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ORIGINAL ARTICLE

Impact of obesity on ocular parameters: A comparative study of normal-weight and obese adults

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Abstract

Purpose: The aim of this study was to investigate the effect of obesity on various ocular examination parameters in a comparative analysis between normal-weight and obese adults.

Methods: Thirty-two normal-weight and 64 obese adults were included in the study. Detailed ophthalmologic examination, including intraocular pressure (IOP), central corneal thickness (CCT), keratometry, anterior chamber depth (ACD), lens thickness (LT), and axial length (AL) by A-mode ocular ultrasonography; and macular and retinal nerve fiber layer thickness (RNFL) by spectral domain optical coherence tomography were compared between normal-weight and obese adults. Obese individuals were further stratified into Class I-II and Class III obesity categories, and the ocular parameters were compared between these three groups.

Results: IOP was significantly higher in the obese group than in the healthy controls (16.7±2.4 vs. 15.1±2.2 mmHg, p=0.002), whereas CCT, keratometry values, ACD, LT, AL, and RNFL thicknesses did not differ between the groups. The mean retinal thicknesses in the inner retinal ring (3 mm) at superior (p=010), nasal (p=0.04), and temporal (p=0.002) quadrants were lower in the class III obese group compared to class I obese individuals. ACD was significantly narrower in class III obese group (p=0.01). Body mass index was significantly correlated with IOP (p<0.001).

Conclusion: Obesity is associated with elevated IOP and signs of retinopathy. An increase in the severity of obesity was found to be associated with a decrease in macular thickness and a narrowing of the anterior chamber.

Keywords: Body mass index; intraocular pressure; obesity; ocular examination.

Obesity is a chronic condition with a rapidly increasing prevalence, now considered a worldwide epidemic. According to a 2016 report by the World Health Organization (WHO), nearly 2 billion people globally are overweight, with 650 million classified as obese.[1] Among European countries, Türkiye has the highest obesity prevalence at 30.3%.[2] Although obesity affects individuals across all age groups, there has been a significant surge in its prevalence among children and young adults.[3] Body mass index (BMI), calculated as weight (kg) divided by the square of height (m^2), is the primary metric used to assess the degree of obesity. The WHO classifies BMI into different categories: underweight (BMI <18.5), normal-weight (BMI between 18.5 and 24.9), overweight (BMI between 25 and 29.9), and obese (Class I: BMI 30–34.9, Class II: BMI 35–39.9, and Class III: BMI 40 or higher).

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Obesity has a well-established association with systemic diseases such as hypertension, hyperlipidemia, and coronary artery disease. In addition, numerous studies have found a correlation between obesity and a range of ocular diseases, including cataracts, age-related macular degeneration, diabetic retinopathy, and glaucoma, requiring specific attentions to predict, diagnose, and manage these adverse outcomes in obese individuals.[4-6] Although a clear pathophysiological explanation for the association of obesity with ocular diseases is currently lacking, chronic oxidative stress leading to the failure of both the mechanical and vascular components of the eye is the most plausible explanation. Yet, obesity may impair vision even in the absence of associated systemic diseases or in preclinical stages.[4] The previous studies has been demonstrated that a higher BMI is related with an increase in intraocular pressure (IOP) and reduced retinal nerve fiber layer (RNFL) thickness, that BMI influences ocular biometry outcomes, and that higher BMI is associated with an increased retinal thickness.[7,8] However, these studies have primarily focused on comparing individuals with morbid obesity (BMI 40 or higher) to normal-weight individuals or have used various BMI thresholds to define normal-weight and obesity. Furthermore, anterior segment and posterior segment parameters were often evaluated separately in most of these studies. Therefore, our study aims to take a comprehensive approach by collectively evaluating all ophthalmic examination findings to understand the integrated impact of obesity on the eye. Furthermore, we intend to examine the association between the severity of obesity and ocular findings.

Materials and Methods

Study Population

This retrospective study recruited obese adult patients who visited the ophthalmology outpatient clinic of Karadeniz Eregli State Hospital between January 2021 and January 2023, underwent a comprehensive ophthalmological examination, and had medical records of anthropometric measures in the dietitian department of the hospital showing that they met the WHO's criteria for obesity (BMI ≥30). The inclusion criteria consisted of patients without additional systemic diseases or medically/surgically treated eye conditions, who had undergone a comprehensive ophthalmological examination encompassing measurements of IOP, central corneal thickness (CCT), keratometry, axial length (AU), anterior chamber depth (ACD), lens thickness (LT), RNFL thickness, and central macular thickness (CMT). Participants were excluded if they had refractive error >2 diopters (D) spherical equivalent and if they have intracranial hypertension, neurological diseases, and papilledema. Due to the possible effect of diurnal fluctuation of IOP, patients were included only if their IOP measurements were obtained between 9 am and 12 am. Age- and sex-matched healthy individuals with a BMI between 18.5 and 24.99 and no additional systemic or ocular diseases were recruited as the control group. Obesity severity was classified as class I (BMI 30–34.9), class II (BMI 35–39.9), and class III (BMI ≥40). The study was performed in adherence to the tenets of the Declaration of Helsinki and approved by the Local Ethics Committee. Informed consent was waived due to the retrospective nature of the study.

Ophthalmic Examination

In this study, only the right eyes of all participants were included in the study. A comprehensive ophthalmic examination was conducted, which included the assessment of best-corrected visual acuity, keratometry obtained through an autorefractometer-keratometer (RK-F2, Canon, Tokyo, Japan), IOP measured using a non-contact tonometer (TX-20, Canon, Tokyo, Japan), and CCT measured with ultrasonic pachymetry (PalmScan AP2000, MMD, California, USA). Both IOP and CCT measurements were obtained 5 times, and the mean of these measurements was selected for statistical analysis. In addition, ACD, LT, and AL were evaluated using ocular ultrasonography (A/B Scan 5500 740, Sonomed Inc., New York, USA), while CMT and RNFL measurements were obtained using spectral domain optical coherence tomography (SD-OCT) (RTVue, Optovue Inc., Fremont, California, USA). All participants had good-quality SD-OCT images, with a signal strength of ≥50, good centering, and uniform brightness. The average retinal thicknesses of the nine macular sectors as defined by the early treatment diabetic retinopathy study (ETDRS) as the circular areas of 1 mm, 3 mm, and 6 mm diameters in the image section passing through the center of the macula were recorded. RNFL thickness measurements were determined in the temporal (T), nasal (N), inferior (I), and superior (S) quadrants of the optic nerve.

Statistical Analysis

Statistical analysis was performed using SPSS version 25.0 statistical software (IBM, Armonk, NY). The Shapiro–Wilk

test was used to examine the data's normality. Continuous variables were expressed as means standard deviation and compared with Student t or Analysis of Variance (ANOVA) tests in univariate analysis. One-way ANOVA with Bonferroni post hoc correction was used for comparisons between obese subgroups. Pearson correlation test was used to determine the linear relationship between variables. Variables showing a significant difference in the univariate analysis were included in the stepwise multivariate logistic regression model. Odds ratios and 95% confidence intervals were calculated for each risk factor. The level of statistical significance was set at <0.05.

Results

The obese group (n=64) and healthy control group (n=32) were comparable in terms of age (35.5±10.5 years, range 18–60 years vs. 34.8±11.2 years, range 18–60 years; p>0.05) and sex distribution (40 females and 24 males in the obese group, compared to 20 females and 12 males in the control group; p>0.05). Among the participants, 22 individuals (34.4% of all obese subjects) were classified as class I obese, 26 individuals (40.6%) as class II obese, and 16 individuals (25.0%) fell into the class III obese category. The mean weight of the obese and normal-weight groups differed significantly (104.4±16.8 kg for obese and 64.4±10.8 kg for controls, p=0.03), while there was no significant difference in terms of height (167.7±8.8 cm for obese and 168.3±9.0 cm for controls, p=0.75). The mean BMI scores were 37.0 and 22.5 for the obese and normal-weight groups, respectively (p>0.05).

The IOP was found to be significantly higher in the obese group compared to the healthy controls (16.7±2.4 mmHg vs. 15.1±2.2 mmHg, p=0.002). However, no significant differences were observed between the groups in terms of CCT, keratometry values, ACD, LT, and AL (p>0.05 for all). The IOP was found to be significant in predicting being obese in the multivariate model (p=0.001; OR=1.517, CI: 1.192–1.919). With a one-unit increase in IOP, the probability of being obese increases by approximately 52%. The measurements of IOP, corneal parameters, and ocular biometry of the study group are given in Table 1. Table 2 shows the results of the multivariate logistic regression analysis.

Table 3 demonstrates that there were no significant differences observed in the macular thickness values of the ETDRS 9 field macular thickness map or the four sector RNFL thicknesses between normal-weight and obese individuals.

Table 1. Intraocular pressure, corneal, and ocular biometry parameters of normal-weight and obese individuals

	Normal-weight	Obese	P-value
IOP	15.1±2.2	16.7±2.4	0.003
CCT	549±34.4	541±36.4	0.29
Flat K	42.6±1.6	42.8±1.5	0.66
Steep K	43.4±1.7	43.7±1.5	0.38
ACD	3.34±0.2	3.39±0.3	0.46
Lens thickness	4.08±0.2	4.04±0.3	0.45
AL	23.2±0.6	23.2±1.0	0.97

ACD: Anterior chamber depth; AL: Axial length; CCT: Central corneal thickness; IOP: Intraocular pressure; K: Keratometry; LT: Lens thickness.

Table 2. Predictors of being an obese patients according to the multiple logistic regression model

Variable	OR	95% CI		P-value
		Lower	Upper	
IOP	1.517	1.192	1.919	0.001
ACD	2.625	0.583	11.818	0.20
Macular thickness (medial ring)				
Superior	1.041	0.966	1.121	0.28
Nasal	0.991	0.948	1.036	0.69
Temporal	0.972	0.897	1.053	0.48
Macular thickness (outer ring)				
Nasal	0.997	0.937	1.059	0.92

ACD: Anterior chamber depth; CI: Confidence interval; IOP: Intraocular pressure; OR: Odds ratio. *Parameters that reached a statistical significance in univariate logistic regression analysis were studied in this model

Table 4 shows the significant differences in IOP, macular thickness at the superior, nasal, and temporal quadrants of the inner 3 mm ring, as well as at the nasal quadrant of the outer 6 mm ring, and ACD were observed within obese subgroups. Post hoc analyses indicated that these differences were primarily attributed to variances between the class I and class III obese groups, except for ACD (p=0.004 for IOP, p=0.04 for superior quadrant of the inner ring, p=0.014 for nasal quadrant of the inner ring, p=0.036 for temporal quadrant of the inner ring, and p=0.036 for nasal quadrant of the outer ring). A significant difference in ACD in the class III obese group compared to both class I and class II obese individuals was identified (p=0.02 for both).

Pearson's correlation analysis revealed a weak but significant correlation between IOP and BMI (r=0.39, p<0.001). However, no significant correlation was found between BMI and other clinical parameters. Table 5 shows correlation analysis of study parameters with BMI.

Table 3. Retinal and optic nerve thickness parameters of normal-weight and obese individuals

	Normal-weight	Obese	P-value
Fovea Central Subfield Thickness, µm (1 mm diameter)	242.6±23.6	249.4±19.6	0.14
Medial ring thicknesses, µm (3 mm diameter)			
Superior	312.0±17.3	313.9±15.0	0.57
Nasal	315.7±16.0	317.2±15.6	0.67
Inferior	305.0±17.3	302.0±19.8	0.46
Temporal	297.6±15.3	297.7±13.0	0.97
Outer ring thicknesses, µm (6 mm diameter)			
Superior	283.6±15.1	282.0±15.0	0.62
Nasal	301.9±14.4	301.2±13.3	0.80
Inferior	284.3±17.8	285.0±14.2	0.85
Temporal	278.1±16.2	277.5±15.5	0.85
Retinal nerve fiber layer thickness, µm			
Superior	124.3±13.7	126.5±15.5	0.50
Nasal	78.9±7.4	80.1±11.0	0.56
Inferior	129.7±16.1	133.3±19.7	0.37
Temporal	83.0±7.7	84.6±9.4	0.41

Table 4. Comparison of all ocular examination parameters among class I, class II, and class III obese individuals

	Obese class I	Obese class II	Obese class III	P-value
IOP	15.6±2.1	16.6±2.6	18.2±1.9	0.005
CCT	540.7±28.8	541.7±45.5	542.3±30.9	0.91
Flat K	42.8±1.3	42.8±1.7	42.6±1.4	0.99
Steep K	43.6±1.4	43.9±1.8	43.6±1.4	0.76
ACD	3.4±0.3	3.4±0.2	3.1±0.2	0.01
LT	4.07±0.3	3.96±0.3	4.11±0.2	0.31
AL	23.3±0.5	23.5±1.3	22.9±0.6	0.20
Fovea central subfield Thickness (1 mm diameter, C1)	256.0±18.3	245.9±17.9	245.9±22.7	0.15
Medial ring thicknesses (3 mm diameter)				
Superior	320.0±12.5	312.1±15.2	308.4±16.0	0.04
Nasal	324.7±14.1	314.9±14.0	310.5±16.5	0.01
Inferior	309.0±19.0	298.1±20.7	298.8±17.7	0.12
Temporal	304.5±11.5	297.5±11.8	288.8±15.6	0.002
Outer ring thicknesses (6 mm diameter)				
Superior	285.0±13.0	281.1±15.0	279.5±17.5	0.50
Nasal	306.9±12.5	299.5±11.9	296.0±14.3	0.03
Inferior	289.9±13.5	282.3±13.3	282.5±15.5	0.13
Temporal	280.8±15.0	276.6±14.9	274.3±17.1	0.42
Retinal nerve fiber layer thickness				
Superior	126.0±15.7	126.1±15.2	127.5±16.6	0.95
Nasal	78.3±11.5	79.0±9.0	84.5±12.7	0.19
Inferior	135.5±18.8	128.9±15.0	137.5±26.6	0.32
Temporal	84.9±8.9	83.7±10.2	85.5±9.1	0.81

P-values were obtained by one-way Analysis of variance test. ACD: Anterior chamber depth; AL: Axial length; CCT: Central corneal thickness; IOP: Intraocular pressure; K: Keratometry; LT: Lens thickness.

Discussion

Our study reveals a significant increase in IOP among obese individuals compared to healthy controls, while no such differences were observed in other ocular parameters such as corneal thickness, keratometry values, and ocular biometry. However, individuals with severe obesity demonstrated a decrease in retinal thickness in certain quadrants and a narrower ACD. Notably, BMI was found to correlate significantly with IOP.

Numerous epidemiological studies have identified obesity

Table 5. Correlation of body mass index with clinical parameters

	R	P-value
IOP	0.39	<0.001
CCT	−0.08	0.38
ACD	0.004	0.97
LT	−0.04	0.65
AL	−0.02	0.84
Fovea central subfield thickness (1mm diameter, C1)	0.13	0.20
Retinal nerve fiber layer thickness (mean value)	0.16	0.11

The mean retinal nerve fiber layer thickness was determined by dividing the sum of the thicknesses of all quadrants by four. ACD: Anterior chamber depth; AL: Axial length; CCT: Central corneal thickness; IOP: Intraocular pressure; K: Keratometry; LT: Lens thickness.

as an independent risk factor for elevated IOP. The Beaver Dam Eye Study, a population-based study conducted in the United States, found a positive correlation between IOP and BMI.[9] This association was also observed in a large Japanese population, both cross-sectionally and longitudinally.[10] Our study corroborates these findings, revealing a significant increase in IOP among obese individuals compared to healthy controls. Notably, the most pronounced increase in IOP was observed in individuals with class III obesity. Among all the parameters, we examined, IOP was the only one that showed a significant, although weak, correlation with BMI. The mechanisms through which obesity influences IOP are fully understood. Proposed theories include the accumulation of adipose tissue within the orbit and increased episcleral venous pressure, both of which could impede the outflow of aqueous humor.[6,11] This "mass effect" mechanism is similar to the causes of increased IOP observed in conditions such as Graves' ophthalmopathy, caroticocavernous fistula, and retrobulbar injections.[12] Supporting this theory, Stojanov et al.[13] found that obese individuals have significantly higher volumes of retrobulbar adipose tissue, which positively correlates with higher IOP. Obesity-related alterations in autonomic vasoregulation and endothelial damage may further alter ophthalmic blood flow, potentially increasing the vulnerability of the optic nerve head to injury under increased or even normal IOP.[14] Elevated blood viscosity, which may increase resistance in the episcleral veins, is another possible factor.[15] However, the relationship between obesity and IOP is not universally agreed on. The Barbados Eye Study reported only a marginally significant positive association between higher BMI and IOP, while several other studies found similar IOP levels across subjects with different BMI values.[16-18] These discrepancies could potentially be attributed to variations in study populations and the distinct research methodologies employed.

Corneal thickness is a crucial factor in the clinical evaluation of several eye disorders, particularly glaucoma. However, studies have produced differing results regarding the relationship between CCT and BMI in obese individuals. The Gutenberg Health Study, for instance, reported a positive correlation between CCT and BMI, suggesting that the metabolic status in obesity might alter corneal endothelial physiology, leading to an increase in CCT.[19] This finding was echoed by the Funagata study from Japan, which also identified significantly higher CCT in obese adults.[20] Contrarily, several studies have found no significant change in corneal parameters between normal-weight and obese individuals.[21,22] Our study aligns with these findings, as we observed no significant difference in CCT between normal-weight and obese individuals, nor within obese subgroups. Furthermore, consistent with the findings of Panon et al.,[23] our study found no significant difference in corneal curvature between the obese group and the controls.

While our study did not find a significant difference in ACD, LT, and AL between normal-weight and obese individuals, our subgroup analysis among obese individuals revealed a significantly narrower ACD in class III obese individuals. Given that the mean height of individuals across all three subgroups was similar (p=0.41), we can rule out the effect of height on AL and ACD. Although not statistically significant, class III obese individuals had a slightly shorter mean AL compared to the other two subgroups, which may account for the decreased ACD observed in our study. This suggests that the "mass effect" associated with increased levels of obesity could potentially explain the decrease in ACD in morbidly obese individuals, who have been noted to have more hyperopic vision and shorter vitreous chambers.[24,25] In a study comparing 34 obese individuals with age- and gender-matched healthy individuals, Gunes et al.[21] found a significantly reduced ACD in the obese group (mean BMI 39.8). In our study, class III obese individuals (mean BMI 37.0) contributed less to the findings of the obese group due to the small number of sample size, which could explain the difference between our results and those of Gunes et al.

Recent studies have proposed retinal and choroidal thickness as potential predictors of an increased risk of concurrent or future vascular pathologies in patients with obesity.[26] However, findings regarding macular thickness

in obesity have been inconsistent. For instance, Teberik et al.[27] reported reduced macular thickness in both the nasal and temporal quadrants in morbidly obese adult patients, while Dogan et al.[28] found thinner CMT in severely obese adults (BMI ≥40). Conversely, Panon et al.[23] reported significantly higher CMT in overweight subjects compared to those of normal-weight. In our study, we observed a decrease in retinal thickness in the nasal, temporal, and superior quadrants within the inner 3 mm ring as the degree of obesity increased. However, this decrease was significant only between class I and class III obese subjects. However, only the nasal quadrant within the outer 6 mm ring was significantly thinner in class III obese individuals. These findings suggest that the inner retinal ring may be more susceptible to the severity of obesity. A study by Wei et al.[29] found a significant difference in the macular thickness within the inner 3 mm ring, but not the 6 mm outer ring, in diabetic patients without retinopathy compared to healthy controls. This result aligns with our findings and may suggest that the inner retina may be more susceptible to metabolic changes. However, further studies evaluating macular thickness maps in obese patients are recommended to confirm these findings.

The impact of obesity on peripapillary RNFL thickness has been a subject of debate, with inconsistent findings across different studies and patient groups. The EPIC-Norfolk Eye Study group found that males with higher BMI had thinner RNFL thickness, but there was no correlation between IOP and RNFL measurements.[30] In a study by Pacheco-Cervera et al.,[31] a negative correlation was found between RNFL thickness and BMI, leptin, and interleukin-6 levels among overweight and obese children. However, RNFL thickness in the temporal quadrant did not differ between groups. The authors attributed this to the critical role of inflammatory factors in retinal cell damage but did not provide a clear explanation of how inflammatory cytokines and oxidative stress affect some quadrants while sparing others. Conversely, studies reporting increased RNFL thickness have hypothesized that protective hormones secreted from orbital fat, such as estrogen, may provide neuroprotection in obese individuals.[32] In our study, we did not observe a significant difference in RNFL thickness between the normal-weight and obese groups, or between the obese subgroups.

The strengths of our study lie in its inclusive approach, encompassing not just morbidly obese individuals but all categories of obesity. We also conducted an internal analysis within the obese group and compared both anterior and posterior segment ocular parameters. However, our single-center study has several limitations. We did not include exophthalmometric measures to assess potential eyeball protrusion caused by increased retrobulbar fat pressure. The relatively small size of our obese subgroups is another limitation. In addition, we were unable to evaluate choroidal thickness with enhanced deep imaging-OCT or foveal vasculature with OCT-angiography due to the lack of related software. These evaluations could potentially reveal changes that might affect retinal and RNFL thickness. We did not include systemic blood tests related to obesity, such as leptin and adiponectin, in our analysis, but this is beyond the scope of our study.

Conclusion

Our study delineates a significant association between obesity and IOP, as well as specific manifestations of retinopathy on particular areas of macula. To elucidate the potential influence of obesity severity on ocular parameters, further studies with larger cohorts are required.

Ethics Committee Approval: This study was approved by Zonguldak Bulent Ecevit University Faculty of Medicine Ethics Committee (Date: 19/07/2023, Number: 2023/14).

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