Letter to the Editor

Taiwan J Ophthalmol 2023;13: 562-563

Response to comment on: "Ganglion cell complex and retinal nerve fiber layer thickness in gestational diabetes mellitus"

Dear Editor,

We thank Thiago G S Martins for providing valuable feedback on our article "Ganglion cell complex and retinal nerve fiber layer thickness in gestational diabetes mellitus," published in *Taiwan J Ophthalmol*. We appreciate the time and effort he has put into reading our manuscript and highlighting major additional points related to the topic.

Since diabetic retinopathy was initially believed to be a microvascular disorder, increasing research reveals neurodegeneration as an early stage in the disease's pathophysiology. The major contributors to diabetic retinal neurodegeneration are glial activation and neural apoptosis. The diabetic donor retinas with no identifiable vascular abnormalities on ocular examination were found to have these neurodegenerative alterations.[1]

Retinal ganglion and amacrine cells are the first neuroretina cells exhibiting signs of diabetes-induced apoptosis. The retina's ganglion, amacrine, and Müller cells undergo apoptosis as a consequence of increased glutamate concentration, which causes neurodegeneration and reduction of the layer of ganglion cells and nerve fibers. Optic coherence tomography might detect these morphological changes.[2] In our study, pregnant females with gestational diabetes mellitus had thinner peripapillary and macular retinal nerve fiber and ganglion cell layers than healthy pregnant females.[3]

In line with the author, we believe that studying neuroprotective therapies may be beneficial in managing diabetic retinopathy. This might prevent the need for more invasive treatment as diabetic retinopathy progresses. Administration of somatostatin and brimonidine tartrate eye drops topically improves blood flow in the retina because of local vasodilation, which might halt diabetic retinopathy progression. Somatostatin levels are often lower in diabetic individuals with damaged ganglion cells, and this may have an effect on preventing neurodegeneration by lowering glutamate concentration. In addition, it works by reducing the synthesis of vascular endothelial growth factor and preventing neovascularization.[4]

The pigment epithelium-derived factor, which protects neurons from glutamate-mediated neural damage and is reduced in diabetic retinopathy, is another strong neuroprotective and antiangiogenic substance.[5] More extensive research is still needed, but it is possible that other neuroprotective substances like neuroprotectin D1, insulin, brain-derived neurotrophic factor, ciliary neurotrophic factor, nerve growth factor, glial cell line-derived neurotrophic factor, and adrenomedullin are also involved in the neurodegenerative process that causes diabetic retinopathy.

Studies demonstrating the ganglion cell and nerve fiber layer thickness before the appearance of vascular changes in diabetic individuals might demonstrate neurodegeneration. Improvements in this research and the use of neuroprotective medications might be helpful in diabetic retinopathy treatment.

Data availability statement

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

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Nil.

Conflicts of interest

The authors declare that there are no conflicts of interests of this paper.

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