

# The effects of nasal septum deviation on eye posterior segment finding

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## Abstract

**Aim:** Deviation of the nasal septum, which can cause upper respiratory tract obstruction and systemic hypoxia, may also affect the eye. The aim of this study was to detect changes in posterior segment parameters of the eye in patients with nasal septal deviation.

**Material and Methods:** The study included 50 patients with nasal septal deviation, and a control group of 30 healthy subjects with no nasal septal deviation. Choroidal thickness, central macular thickness and retinal nerve fiber layer thickness were measured with Optical Coherence Tomography.

**Results:** Choroidal thickness was significantly lower in patients with severe nasal septal deviation compared to the control group. ( $p=0.001$ ) There is no statistically significant change in other measurements.

**Conclusion:** The study results suggest that marked nasal septal deviation may lead to significant hypoxia and sympathetic activation, resulting in deterioration of the choroidal blood flow and consequent choroidal thickening.

**Keywords:** Choroidal thickness; EDI-OCT; nasal septal deviation; NOSE score; sleep-disordered breathing

## INTRODUCTION

Nasal septum deviation (NSD) is very widespread otorhinolaryngological condition. Bilateral or unilateral disruptions of the bony and/or cartilage septum may cause disruption to the nose airflow. Insufficient nasal breathing can disturb quality of life and cause sleep disorders. Various studies have defined the negative impact of NSD on cardiac circulation function (1). Nasal blockage can impede the nasopulmonary reflex arch, leading to a long-term decrease in respiratory capacity (2). Reduced airflow may cause hypoxia, leading to pulmonary vasoconstriction. These changes stimulate the expression of reactive oxygen radicals (3). Obstructive sleep apnea syndrome (OSAS) is a widespread chronic disease characterised by recurrent complete or partial upper airway blockages during sleep. NSD is a risk factor for OSAS. The physiological outcomes of these situations are sympathetic activity, hypoxia, and hypercapnia. Studies of patients with OSAS have shown that hypoxia, sympathetic activity, and hypercarbia decrease choroidal thickness in these patients (4).

Spectral domain optical coherence tomography (OCT), which is a comparatively recent device for clinical in vivo

monitoring and retinal layer assessment, can provide recurrent, high-resolution, cross-sectional examinations of the retina. The scan is quantitative compared to an ophthalmoscope scan, and even minor alterations in the retina can be defined. Moreover, the choroidal thickness can be measured with a special mode.

The aim of this study was to compare choroidal thickness (CT) in patients with NSD and healthy subjects and to research how the choroid responds to hypoxia. To the best of our knowledge, this is one of the first studies evaluating choroidal thickness in patients with NSD and it can therefore be considered to contribute to the literature.

## MATERIAL and METHODS

### Subjects

This prospective study included 50 eyes (right eye) of 50 patients with NSD, and 30 eyes (right eye) of 30 healthy non-NSD subjects as a control group. All study processes were carried out in accordance with the Declaration of Helsinki. Approval for the study was granted by the Local Ethics Committee (2019/03-19) and written informed consent was obtained from all subjects before participation. The study group comprised 50 patients over the age of 18 years who were diagnosed with NSD based on radiological imaging or endoscopic examination.

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Exclusion criteria were as follows: the presence of hypercholesterolemia, hypertension, heart failure, coronary artery disease, alcohol use, smoking, metabolic syndrome, obstructive sleep apnea syndrome and pregnancy. The presence of anterior segment pathologies such as dense cataract, central corneal pathology, uveitis, high myopia or hyperopia (-4 or +4 diopters of spherical equivalent), or iris abnormalities which hindered the imaging of the posterior segment were also determined as exclusion criteria. Patients whose choroid scleral junction was not observed during the measurement and the subjects causing suspicion in marking were not included in the study.

### Examination

After demographic examination, biomicroscopic eye examination was performed on all participants and the refraction measurement was performed with a Tonoref™ III (Nidek Co. Ltd. Japan) autorefractometer. Intraocular pressure measurement was taken with an I-Care tonometer. (ICare; Finland Oy) BCVA was evaluated as 20/20 Snellen equivalent in all eyes.

A nasal obstruction-related quality of life evaluation scale was applied to the study group patients. The nose obstruction symptom evaluation (NOSE) is centred specifically on nose congestion and is used in assessment before and after therapy (5). Patients were separated as mild, moderate, or severe according to the NOSE scale. Scoring of the NOSE scale is a 0-4 point scale. (0: no problem; 1: very mild problem; 2: mild problem; 3: quite bad problem; and 4: severe problem)

### Choroidal thickness measurements

The subfoveal choroidal thickness of 80 eyes was measured with Optovue® iVue spectral-domain OCT (Optovue Inc. Fremont, CA). All subjects were assessed using EDI-OCT imaging with pupil dilation. The method of acquisition of EDI-OCT images was first reported by Spaide et al (6). The OCT apparatus is placed close to the eye to acquire a reverse image. Each section is acquired using eye tracking, and 100 B-scans are averaged to develop the signal-to-noise proportion. In the study, a horizontal image including the fovea was obtained at a distance of 9 mm. For the choroidal thickness, the distance from the exterior of the high reflective layer corresponding to the sclerocoroidal interface to the retinal pigment epithelium (RPE)/Bruch reflective complex was measured. Average choroidal thickness values were taken at 09.00 am in order not to be affected by diurnal fluctuation. All measurements were recorded by a single experienced person, blinded to the NOSE score, averaging three different measurements. Only subfoveal choroid thicknesses were examined to minimize user error and ensure standardization. Optovue OCT (Optovue Inc., Fremont, CA, USA) was used to measure the mean central macular thickness (CMT) and retinal nerve fibre layer thickness in two quadrants.

### Statistical analysis

Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS) version 23.0

for Windows software (SPSS Inc., Chicago, IL, USA). Conformity of the data to normal distribution was evaluated using the Shapiro-Wilk test. The Independent sample t-test, One-Way Anova and Post Hoc test were used to compare the mean of numerical variables between the two groups. The Chi-square test was used to compare the mean of categorical variables between the two groups. Pearson correlation analysis was used to estimate the linear relationship between continuous variables. A value of  $p < 0.05$  was considered statistically significant.

## RESULTS

The NSD group (n: 50) consisted of 29 male and 21 female patients with a mean age of 28 years. The control group (n: 30) consisted of 18 male and 12 female subjects with a mean age of 26 years. No significant difference was found between the NSD and control groups in respect of age distribution. ( $p=0.177$ ) No significant difference in BCVA, IOP, spherical and cylindrical equivalent was found between the groups ( $p=0.099$ ,  $p=0.695$ ,  $p=0.786$ ,  $p=0.527$  respectively) (Table 1).

**Table 1. Demographic and ophthalmological characteristics of the groups**

	NSD group n=50	Control group n=30	P
Age	28.2264	26.1667	0.177
Gender (M/F)	29 M/21 F	18M/12F	-
BCVA	0.92±0.15	0.94±0.10	0.099
IOP	15.64±2.14	15.50±2.41	0.695
Spherical Equivalent	-0.39±1.51	-0.40±1.50	0.786
Cylendric equivalent	-0.61± 0.81	-0.65±0.82	0.527

M: Male, F: Female, BCVA: Best corrected visual acuity, IOP: Intra ocular pressure

**Table 2. Choroidal thickness measurements at groups**

	N	Mean	Std. deviation	P Value
Control group	30	272.62µm	14.45	
Mild NSD	17	275.11µm	13.19	0.277
Moderate NSD	16	267.68µm	6.48	0.071
Severe NSD	17	258.10µm	6.51	0.001*

Std: Standart Deviation, NSD: Nasal Septum Deviation

In the study group, NSD was classified as mild in 17, moderate in 16 and severe in 17 patients. The average central choroidal thickness in mild, moderate and severe NSD according to the NOSE score was 258,10µm, 267.68µm and 275.11µm, respectively and 250.20 µm in the control group (Table 2). In terms of choroidal thickness, there was a significant difference between the

control group and the severe NOSE score group ( $p=0.001$ ). No significant difference was found between the groups with mild and moderate NOSE score. ( $p=0.894$ ,  $p=0.519$ , respectively) As a result of the correlation analysis, there was a moderate negative correlation between septum deviation (according to the NOSE score) and choroidal thickness. ( $p < 0.001$ ,  $r = -0.456$ )

The average macular thickness in the mild, moderate and severe NSD groups according to the NOSE score was  $255.76\mu\text{m}$ ,  $255.18\mu\text{m}$  and  $251.40\mu\text{m}$ , respectively. No significant difference was found between the control group and the mild, moderate and severe groups in terms of macular thickness ( $p=0.763$ ,  $p=0.826$ ,  $p=0.996$  respectively) (Table 3).

	N	Mean	Std. deviation	P Value
<b>Control group</b>	30	250.20 $\mu\text{m}$	17.07	
<b>Mild NSD</b>	17	255.76 $\mu\text{m}$	17.80	0.763
<b>Moderate NSD</b>	16	255.18 $\mu\text{m}$	20.91	0.826
<b>Severe NSD</b>	17	251.40 $\mu\text{m}$	16.51	0.996

**Std: Standart Deviation, NSD: Nasal Septum Deviation**

The average superior retinal nerve fiber layer thickness in the mild, moderate and severe NSD groups according to the NOSE score was  $104.52\mu\text{m}$ ,  $107.50\mu\text{m}$ , and  $104.65\mu\text{m}$ , respectively. The average inferior retinal nerve fiber layer thickness in the mild, moderate and severe NSD groups was  $102.564\mu\text{m}$ ,  $101.00\mu\text{m}$ , and  $101.70\mu\text{m}$ , respectively. No significant difference was found between the control group and the mild, moderate and severe groups in respect of the superior and inferior retinal nerve fiber layer thickness. ( $p=0.238$ ,  $p=0.061$ ,  $p=0.206$  (respectively superior)  $p=0.203$ ,  $p=0.396$ ,  $p=0.266$  (respectively inferior)) (Table 4).

<b>RNFL Sup</b>				
	N	Mean	Std. deviation	P Value
Control	30	102.41 $\mu\text{m}$	10.48	
Mild	17	104.52 $\mu\text{m}$	9.78	0.238
Moderate	16	106.50 $\mu\text{m}$	7.33	0.061
Severe	17	104.65 $\mu\text{m}$	9.98	0.206
<b>RNFL Inf</b>				
	N	Mean	Std. deviation	P Value
Control	30	99.41 $\mu\text{m}$	7.50	
Mild	17	102.56 $\mu\text{m}$	7.04	0.203
Modreate	16	101.00 $\mu\text{m}$	11.77	0.396
Severe	17	101.70 $\mu\text{m}$	9.68	0.266

**Std: Standart Deviation, RNFL: Retinal Nerve Fiber Layer**

## DISCUSSION

In this study, how choroidal thickness was affected by different degrees of NSD was investigated and compared with non-NSD control subjects. EDI-OCT is a recent method that allows in vivo evaluation of choroidal thickness. Recent studies have shown that EDI-OCT based choroidal thickness studies are increasing in number (7). The aim of this study was to evaluate choroidal thickness in patients with NSD using EDI-OCT.

Nasal blockage is a widespread finding on presentation in otorhinolaryngology practice. NSD is a common cause of chronic and recurrent nasal obstruction. Persistent nasal blockage causes increased superior respiratory airway resistance and this leads to hypercarbia and chronic hypoxia as a result of alveolar hypoventilation (8). NSD is identified by permanent and recurrent upper respiratory tract obstruction with simultaneous hypercapnic acidosis and hypoxia. Previous studies have suggested that pulmonary artery and systemic hypertension are seen in patients with upper airway obstruction (9). The retina is one of the most metabolically active tissues in the body with rapid oxygen ( $\text{O}_2$ ) consumption, just like the brain (10).  $\text{O}_2$  requisition of the retina is extreme and sustained  $\text{O}_2$  procurement is crucial for metabolic activity (11). This requirement is provided by both retinal and choroid circulations. The autoregulation ability of retinal circulation has been shown in previous studies (12).  $\text{O}_2$  and  $\text{CO}_2$  levels as local metabolic factors are responsible for the autoregulation of retinal circulation. Hyperoxia leads to vasoconstriction of the retinal vessels and reduces blood flow, while hypoxia and hypercapnia expand the retinal vessels and increase blood flow (13-14).

However, the effect of  $\text{CO}_2$  and  $\text{O}_2$  on choroidal blood flow has not yet been fully clarified (15). A few studies have proposed that autonomic control of choroidal blood flow is predominant compared to regional metabolic control of retinal vasculature (16). However, Riva et al showed that inhalation of 7.5%  $\text{CO}_2$  caused an increase in choroidal blood flow, and claimed that hyperoxia does not influence choroidal blood flow (17).

In the current study, subfoveal choroidal thickness was found to be significantly thinner in severe NSD. This was consistent with studies in literature related to choroid thickness in cases with hypoxia (18). It was thought that in cases with NSD, continuous airflow blockage causes a decrease in the mean saturation of oxygen and leads to hypoxia. Long-term hypoxia can lead to inflammation and oxidative stress, harming the vascular inner layer, reducing susceptibility to vasodilator agents, and stimulating the sympathetic nervous system (19). These critical conditions lead to vascular irregularity, change choroidal blood flow, and consequently decrease choroidal thickness. In a study by He et al. a notable reduction in choroidal thickness was reported in OSAS cases compared to a control group (20). The findings of He et al, imilar to those of the current study, showed that choroidal thickness of severe OSAS cases was detected to be thinner than that of control cases,

while there were similar measurements in the control subjects and mild and moderate OSAS groups. The similar CT measurements in the mild and moderate OSAS and control cases were explained by choroidal autoregulation systems. In mild and moderate OSAS cases, the balance between the parasympathetic and sympathetic systems in the choroid was assumed to be protected. In the current study, NSD may have had a restricted impact on choroidal autoregulation in mild and moderate OSAS. The choroid is the most important structure that feeds the greater part of the eye. Therefore, the effect of ischemia hypoxia status can be manifested more in the macular central fovea owing to the maximum oxygen consumption of the photoreceptors in this area. Karaca et al. reported that no correlation was found between all groups of NSD and choroidal thickness, and suggested that recently diagnosed cases had healthy parasympathetic excitations of the choroid and thus the choroid circulation may be conserved in this group of cases (21). In the current study, since all patients with new and old diagnoses were included, the long-term effects of NSD-induced hypoxia may be understood more objectively.

In the CMT evaluation in the current study, no significant difference was determined between the groups. Similarly, Bezgin et al. investigated central macular thickness in patients with nasal septum deviation and also reported no significant difference between the two groups (22).

The main limitation of our study is that the relative number of cases is low. In addition, since marking of choroid in measurements of choroid thickness with EDI-OCT is done manually, it can be considered a limitation of the study. Another limitation is that choroid measurements are not made in 5 quadrants. In most of the previous studies, the measurements in 5 quadrants were compatible with each other, so we took measurements from one quadrant in order to minimize user error and standardization.

## CONCLUSION

In conclusion, the results of this study suggest that nasal obstruction in patients with severe NSD may cause marked hypoxia and sympathetic activation, leading to impaired choroidal blood flow and consequent choroid thinning.

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