

## MRI Reveals Comorbid Cerebrovascular Lesions in Glaucoma

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### Editorial

Everyone above the age of 40 years is at risk for glaucoma, particularly in those with diabetes, a familial history of glaucoma and a history of eye injury. It is one of the dominating causes of blindness in worldwide. Owing to its asymptomatic nature, a significant, irreversible and progressive centripetal visual field loss subsequent to the damage of the optic nerve head and the loss of retinal nerve fiber layer are commonly found in newly diagnosed individuals. Today, the main modifiable treatment option to delay the progression of glaucoma and preserve the visual field remains relying on the reduction of intraocular pressure. However, rigorous control of intraocular pressure in many glaucoma patients failed to prevent the deterioration of glaucomatous changes, and a minuscule proportion of normal tension glaucoma subjects had developed an onset of visual field loss [1]. Therefore, elevated intraocular pressure is now considered as an important, but not the sole risk factor causing retinal ganglion cells death, meaning that certain risk factors must be involved in the pathogenesis of glaucoma.

Over the last decade, researchers have been making great progress in identifying new novel approaches to uncover the pathophysiology of glaucoma in the genetic, molecular and tissue level. Recent evidence-based theory confirmed vascular dysregulation is another primary risk factor for the pathogenesis of glaucoma [2]. The vascular dysregulation theory defines the glaucomatous condition is the consequence of a vessel that cannot adapt to the demands of the respective tissue. It is the result of a dysfunction of the autonomic nervous system and endothelial cells, leading to an impairment of vascular tone autoregulation and causing the subsequent ischaemic blood flow, unstable supply of oxygen and oxidative stress to the optic nerve [3]. Some studies have also pointed out that glaucomatous disease manifestation, similar to the endothelial dysfunction, could be a systemic disease process in the body [4]. As a result, vascular dysregulation as expressed by systemic endothelial dysfunction might be one of the risk markers for glaucoma.

Vascular dysfunction in glaucoma is not necessarily limited in the eyes, as the microcirculation at the ocular and cerebral level also shared a large number of anatomical and physiological similarities. A reduction in orbital blood flow will cause a fluctuation of oxygen supply and oxidative stress, leading to the optic nerve atrophy [5]. Similarly, in the brain, a reduction in cerebral blood supply will lead to cerebral infarction. Likewise, the degeneration of the retinal ganglion cells may also elicit a spread of glaucomatous injury along vision pathways, and the damage may extend from the eye to the visual cortex, leading to the similar comorbid cerebrovascular conditions in glaucoma patients [6]. Thus, it was suggested that vascular insufficiency at the microcirculation feeding the central visual pathways and the cerebral visual cortex might associated with

glaucoma and its comorbid cerebrovascular conditions at the optic nerves, lateral geniculate bodies and visual cortex.

The assessment of the visual pathways using clinical MRI scanner not only can visualize the anatomical information such as lateral geniculate nucleus, but it also permits the assessment of the structural integrity of the more distal area of the visual system, and the assessment of the shrinkage of visual structures along the geniculocortical pathway in the brain. Recent studies have shown the degeneration of retinal ganglion cell can extend to the primary visual cortex, leading to the subsequent loss of cortical volume and function [7,8]. In addition, evidence of microvascular disease in the brain, as demonstrated by the presence of white matter hyperintensities on magnetic resonance imaging scans, has also been found in the glaucoma patients [9,10]. These observations therefore supports the knowledge that neuronal damage in glaucoma involves central stations of the visual pathway.

All in all, although the clinical presentation of glaucoma is well defined, the pathogenesis of this heterogeneous group of disease, as well as the association of its comorbid cerebrovascular conditions, particularly at the optic nerves, lateral geniculate bodies and the visual cortex, among patients with glaucoma were not fully understood. Being a valuable research and clinical tool to detect and stage the central glaucomatous damage, the application of MRI can therefore provide new insight to extend the knowledge about this disease.

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