

Homocysteine in ocular diseases

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Abstract

Homocysteine (Hcy) is a derived sulfur-containing and non-proteinogenic amino acid. The metabolism of Hcy occurs either through the remethylation to methionine or transsulfuration to cysteine. Studies have identified hyperhomocysteinemia (HHcy) as one of the possible risk factors for a multitude of diseases including vascular, neurodegenerative and ocular diseases. Association of HHcy with eye diseases such as retinopathy, pseudoexfoliative glaucoma maculopathy, cataract, optic atrophy and retinal vessel atherosclerosis is established. The molecular mechanism underlying these ocular diseases has been reported as impaired vascular endothelial function, apoptosis of retinal ganglion cells, extracellular matrix alterations, decreased lysyl oxidase activity and oxidative stress. The formed homocysteine-thiolactone in HHcy has stronger cytotoxicity and pro-inflammatory properties which can induce lens opacification and optic nerve damage. The metabolism of Hcy requires enzymes with vitamins such as folic acid, vitamins B12 and B6. Despite the mixed conclusion of various studies regarding the level of these vitamins in elder people, studies recommended the treatment with folate and B12 to reduce Hcy levels in subjects with or without any defect in the enzymes involved in its metabolism. The levels of Hcy, folate, B6 as well as B12 should be measured early in patients with visual impairment that would aid to screen patients for life-threatening disorders related with HHcy. Elder patients may supplement with these vitamins in order to attenuate the ocular damages. This article discusses the association of Hcy in ocular diseases and the possible mechanism in the pathogenesis.

Keywords: Cataract; Diabetic retinopathy; Folate; Glaucoma; Hyperhomocysteinemia; Visual impairment; Vitamin B12.

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