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EDITORIAL (BY INVITATION)

An innovative approach to the management of IOP in glaucoma

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Glaucoma is a group progressive optic neuropathies characterized by degeneration of retinal ganglion cells and axonal loss detected both at the retinal nerve fiber layer and optic nerve head. Elevated intraocular pressure (IOP) causes axonal damage, either by direct nerve compression or diminution of blood flow. Despite the possibility that other risk factors (genetic, anatomical, neurological, vascular, inflammatory etc.) may play a role, IOP lowering strategies have remained for more than a century the main target of medical and surgical treatment of glaucoma [1]. This is true also in the paradoxical case of low-tension glaucoma. Noteworthy, alternative modalities of treatment such as neuroprotection were disappointing as in the case of the failure of the monumental Allergan RCT on memantine [2].

The publication in this issue of the Journal entitled "VEGF-A-induced changes in distal outflow tract structure and function" by Loewen et al. [3] may introduce an innovative approach to the management of IOP in glaucoma. The authors performed an elegant study on porcine eyes with and without trabecular meshwork. They reconstructed the 3D vasculature of the outflow pathways using SD-OCT and they continuously monitored IOP up to 72 h following VEGF-A treatment. Thanks to this rigorous experimental setting, the authors were able to show that VEGF-A would increase outflow facility of the distal tract, independently from trabecular meshwork. Noteworthy, this was associated with a significant increase of the vessel volume of the distal outflow tract.

If the preclinical results of this paper will be confirmed by clinical studies, a new class of treatment might be added in glaucoma medical therapy. Whatever the case, the study has the merit of stimulating original research on the pathogenesis and medical treatment of glaucoma, a topic which suffers from surprising tiredness and monotony since the unsurpassed, good old times of the introduction of timolol and prostaglandin analogues in clinical practice.

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