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Association between interferon beta levels and neuro-retinal degeneration in primary angle closure glaucoma

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Abstract

Introduction: Glaucoma is one of the major causes of irreversible blindness worldwide. The vision loss here is related to elevated intraocular pressure (IOP) mediated degeneration of the optic nerve. Obstruction to aqueous humour outflow is the main reason for increased IOP and the two major types of glaucoma based on this include primary open angle glaucoma (POAG) and primary angle closure glaucoma (PACG). Current strategies are directed against reducing IOP, but progression of neurodegeneration continues despite managing IOP in some patients. This suggests the existence of and the need to explore additional mechanisms that may contribute to disease progression. Interferon beta (IFNβ), an immunomodulator has been used quite effectively in the management of neurodegenerative conditions, hence its role in glaucoma is worthy of investigation.

Objective: To study the relationship between the endogenous expression of IFNβ in the ocular tissues with reference to the severity of PACG.

Methods: Iris, trabecular meshwork (TM) and aqueous humour (AH) were obtained surgically during trabeculectomy from PACG (n=34) patients operated as part of standard of care. Severity of glaucoma was based on Visual Field Index on Humphrey visual fields 24-2 program. Further categories based on type of medication use were also done. RNA extracted from iris and TM was used to determine the expression of IFNβ by quantitative-PCR and IFNβ in AH was measured using ELISA. Institutional ethics committee approval was obtained for the study.

Results: Normalized IFNβ gene expression in iris was lower in severe (0.0557±0.01) compared to mild/moderate (0.1197±0.04) cases. Likewise, its expression in TM was reduced in severe (0.0745±0.06) compared to mild/moderate (0.1945±0.11) cases. IFNβ levels in aqueous humour was lower in severe (149±36 pg/µg of total protein) compared to mild/moderate (439±13 pg/µg of total protein) cases. Anti-glaucoma medications with or without prostaglandin analogs did not alter the trend of lower expression of IFNβ in severe compared to mild/moderate cases in iris and TM.

Conclusion: Decreased levels of IFNβ observed in severe cases suggest that it can predispose to the neurodegeneration in glaucoma. However, its causal role and relevance in theragnostics are yet to be explored.

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