

Perspectives on Genetics of Congenital Glaucoma

HarishP*

Institutional affiliations

Postgraduate Institute of Ophthalmology, India

Corresponding author

Harish Pazar, Eye Hospital & Postgraduate Institute of Ophthalmology, India E-mail: harishpazar@yahoo.co.in

Received Date: November 29 2021

Accepted Date: November 30 2021

Published Date: December 27 2021

Editorial

As everyone is aware, eye disease is the leading cause of irreversible visual impairment worldwide. Although the majority of eye disease patients are elderly, inherent eye disease (CG) and childhood glaucomas are important causes of visual incapacity. Inherited eye disease is a single, non-syndromic eye disease that occurs within the first three years of life and is one of the leading causes of childhood visual impairment. Early-onset glaucomas may develop as a result of biological process abnormalities, such as glaucomas associated with aniridia or as part of Axenfeld-Rieger syndrome and/or Peters' anomaly. Inherited glaucomas have strong genetic bases, and disease-causing mutations have been discovered in many genes since Sarfarazi's discovery in 1995 [1]. Mutations in three genes (CYP1B1, LTBP2, and TEK) are suspected in patients with inherent eye disease.

Clinically, inherent eye disease is characterised by high pressure (IOP), epiphora, tissue layer swelling, photophobia, cramp and ocular enlargement. The illness is bilateral in around seventy fifth of patients. CG incidence varies considerably among countries: it's calculable to occur in regarding 1/10000 births in Europe, in 1/2500 in Asian nation and 1/1250 within the Bohemian population in European nation (Online monastic Inheritance in in Man, OMIM 231300). From a pathophysiological purpose of read, a rise in IOP, untreated either with surgery or pharmacologic medical care, ends up in ocular enlargement (buphthalmos) and quickly progressive bloodletting of the second cranial nerve with severe and irreversible harm of the sight view. Early recognition and medical care will considerably improve the child's visual operate. Onset of associate degree aggressive type of eye disease happens between birth and three years archaic. The illness has higher prevalence in males (2:1; M/F). Most cases of CG square measure unpredictable and recessive inheritance of CG is common, with virtually complete penetrance in populations with high family relationship rate. Reduced penetrance (40% in some populations) and numerous composition forms recommend a inheritable inheritance pattern or multi factorial aetiologies. {in a|during a|in associate degree exceedingly|in a very} paper printed in Ophthalmic biological science in 2011 we tend to confirmed the main role of CYP1B1 factor in inherent eye disease and that we urged an autosomic recessive role of MYOC/TIGR during a digenic inheritance model [3].

Treatment of diseases with factor medical care is advancing quickly and is that the way forward for the medical care of eye disease. Genes provide varied blessings over standard medicine. The factor medical care use each microorganism and nonviral vectors. the attention is a perfect organ for factor medical care. it's simply accessible associate degreeed it's an immune-privileged website. Currently, there square measure clinical trials for diseases poignant much each tissue of the attention, largely attempting to revive

Vision in patients stricken by Leber inherent visual defect. the quantity of eye trials compared with those for general diseases is kind of low (1.8%). One space for eye factor medical care is eye disease, wherever a long-run factor drug would eliminate daily applications and compliance problems. the longer term of eye disease medical care is that the chance for treating the trabeculate material to lower pressure (IOP) and therefore the retinal neural structure cells (RGC) to guard them from neurodegeneration [4].

References

1. Sarfarazi M, Akarsu AN, Hossain A, Turacli ME, Aktan SG, et al. (1995) Assignment of a locus (GLC3A) for primary congenital glaucoma (Buphthalmos) to 2p21 and evidence for genetic heterogeneity. *Genomics* 30(2): 171-177.
2. Lewis CJ, Hedberg-Buenz A, De Luca AP, Stone EM, Alward WLM, et al. (2017) Primary congenital and developmental glaucomas. *Hum Mol Genet* 26(R1): R28-R36.
3. Giuffre I (2011) Molecular analysis of patients affected by congenital glaucoma. *Ophthalmic genetics* 2011-08-04 e pub ahead of print.
4. Borrás T (2017) The pathway from genes to gene therapy in glaucoma: a review of possibilities for using genes as glaucoma drugs. *Asia Pac J Ophthalmol* 2017 6(1): 80-93.