding the interplay between insulin resistance, lipid profile, and macular oedema in patients with type 2 diabetes is of significant clinical importance. CSMO is a severe complication of diabetic retinopathy, leading to visual impairment and blindness [10]. However, distinguishing between clinically significant and non-clinically significant macular oedema (non-CSMO) is crucial for treatment decisions and prognosis. Investigating the differences in insulin resistance and lipid profiles between these two subgroups can provide valuable insights into the underlying pathophysiology and potentially guide personalised therapeutic approaches. Therefore, this study aimed to investigate the association of CSMO among patients with type 2 diabetes mellitus with insulin resistance and dyslipidaemia at a tertiary care centre in north India.

Materials and Methods

This cross-sectional study was conducted in the Department of Ophthalmology at Era's Lucknow Medical College, Lucknow, over two years (Aug 2021-Aug 2023). A total of 86 patients with macular oedema due to type 2 diabetes mellitus, aged over 18 years, and attending the Ophthalmology OPD at Era's Lucknow Medical College and Hospital were included in the study following ethical clearance and informed consent, following the Helsinki Declaration [11]. Exclusion criteria included patients undergoing insulin treatment and those taking lipid-regulating drugs (such as statins and fibrates), as well as patients receiving treatment for diabetic macular oedema, individuals who had undergone intraocular surgery or laser treatment or received intravitreal injections within the past three months. Additionally, patients with a history of using drugs affecting macular thickness (such as corticosteroids or nephrotoxic

drugs) within the past three months were excluded. Those with significant media haziness, which would prevent proper fundus visualisation, were also excluded. Patients diagnosed with diabetic macular oedema were categorised into two groups: Group CSMO and Group non-CSMO, each consisting of 43 patients. Following enrolment, participants' demographic details, including age and gender, were recorded. A comprehensive systemic examination was undertaken to collect medical histories of chronic conditions such as hypertension, ischaemic heart disease, nephropathy, and neuropathy. Concurrently, information on current medications was recorded. Patients observed an overnight fast before providing a 2 mL blood sample to assess glycaemic control and lipid levels. Parameters such as glycated haemoglobin (HbA1c), fasting blood sugar (FBS), postprandial blood sugar (PPBS), and homeostatic model assessment of insulin resistance (HOMA-IR) were meticulously recorded alongside serum lipid levels. A subsequent blood sample was obtained post-meal to evaluate postprandial blood glucose levels. An ophthalmological assessment was then conducted, including measurements of bestcorrected visual acuity using Snellen's chart and intraocular pressure (IOP) via Goldmann's Applanation Tonometer.

General examinations included height, weight, body mass index (BMI), and blood pressure measurements. Ocular assessments comprised evaluations of uncorrected visual acuity (UCVA) and best-corrected visual acuity (BCVA), torchlight examination, distant direct ophthalmoscopy, slitlamp examination, and applanation tonometry. Fundus examination involved indirect ophthalmoscopy for a comprehensive view of the fundus, allowing for the exclusion of pathologies and retinal detachments. Using a +90D lens and a binocular slit-lamp microscope, a real inverted image was produced with a magnification of 0.75 times. Macular thickness was assessed using Cirrus HD Spectral Domain Optical Coherence Tomography (SD-OCT) from ZEISS, employing an optic disc cube generated from a three-dimensional dataset centred on the optic disc. Measurements were taken in various segments, including central, superior, inferior, temporal, nasal, inner superior, inner inferior, inner temporal, and inner nasal segments. Scans with a signal strength below six were excluded to ensure data reliability and accuracy.

Statistical analysis: The data was analysed using IBM SPSS Inc.'s Statistical Package for Social Sciences, version 21.0, based in Chicago, IL, USA. Data is presented in numerical form, including percentages and the mean value along with its standard deviation (SD). Comparisons were made using the chi-square test, independent samples – using t-test, with a p-value below 0.05 considered statistically significant. Additionally, odds ratios were calculated for various categorical evaluations.

Results

The study compared patient characteristics between those with CSMO and those with nonCSMO. A significant age difference was observed, with a majority of CSMO patients aged >60-70 years (51.2%), whereas the non-CSMO group had a higher proportion aged >50-60 years (62.8%, p = 0.003) (Fig. 1). Gender distribution also varied, with more males in the CSMO group (53.5%) and more females in the non-CSMO group (55.9%) (Fig. 2).

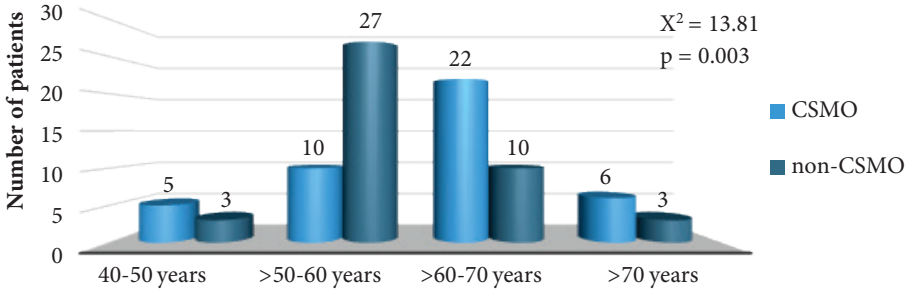

Figure 1. Distribution of patients with CSMO and non-CSMO across different age groups
Source: compiled by the authors

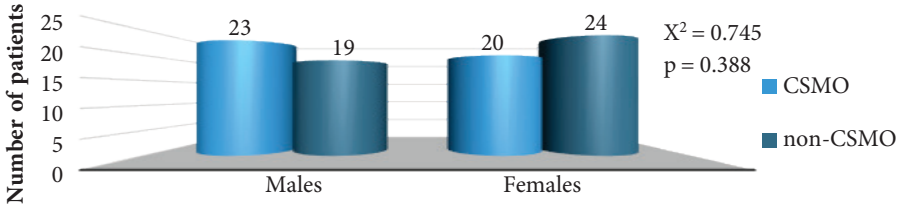

Figure 2. Comparison of gender distribution between patients with CSMO and non-CSMO
Source: compiled by the authors

The CSMO group demonstrated higher mean diabetes duration and BMI than the non-CSMO group. Although IOP was higher in the CSMO group, this difference was not statistically significant (Table 1).

Table 1. Baseline characteristics of CSMO and non-CSMO patients in both groups

Baseline characteristics		CSMO (n = 43)	non-CSMO (n = 43)	p-value
Age (years)	40-50	5 (11.6%)	3 (7.0%)	$X^2 = 13.81$ p = 0.003*
	>50-60	10 (23.3%)	27 (62.8%)	
	>60-70	22 (51.2%)	10 (23.3%)	
	>70	6 (14.0%)	3 (7.0%)	
Gender	Males	23 (53.5%)	19 (44.2%)	$X^2 = 0.745$ p = 0.388
	Females	20 (46.5%)	24 (55.9%)	
Duration (years)	Mean ± SD	17.28 ± 5.61	10.74 ± 1.85	<0.001*
BMI (males)		27.53 ± 1.52	26.59 ± 2.18	0.0228*
BMI (females)		27.86 ± 3.57	25.41 ± 1.99	0.0001*
IOP		15.97 ± 3.86	15.54 ± 3.81	0.606

Notes: * – significant; X^2 – Chi-Square test; mean values compared using t-test
Source: compiled by the authors

Analysis of diabetes duration revealed that the majority in the CSMO group had diabetes for over 20 years (51.2%), while most in the non-CSMO group had a duration of less than 20 years (74.4%). Odds ratios indicated an increasing likelihood of CSMO with longer diabetes durations. This finding suggests that patients with CSMO tend to be older compared to those without CSMO. Furthermore, the observed age difference implies that advancing age might be a risk factor for developing CSMO. This observation aligns with the current understanding that the prevalence of diabetic complications, including CSMO, increases with age due to prolonged exposure to hyperglycaemia and its detrimental effects on retinal vasculature. Regarding BMI, the odds of CSMO were higher in females with increased BMI, while in males, the odds of CSMO occurrence were below one across all BMI categories. Blood glucose levels, HbA1c, and insulin resistance were also found to affect the likelihood of CSMO (Fig. 3). Conversely, neither gender nor IOP demonstrated significant associations with CSMO. This finding suggests that both males and females have similar risks of developing CSMO and that IOP alone does not significantly impact CSMO development in diabetic patients (Fig. 4).

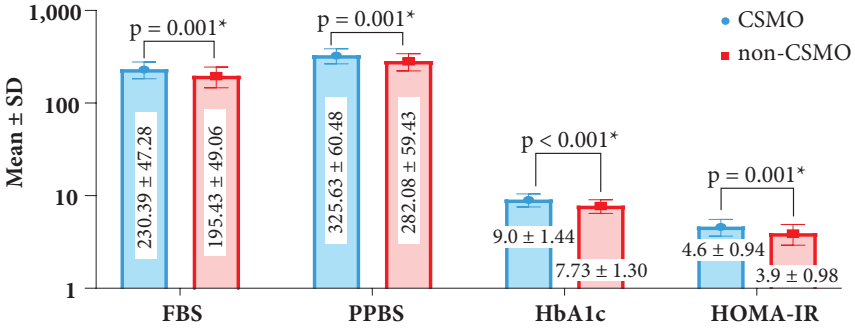

Figure 3. Comparison of blood sugar levels (FBS, PPBS, HbA1c) and HOMA-IR index between patients with CSMO and non-CSMO

Notes: * – significant
Source: compiled by the authors

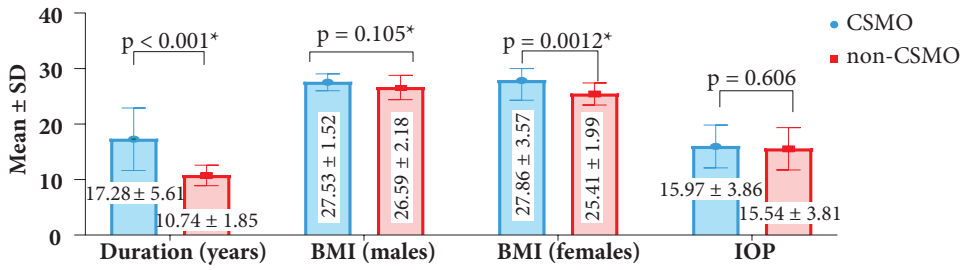

Figure 4. Comparison of mean duration of diabetes (years), BMI (males and females), and IOP between patients with CSMO and non-CSMO group

Notes: * – significant
Source: compiled by the authors

The prolonged duration of diabetes leads to cumulative microvascular damage in the retina, contributing to the pathogenesis of CSMO. This finding highlights the importance of early and sustained glycaemic control to prevent long-term complications, such as CSMO. Significant differences in BMI were observed between the two groups for both males and females, indicating an association between higher BMI and the presence of CSMO in both genders. Elevated BMI is often linked to poor glycaemic control and increased inflammatory markers, both of which can exacerbate retinal vascular permeability and lead to macular oedema. These results underscore the need for weight management as part of comprehensive diabetic care to reduce the risk of CSMO. Patients with CSMO exhibit significantly higher FBS, PPBS, and HbA1c levels, indicating poor glycaemic control. Elevated HOMA-IR values further suggest increased insulin resistance in these patients. These findings underscore the critical role of maintaining strict glycaemic control to prevent the onset of CSMO.

Assessment of glycaemic control revealed significantly higher values in the CSMO group; however, no significant differences were observed in total cholesterol (TC) and LDL levels. Table 2 indicates that patients with CSMO exhibit poorer glycaemic control, as evidenced by higher levels of FBS, PPBS, HbA1c, and HOMA-IR compared to non-CSMO patients. Additionally, CSMO patients have poorer lipid profiles, characterised by higher triglyceride levels and lower HDL cholesterol levels, although there were no significant differences in TC and LDL levels between the two groups. These findings suggest that poor glycaemic control and dyslipidaemia are associated with the presence of clinically significant macular oedema in diabetic patients (Fig. 5).

Table 2. Glycaemic control and Lipid profile assessment of CSMO and non-CSMO patients in both groups

Variables		CSMO	non-CSMO	p-value
Blood sugar	FBS	230.39 ± 47.28	195.43 ± 49.06	0.001*
	PPBS	325.63 ± 60.48	282.08 ± 59.43	0.001*
	HbA1c	9.00 ± 1.44	7.73 ± 1.30	<0.001*
	HOMA-IR	4.60 ± 0.94	3.90 ± 0.98	0.001*
Lipid profile	TC	260.11 ± 46.89	257.37 ± 41.47	0.774
	TG	175.97 ± 39.33	155.85 ± 31.48	0.010*
	HDL	41.95 ± 5.63	50.30 ± 7.85	<0.001*
	LDL	182.97 ± 46.07	175.90 ± 40.29	0.451

Notes: * – significant; mean values compared using t-test
Source: compiled by the authors

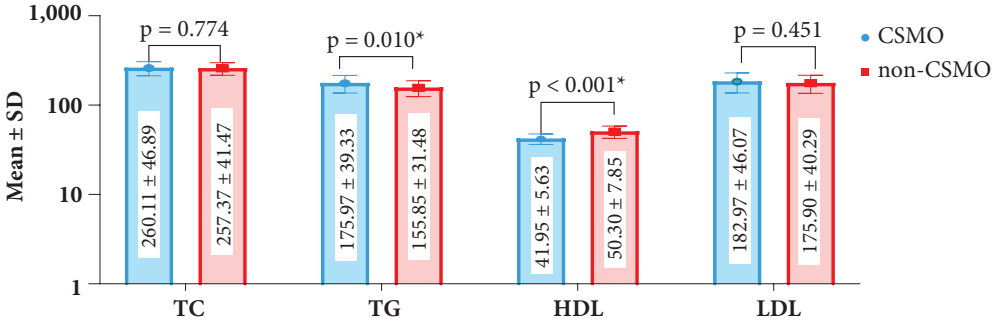

Figure 5. Comparison of lipid levels

Notes: * – significant
Source: compiled by the authors

Measurements of macular thickness in various regions indicated that central macular thickness was significantly higher in the CSMO group (269.79 ± 219.0 vs. 268.91 ± 1.0, p < 0.001), suggesting a potential association between central macular thickness and CSMO. The data indicate a significant increase in central macular thickness in CSMO patients compared to non-CSMO patients. This finding aligns with the characteristic central retinal swelling observed in CSMO. However, no significant differences were found in macular thickness measurements across the other regions (superior, inferior, temporal, nasal, and their inner counterparts), suggesting that macular oedema primarily affects the central region in these patients (Table 3).

Table 3. Macular thickness measurements of CSMO and non-CSMO patients in both groups

Macular thickness	CSMO	non-CSMO	p-value
Central	269.79 ± 219.0	268.91 ± 1.0	<0.001*
Superior	297.73 ± 246.0	298.51 ± 270.0	0.916
Inferior	298.17 ± 245.0	294.84 ± 265.0	0.587
Temporal	274.50 ± 236.0	283.40 ± 252.0	0.199
Nasal	302.30 ± 268.0	309.25 ± 290.0	0.141
Inner superior	324.74 ± 276.0	323.09 ± 310.0	0.881
Inner inferior	322.09 ± 300.0	321.67 ± 308.0	0.954
Inner temporal	306.91 ± 279.0	308.84 ± 295.0	0.813
Inner nasal	318.86 ± 290.0	322.50 ± 312.8	0.610

Notes: * – significant; mean values compared using t-test
Source: compiled by the authors

In the present study, individuals with total cholesterol levels exceeding 240 mg/dL (26 cases) exhibited significantly higher odds of CSMO, with an odds ratio of 9.39. Furthermore, a progressive increase in the odds of CSMO was noted with rising triglyceride levels. Surprisingly, abnormal LDL levels were associated with lower CSMO odds, while elevated HDL levels showed a contrasting trend, indicating higher odds of CSMO in the present study population. These findings underscore the complex relationship between lipid profiles and CSMO risk. Furthermore, the CSMO group had higher odds of developing a BCVA of <3/60 PL positive (Table 4). A multivariate model identified the duration of diabetes, BCVA, and HDL as significantly associated with CSMO, highlighting their potential as independent predictors (Table 5).

Table 4. Macular thickness and best-corrected visual acuity odds ratio in CSMO and non-CSMO patients in both groups

Variables		CSMO	non-CSMO	Odds ratio
Macular thickness	<250 μ (normal)	0	0	-
	>250-260 μ (mild)	9 (20.93%)	22 (51.16%)	Ref.
	>261-270 μ (moderate)	19 (44.18%)	16 (37.20%)	2.90
	>271 μ (severe)	15 (34.88%)	5 (11.62%)	9.17
Best-corrected visual acuity	6/6 to 6/9	0	0	-
	<6/9 to 6/18	10 (11.6%)	29 (45.3%)	Ref.
	<6/18 to 6/60	20 (23.3%)	49 (56.97%)	1.49
	<3/60 to PL+	56 (65.1%)	8 (9.3%)	20.30
	PL negative	0	0	-

Source: compiled by the authors

Table 5. Multivariate analysis of independent predictors for progression of CSMO from non-CSMO groups

Factors	Variable Type	β ± SE	p-value	OR (95% CI)
BMI	Linear	0.513 ± 0.382	0.153	1.86 (0.84-3.56)
Duration of diabetes	Linear	0.942 ± 0.338	0.005*	2.57 (1.33-4.95)
BCVA	Ordinal	-2.327 ± 1.181	0.049	0.10 (0.01-0.99)
PPBS	Linear	0.015 ± 0.012	0.213	1.02 (0.99-1.04)
HbA1c	Linear	0.666 ± 0.40	0.091	1.95 (0.89-4.28)
Insulin resistance #	Linear	0.616 ± 0.579	0.287	1.85 (0.60-5.76)
Triglyceride	Linear	0.044 ± 0.024	0.065	1.05 (1.00-1.10)
HDL	Linear	-0.327 ± 0.129	0.011*	0.72 (0.56-0.93)
Constant	Fixed value	-22.857 ± 11.876	0.052	1.67 (0.84-3.33)

Notes: * – significant; # – binary logistic regression after replacing fasting blood sugar and fasting insulin with insulin resistance; β ± SE – estimated coefficients (β) with standard errors (SE)
Source: compiled by the authors

Proximity to BCVA and duration of diabetes (β = 0.942, p = 0.005, OR = 2.57, 95% CI: 1.33-4.95) were identified as statistically significant predictors of CSMO progression among the factors analysed (β = -2.327, p = 0.049, OR = 0.10, 95% CI: 0.01-0.99). More specifically, the likelihood of developing CSMO increased 2.57-fold with the length of time a patient had the condition, whereas the likelihood decreased ten-fold with suboptimal BCVA. A protective effect was also indicated by the inverse association between elevated levels of HDL cholesterol and the progression of CSMO (β = -0.327, p = 0.011, OR = 0.72, 95% CI: 0.56-0.93). Statistically, CSMO progression could not be predicted by additional variables, including triglyceride levels, BMI, PPBS, HbA1c, or insulin resistance, as determined by this analysis. To identify diabetic patients at greater risk for the progression of CSMO and to enable the implementation of targeted interventions to preserve vision, it is critical to monitor their HDL cholesterol levels, duration of diabetes, and BCVA.

Discussion

Macular oedema, commonly linked to diabetes, reflects chronic damage to retinal neurovascular structures, progressing from peripheral to clinically significant central involvement. Previous studies have highlighted the role of insulin resistance and dyslipidaemia in macular oedema [12, 13]. This cross-sectional study explored the role of lipid levels and insulin resistance in 86 patients with T2DM presenting with macular oedema (43 CSMO, 43 non-CSMO). The majority of patients in the CSMO group were aged over 60 years (65.2%), whereas the majority of non-CSMO patients were aged under 60 years (69.8%). A significant difference was observed in the ages of patients in the two groups [14, 15]. Concerning gender, the majority of CSMO patients in the present study were male (53.5%), while the majority of non-CSMO patients were female (55.9%); however, this difference was not statistically significant. Both L. Feng *et al.* [15] and the present study on T2DM patients with macular oedema highlight the significant impact of age and gender on lipid levels and disease outcomes. L. Feng *et al.* [15] demonstrated age-related variations in lipid profiles with gender-specific trends, while the present study suggests that older age might be associated with more severe macular complications in diabetes. These findings emphasise the need for age and gender-specific approaches in managing lipid levels to prevent or manage diabetes-related complications effectively. In comparison to the present study, R. Raman *et al.* [14] reported that most CSMO and non-CSMO patients were male and did not find a significant difference between the two groups. K. Kamoi *et al.* [16] also did not find a significant difference in the age or gender of patients with CSMO and nonCSMO. Most other studies evaluating the relationship between macular oedema and lipid levels and/or insulin resistance have generally compared a diabetic population with macular oedema to a diabetic population without macular oedema [12, 17], or compared patients with DMO and diabetic retinopathy DR [13], typically in cross-sectional studies, which the present study similarly envisaged. In a study, P. Romero-Aroca *et al.* [18] reported that in the type 1 diabetic population, the mean age of patients with no diabetic retinopathy or macular oedema, those with diabetic retinopathy, and those with macular oedema showed an incremental trend, specifically 31.84, 43.54, and 50.05 years, respectively. In their study, most patients without diabetic retinopathy or macular oedema and those with diabetic retinopathy were male, whereas the majority of patients with macular oedema were female. G.S. Prakash & M. Kothari [19] reported the mean age of diabetic patients with and without CSMO as 57.02 and 56.42 years, respectively, and found no significant difference between the two groups. In both groups, males predominated. The inability to match the age profile of the patients was attributed to the COVID-19 pandemic, which resulted in a limited number of cases with a matched age and sex profile, as OPD services were affected and patient footfall remained low during this study period. Although the mean BMI of both male and female CSMO patients was higher than that of non-CSMO patients, the present study revealed that the odds of CSMO exhibited an incremental trend with increasing BMI. However, among males, the odds of CSMO were lower in those with higher BMI compared to those with lower BMI. The findings of the present study, at least for male patients, replicate the observations of E. Martín-Merino *et al.* [17], who also reported that obesity was associated with a lower risk of macular oedema in diabetic patients. However, regarding the association of higher BMI with CSMO compared to non-CSMO, R. Raman *et al.* [14] did not evaluate the BMI of patients in the two groups. P. Romero-Aroca *et al.* [18] also did not assess BMI or any other marker of obesity. Similarly, G.S. Prakash & M. Kothari [19] did not evaluate BMI or any other marker of obesity. Although K. Kamoi *et al.* [16] evaluated the role of BMI as a discriminatory factor between CSMO and non-CSMO, they failed to determine a significant difference between the two groups. In the present study, the duration of diabetes was significantly higher in patients with CSMO compared to those with non-CSMO. Upon stratified evaluation, the present study found that compared to individuals with a duration of diabetes of less than 10 years, those with a longer duration had higher odds of CSMO. These findings agree with the observations of R. Raman *et al.* [14], which similarly found a significant association of CSMO with the duration of diabetes. However, K. Kamoi *et al.* [16] reported that the duration of diabetes was longer in the non-CSMO group than in the CSMO group; still, they did not find this difference to be statistically significant. In their study, P. RomeroAroca *et al.* [18] found a longer duration of diabetes to be significantly associated with DR and DMO. G.S. Prakash & M. Kothari [19], like the present study, found a significant association between a longer duration of diabetes and CSMO. Regarding the relationship with IOP levels, the present study found that IOP was higher in patients with CSMO; however, this was not

statistically significant. R. Raman *et al.* [14] did not evaluate the role of IOP in their study. Limited data suggests its pathogenic role in the causation and progression of DMO. In the present study, fasting blood sugar, post-prandial blood sugar, HbA1c, and HOMA-IR were higher in patients with CSMO than in those with non-CSMO. With increasing levels of all these markers of glycaemic control and insulin resistance, the odds of CSMO exhibited an incremental trend. Consistent with the findings of the present study, R. Raman *et al.* [14] also found a significant association between poor glycaemic control and CSMO in both univariate and multivariate assessments. However, K. Kamoi *et al.* [16] did not find this association to be significant when evaluating HbA1c. P. Romero-Aroca *et al.* [18] reported that high HbA1c was a significant risk factor for both DR and DMO. G.S. Prakash & M. Kothari [19] found mean HbA1c levels to be significantly higher in patients with CSMO compared to the control group, with no significant association between fasting and post-prandial blood glucose levels and CSMO. In the present study, although mean TC, TG, and LDL levels were higher and mean HDL was lower in patients with CSMO compared to those with non-CSMO, the difference was statistically significant for TG and HDL levels only. Increasing total cholesterol and triglyceride levels were associated with an increase in the odds of CSMO. However, for LDL levels, the odds of developing CSMO were lower at higher LDL levels than those at optimal LDL levels. When HDL was considered, the present study found increased odds of developing CSMO. This finding could be attributed to the small sample size, indicating a need for further research. Regarding lipid levels, R. Raman *et al.* [14] discovered a significant association between high total serum cholesterol, high serum LDL cholesterol, and high serum non-HDL cholesterol with CSMO during univariate assessment. In the multivariate analysis, high serum LDL cholesterol, high serum non-HDL cholesterol, and a high cholesterol ratio were found to be related to CSMO. It is worth noting that E. Martín-Merino *et al.* [17] found a strong correlation between DMO and elevated levels of total cholesterol and LDL. However, they discovered that elevated triglyceride levels were strongly linked to a reduced risk of DMO. In the present study, higher LDL levels were associated with a lower risk of DMO. On the other hand, K. Kamoi *et al.* [16] did not find a significant discriminant role of lipid levels in distinguishing CSMO from non-CSMO. P. Romero-Aroca *et al.* [18] found no significant association of these lipids with DMO in their study. G.S. Prakash & M. Kothari [19] reported that lipid levels (higher TC, TG, LDL, VLDL and lower HDL were significantly associated with CSMO. In multivariate analysis, after adjusting for BMI, PPBS, HbA1c, insulin resistance, and triglyceride levels, BCVA and HDL emerged as independent predictors of CSMO only during diabetes. In their study, R. Raman *et al.* [14] found high serum LDL cholesterol, high serum non-HDL cholesterol, and high cholesterol ratio related to non-CSMO, poor glycaemic control, and high serum total cholesterol related to CSMO. Both the present study and the research by I. Vivsiana & M. Marushchak [20] explore the complexities of lipid profiles in patients with T2DM, emphasising the influence of additional comorbidities on lipid levels. Findings indicate significant differences in TG and HDL levels between T2DM patients with and without CSMO, linking higher TG and lower HDL levels to an increased likelihood of CSMO. Interestingly, higher LDL levels correlated with lower odds of CSMO, suggesting a more nuanced role of LDL in diabetic retinopathy. I. Vivsiana & M. Marushchak's [20] study reinforces the impact of comorbid conditions such as obesity and hypertension on worsening lipid profiles in T2DM patients, showing significantly higher levels of total cholesterol and TG among those with additional comorbidities. Together, these studies highlight the critical role of managing lipid levels in T2DM patients, particularly those with additional risk factors, to mitigate complications such as macular oedema and underscore the importance of targeted therapeutic strategies addressing dyslipidaemia in the presence of comorbid conditions. Similar to the present study, a significant reduction in the number of independent predictors for the prediction of CSMO was observed in both models. The present study also found poor BCVA to be associated with CSMO; however, this relationship does not require further explanation owing to its temporal nature. The present study's findings endorse the role of lipid dysregulation and insulin resistance in the progression of CSMO when compared with non-CSMO.

Conclusions

In this cross-sectional study, the analysis of baseline characteristics, glycaemic control, and lipid profiles between CSMO and non-CSMO groups provided critical insights. Key findings include a significant association between older age and longer duration of diabetes with the presence of CSMO, indicating that prolonged hyperglycaemia and ageing contribute to the development of this complication. Higher BMI, particularly in females, and poor glycaemic control, as reflected by elevated FBS, PPBS, HbA1c, and HOMA-IR levels, were significant risk factors for CSMO. These findings emphasise the importance of maintaining strict glycaemic control and managing weight to reduce the risk of CSMO. Lipid profile analysis revealed that higher triglyceride levels and lower HDL levels are associated with CSMO, highlighting dyslipidaemia as a contributing factor. The study also demonstrated that central macular thickness was significantly higher in CSMO patients, consistent with the characteristic retinal swelling observed in this condition. However, no significant differences were found in macular thickness across other regions, indicating that CSMO primarily affects the central retina. Multivariate analysis identified the duration of diabetes, BCVA, and HDL levels as independent predictors of CSMO progression. Specifically, a longer duration of diabetes and poorer BCVA increased the risk, while higher HDL levels had a protective effect.

These findings underscore the need for comprehensive diabetes management, focusing on early detection and sustained glycaemic control, weight management, and targeted lipid management to prevent the onset and progression of CSMO. Regular monitoring of these parameters in diabetic patients can help identify those at higher risk for CSMO, allowing for timely interventions to preserve vision and improve the quality of life for these individuals. The study highlights the multifactorial nature of CSMO, necessitating a holistic approach to diabetes care to mitigate this vision-threatening complication. The prospects for further research lie in conducting long-term longitudinal studies with larger sample sizes and focusing on personalised approaches to managing patients with T2DM to prevent or mitigate vision-threatening conditions such as CSMO.

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Conflict of Interest
None.

References
[1] GBD 2021 Diabetes Collaborators. Global, regional, and national burden of diabetes from 1990 to 2021, with projections of prevalence to 2050: A systematic analysis for the Global Burden of Disease Study 2021. Lancet. 2023;402(10397):203–34. DOI: 10.1016/S0140-6736(23)01301-6
[2] International Diabetes Federation (IDF) Diabetes Atlas [Internet]. [cited 2024 October 8]. Available from: https://diabetesatlas.org/atlas/tenth-edition/
[3] Kumar A, Gangwar R, Zargar AA, Kumar R, Sharma A. Prevalence of diabetes in India: A review of IDF Diabetes Atlas 10th edition. Curr Diabetes Rev. 2024;20(1):e130423215752. DOI: 10.2174/1573399819666230413094200
[4] Chan LKY, Lin SS, Chan F, Ng DS. Optimizing treatment for diabetic macular edema during cataract surgery. Front Endocrinol (Lausanne). 2023;14:1106706. DOI: 10.3389/fendo.2023.1106706
[5] Rao H, Jalali JA, Johnston TP, Koulen P. Emerging roles of dyslipidemia and hyperglycemia in diabetic retinopathy: Molecular mechanisms and clinical perspectives. Front Endocrinol (Lausanne). 2021;12:620045. DOI: 10.3389/fendo.2021.620045
[6] Park SJ, Park DH. Revisiting lipids in retinal diseases: A focused review on age-related macular degeneration and diabetic retinopathy. J Lipid Atheroscler. 2020;9(3):406–18. DOI: 10.12997/jla.2020.9.3.406
[7] Xu YX, Pu SD, Zhang YT, Tong XW, Sun XT, Shan YY, et al. Insulin resistance is associated with the presence and severity of retinopathy in patients with type 2 diabetes. Clin Exp Ophthalmol. 2024;52(1):63–77. DOI: 10.1111/ceo.14344
[8] Meng C, Xing Y, Huo L, Ma H. Relationship between estimated glucose disposal rate and type 2 diabetic retinopathy. Diabetes Metab Syndr Obes. 2023;16:807–18. DOI: 10.2147/DMSO.S395818
[9] Behera R, Biswas J, Ray S, Sarkar I, Dey AK. Association of serum lipid profile and body mass index with diabetic retinopathy in type ii diabetes mellitus – a cross sectional study. Eur J Cardiovasc Med. 2023;13(4):419–24.
[10] Alvi R, Memon MS, Shera S, Mumtaz SN, Shaikh SA, Fahim MF. Visual outcome of laser treatment in diabetic macular edema: Study from an Urban Diabetes Care Center. Pak J Med Sci. 2016;32(5):1229–33. DOI: 10.12669/pjms.325.10597
[11] Hellmann F, Verdi M, Schlemper BR Jr, Caponi S. 50th anniversary of the Declaration of Helsinki: The double standard was introduced. Arch Med Res. 2014;45(7):600–1. DOI: 10.1016/j.arcmed.2014.10.005
[12] Luxmi S, Ritika M, Lubna A, Pragati G, Lal BB. Diabetic macular edema and its association to systemic risk factors in an urban north Indian population. J Clin Ophthalmol. 2018;2(2):86–91. DOI: 10.35841/clinical-ophthalmology.2.2.86-91
[13] Graue-Hernandez EO, Rivera-De-La-Parra D, Hernandez-Jimenez S, Aguilar-Salinas CA, Kershenobich-Stalnikowitz D, Jimenez-Corona A. Prevalence and associated risk factors of diabetic retinopathy and macular oedema in patients recently diagnosed with type 2 diabetes. BMJ Open Ophthalmol. 2020;5(1):e000304. DOI: 10.1136/bmjophth-2019-000304
[14] Raman R, Rani PK, Kulothungan V, Rachepalle SR, Kumaramanickavel G, Sharma T. Influence of serum lipids on clinically significant versus nonclinically significant macular edema: SN-DREAMS Report number 13. Ophthalmology. 2010;117(4):766–72. DOI: 10.1016/j.ophtha.2009.09.005
[15] Feng L, Nian S, Tong Z, Zhu Y, Li Y, Zhang C, et al. Age-related trends in lipid levels: A large-scale cross-sectional study of the general Chinese population. BMJ Open. 2020;10(3):e034226. DOI: 10.1136/bmjopen-2019-034226
[16] Kamoi K, Takeda K, Hashimoto K, Tanaka R, Okuyama S. Identifying risk factors for clinically significant diabetic macula edema in patients with type 2 diabetes mellitus. Curr Diabetes Rev. 2013;9(3):209–17. DOI: 10.2174/1573399811309030002

[17] Martín-Merino E, Fortuny J, Rivero-Ferrer E, Lind M, Garcia-Rodriguez LA. Risk factors for diabetic macular oedema in type 2 diabetes: A case-control study in a United Kingdom primary care setting. Prim Care Diabetes. 2017;11(3):288–96. DOI: 10.1016/j.pcd.2017.03.002
[18] Romero-Aroca P, Baget-Bernaldiz M, Fernandez-Ballart J, Plana-Gil N, Soler-Lluis N, Mendez-Marin I, et al. Ten-year incidence of diabetic retinopathy and macular edema. Risk factors in a sample of people with type 1 diabetes. Diabetes Res Clin Pract. 2011;94(1):126–32. DOI: 10.1016/j.diabres.2011.07.004
[19] Prakash GS, Kothari M. Risk factors associated with clinically significant macular edema in patients with type 2 diabetes mellitus. Int J Sci Stud. 2016;3(12):120–4.
[20] Vivsiana I, Marushchak M. Does the lipid profile depend on the comorbidity of overweight/obesity and arterial hypertension in patients with type 2 diabetes mellitus? Bull Med Biol Res. 2021;3(3):5–11. DOI: 10.11603/bmbr.2706-6290.2021.3.12560

Порівняння інсулінової резистентності та ліпідного профілю при клінічно значущому та клінічно незначущому макулярному набряку у пацієнтів з цукровим діабетом типу 2

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Анотація. Розлади, пов'язані зі способом життя, зокрема діабет, є серйозними викликами для глобального здоров'я. Діабетичний макулярний набряк, як мікросудинне ускладнення, підкреслює важливість управління інсуліновою резистентністю та гіперліпідемією для досягнення ефективних клінічних результатів. Розуміння взаємодії цих факторів є ключовим для оптимізації терапевтичних стратегій та покращення медичної допомоги. Це перехресне дослідження мало на меті порівняти інсулінову резистентність і ліпідні профілі

між клінічно значущим макулярним набряком і клінічно незначущим макулярним набряком у пацієнтів з цукровим діабетом типу 2. Дослідження може призвести до раннього виявлення та класифікації макулярного набряку, що дозволяє здійснювати більш своєчасні та специфічні втручання. Загалом, 86 пацієнтів з діабетом типу 2 з макулярним набряком були поділені на дві групи: клінічно значущий макулярний набряк та клінічно незначущий макулярний набряк. Було зафіксовано комплексні демографічні дані, медичні історії та поточні режими медикаментозного лікування. Оцінювалися рівень глікемічного контролю та ліпідні профілі, а офтальмологічні оцінки включали вимірювання гостроти зору та оцінки внутрішньоочного тиску. Була виявлена значна різниця між групами клінічно значущого макулярного набряку і клінічно незначущого макулярного набряку, особливо у тривалості діабету, індексі маси тіла, рівнях глюкози в крові та ліпідних профілях. Пацієнти з клінічно значущим макулярним набряком демонстрували більшу тривалість діабету, вищий індекс маси тіла та підвищені рівні глюкози в крові. Рівні тригліцеридів були значно вищими, тоді як рівні ліпопротеїнів високої щільності були нижчими у пацієнтів з клінічно значущим макулярним набряком. Мультиваріантний аналіз виявив значні асоціації між ймовірністю клінічно значущого макулярного набряку та тривалістю діабету, гостротою зору та рівнями ліпопротеїнів високої щільності, що вказує на їх потенційні ризики для розвитку клінічно значущого макулярного набряку. Дослідження підкреслює важливість управління інсуліновою резистентністю та дисліпідемією у пацієнтів з цукровим діабетом типу 2 для зменшення ризику клінічно значущого макулярного набряку

Ключові слова: діабетичний макулярний набряк; дисліпідемія; захворювання сітківки; діабетичні нейропатії; оклюзія вен сітківки