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Original Research

Effect of nicotine in fundus of type II diabetics

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ABSTRACT:

Background: Diabetes is a long-term medical condition marked by high blood glucose levels and abnormal protein and fat metabolism. The present study was conducted to study the effect of nicotine in fundus of type II diabetics. **Materials & Methods:** 84 patients visiting for eye examination of both genders were divided into 2 groups. Group I had cases and group II had control. Snellen's alphabet chart was used to record each patient's best corrected visual acuity (BCVA). Anterior segment examination utilizing slit lamp biomicroscopy was performed after torch light examination. **Results:** Fundus changes were none in 21 in group I and 3o in group II, mild in 10 in group I and 9 in group II, moderate in 11 in group I and 3 in group II. The difference was significant (P< 0.05). The mean LogMar value in group I was 1.15 and in group II was 0.89. The difference was significant (P< 0.05). **Conclusion:** Retinal morphology showed notable temporal alterations in response to exposure to nicotine, diabetes, and the combined effects of nicotine and diabetes.

Key words: Diabetes, fundus, Nicotine

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INTRODUCTION

Diabetes is a long-term medical condition marked by high blood glucose levels and abnormal protein and fat metabolism. Because the pancreas is not producing enough insulin or the cells are not able to use the insulin that is being produced efficiently, blood glucose levels rise because the glucose cannot be digested in the cells.[1] Three main forms of diabetes exist: Type 1 diabetes, in which the pancreas fails to produce insulin; type 2 diabetes, in which body cells become resistant to the action of insulin produced; and type 3 diabetes, which develops during pregnancy, can result in complications both during and after childbirth and raise the risk of type 2 diabetes.[2]

The most common cause of new cases of blindness in adults between the ages of 20 and 74 is diabetic retinopathy. Nearly all individuals with type 1 diabetes and more than 60% of people with type 2 diabetes get retinopathy over the first 20 years of their condition.[3] The disease's characteristic retinal damage is caused by non-perfusion and retinal vascular leakage. Retinal leukocyte stasis is temporally and geographically correlated with diabetic retinal vascular leakage, capillary non-perfusion, and endothelial cell destruction.[4]

Within days after acquiring diabetes, retinal leukostasis rises and is correlated with increased expression of CD18 and retinal intercellular adhesion molecule-1 (ICAM-1). When compared to non-diabetic controls and diabetic people without retinopathy, retinal blood flow is markedly higher in diabetic retinopathy. There is evidence connecting cigarette smoking to a larger retinal venular caliber.[5]The present study was conducted to study the effect of nicotine in fundus of diabetic patients.

MATERIALS & METHODS

The present study comprised of 84 patients visiting for eye examination of both genders. All gave their written consent to participate in the study.

Data such as name, age, gender etc. was recorded. Two groups were made. Group I had cases and group II had control. Snellen's alphabet chart was used to record each patient's best corrected visual acuity (BCVA). Anterior segment examination utilizing slit lamp biomicroscopy was performed after torch light examination. The patient's pupillary response and

anterior segment inspection were evaluated using slit lamp microscopy, and then tropicamide (1%) and phenylephrine (2.5%) eye drops were used to dilate their pupils. Further management was carried out in accordance with the grade and type of diabetic retinopathy, as determined by the ETDRS categorization of the post-dilatation type and grade.Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Assessment of fundus changes in both groups

Fundus changes	Group I (42)	Group I (42)	P value
None	21	30	0.05
Mild	10	9	
Moderate	11	3	
Severe	0	0	

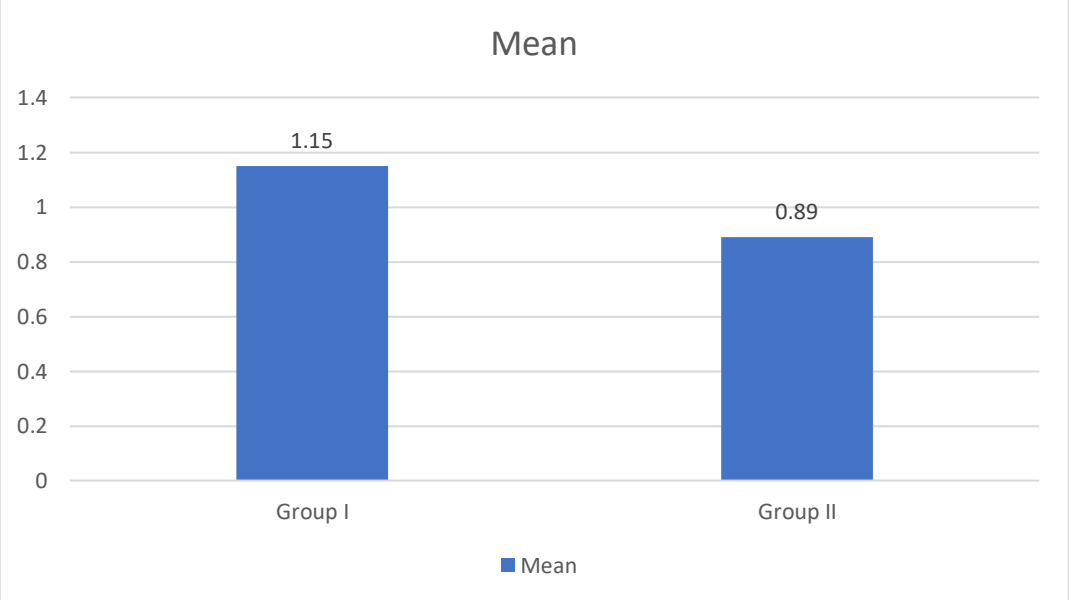
Table I shows that fundus changes were none in 21 in group I and 3o in group II, mild in 10 in group I and 9 in group II, moderate in 11 in group I and 3 in group II. The difference was significant (P< 0.05).

Table II Assessment of LogMar in both groups

Groups	Mean	P value
Group I	1.15	0.05
Group II	0.89	

Table II shows that mean LogMar value in group I was 1.15 and in group II was 0.89. The difference was significant (P< 0.05).

Graph I Assessment of LogMar in both groups



DISCUSSION

Increased intraocular VEGF levels brought on by retinal ischemia from capillary blockage of retinal arteries are the cause of proliferative diabetic retinopathy.[6] Neovascularization in the optic disc or elsewhere in the retina (neovascularization elsewhere) can both lead to increased proliferation into the vitreous. During fluorescein angiography, these recently created arteries leak, which may result in vitreous hemorrhage and, in the end, tractional retinal detachment.[7] The neurosensory retina and the retinal pigment epithelium separate as a result of tractional retinal detachment.[8,9]The present study was conducted to study the effect of nicotine in fundus of diabetic patients.

We found that fundus changes were none in 21 in group I and 3o in group II, mild in 10 in group I and 9 in group II, moderate in 11 in group I and 3 in group II. Omae et al.'s[10] study looked at how smoking cigarettes affected patients' retinal circulation over time who had type 2 diabetes. A total of 74 patients with type 2 diabetes mellitus and minimal diabetic retinopathy (DR)—that is, nil or mild non-proliferative DR—were assessed. Based on their smoking history, these individuals with type 2 diabetes were split into three groups: current smokers (n = 19), previous smokers (n = 20), and never smokers (n = 35). Using laser Doppler velocimetry, the retinal circulation parameters were assessed and compared between the groups. Retinal blood flow

(RBF; 8.9 ± 2.9 vs. 11.6 ± 3.1 μL/min, P = 0.009) and blood velocity (V; 29.6 ± 6.8 vs. 37.8 ± 9.0 mm/s, P = 0.003) were significantly lower, although there was no difference in vessel diameter (D; 112.0 ± 11.9 vs. 113.7± 8.6 μm, P = 0.57) in the current smokers compared with those who never smoked).When comparing the RBF, blood V, and vascular D of former smokers to non-smokers and current smokers, no differences were found. A multiple regression analysis revealed a substantial and independent correlation between lower RBF and current smoking, as well as a negative correlation between the RBF and creatinine level. The findings suggested that continuous smoking may be linked to decreased RBF, most likely through lower blood V in the retinal arterioles in early-phase DR. Patients with type 2 diabetes who smoke regularly may have decreased blood V and RBF in the retinal arterioles.

We observed that mean LogMar value in group I was 1.15 and in group II was 0.89. Using a well-established mouse model, Boretsky et al[11] studied the impact of nicotine on retinal changes in early stage diabetes. The measurements of retinal thickness in the nasal, temporal, superior, and inferior quadrants were made using 20° x 20° volume scans of SD-OCT that were centered on the optic disc. To further investigate changes in each treatment group, separate retinal layers were segmented on a subset of SD-OCT cross-sections. Using confocal morphometric imaging, the neuronal survival inside the ganglion cell layer (GCL) was evaluated. Throughout the trial, the control group did not undergo any notable changes. Total retinal thickness (TRT) decreased by 9.4 μm on average in the nicotine treatment group, with segmentation revealing that the outer nuclear layer (ONL) accounted for the majority of the loss.While segmentation analysis of the DR group revealed considerable thinning inside the ONL, the diabetic group showed a trend toward decreased TRT. When diabetes and nicotine were combined, there was a substantial 8.9 μm increase in TRT (P < 0.05) and a corresponding drop in GCL neuron count.

The limitation of the study is small sample size.

CONCLUSION

Authors found that retinal morphology showed notable temporal alterations in response to exposure to nicotine, diabetes, and the combined effects of nicotine and diabetes.

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