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Aqueous outflow - A continuum from trabecular meshwork to episcleral veins

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ABSTRACT

In glaucoma, lowered intraocular pressure (IOP) confers neuroprotection. Elevated IOP characterizes glaucoma and arises from impaired aqueous humor (AH) outflow. Increased resistance in the trabecular meshwork (TM), a filter-like structure essential to regulate AH outflow, may result in the impaired outflow. Flow through the 360° circumference of TM structures may be non-uniform, divided into high and low flow regions, termed as segmental. After flowing through the TM, AH enters Schlemm's canal (SC), which expresses both blood and lymphatic markers; AH then passes into collector channel entrances (CCE) along the SC external well. From the CCE, AH enters a deep scleral plexus (DSP) of vessels that typically run parallel to SC. From the DSP, intrascleral collector vessels run radially to the scleral surface to connect with AH containing vessels called aqueous veins to discharge AH to blood-containing episcleral veins. However, the molecular mechanisms that maintain homeostatic properties of endothelial cells along the pathways are not well understood. How these molecular events change during aging and in glaucoma pathology remain unresolved. In this review, we propose mechanistic possibilities to explain the continuum of AH outflow control, which originates at the TM and extends through collector channels to the episcleral veins.

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1. Introduction

1.1. Glaucoma and aqueous humor outflow: Overview

Glaucoma is a group of diseases leading to irreversible blindness. The diseases are characterized by a pressure sensitive optic neuropathy (Coleman, 1999) with progressive retinal ganglion cell (RGC) death and visual field loss (Coleman, 2003). Worldwide, the resultant silent, painless progressive loss of sight affects over 60.5 million people. This number of people affected continues to increase thereby rendering glaucoma a sight threatening public health problem of broad significance (Quigley and Broman, 2006).

The disease occurs predominantly later in life and typically progresses; however, dysgenesis of the outflow system at times occurs early in life. Manifestations of dysgenesis are present in congenital and juvenile forms of glaucoma but are often not as readily apparent in other glaucoma conditions (Grover et al., 2015). Primary open angle glaucoma (POAG) is the most common form of glaucoma and frequently occurs with elevated intraocular pressure (IOP) (Anderson, 1989; Morrison and Acott, 2003).

Lowering IOP remains a proven intervention, even in normal tension glaucoma (NTG) where IOP remains in the normal range

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