

For the Primer, visit [doi:10.1038/nrdp.2016.67](https://doi.org/10.1038/nrdp.2016.67)

→ Primary open-angle glaucoma (POAG) is an optic neuropathy that is characterized by the progressive degeneration of the optic nerve, which leads to visual impairment.

EPIDEMIOLOGY

Between 35 and 58 million people were estimated to have POAG worldwide in 2015. Prevalence can vary according to ethnicity; black individuals have the highest prevalence of POAG, with >5% of individuals >60 years of age compared with 2.7% among Hispanic or Latino individuals, ~2% among Asian populations and ~1.5% among white individuals.

! Age is an important risk factor for the development of POAG; prevalence is expected to increase to 53–65.5 million affected individuals by 2020, owing to the ageing world population

Rx MANAGEMENT

The aim of POAG treatment is to preserve vision, which is currently achieved by reducing IOP to a level that is unlikely to cause further damage to the optic nerve. Topical prostaglandin analogues are the first-line treatments, but α -adrenergic agonists, carbonic anhydrase inhibitors and β -blockers are also available for POAG. These medications reduce the flow of aqueous humour into the eye or increase the outflow of aqueous humour, or both. Laser trabeculoplasty and surgical trabeculectomy are other standard treatments for POAG.



MECHANISMS

Increased intraocular pressure (IOP) is the result of blockage of the trabecular meshwork that drains the aqueous humour from the anterior chamber of the eye. Increased IOP is the leading risk factor for POAG.

ANTERIOR CHAMBER
TRABECULAR MESHWORK
VEOSCLERAL OUTFLOW

OUTLOOK

New treatment options are needed for POAG as many patients will experience progressive visual loss, despite the use of topical or surgical therapies. New

generations of laser treatments and minimally invasive glaucoma surgeries have been developed but require further investigation. Promising data from animal

The optic nerve head (ONH) is the point at which retinal ganglion cell (RGC) axons leave the eye and converge to form the optic nerve

HEALTHY

POAG is an optic neuropathy in which a range of factors, including increased IOP, contribute to optic nerve damage and result in loss of vision

GLAUCOMA

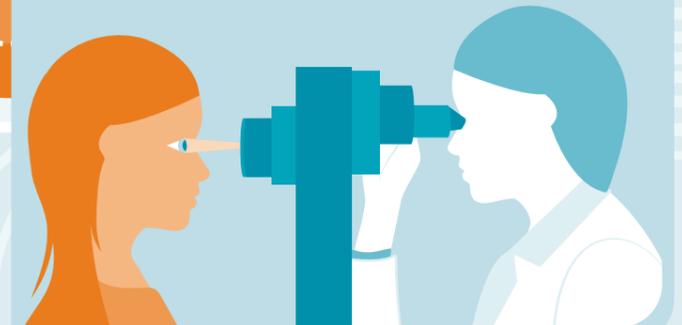
The ONH is the primary site of injury and 'cupping' of the structure is the characteristic feature of glaucoma. Aside from this mechanical deformation, other mechanisms can contribute to the degeneration of RGC axons (the retinal nerve fibre layer) and somas, including ischaemia, reactive gliosis and oxidative stress.

models indicate that promoting regeneration of the optic nerve might be beneficial in patients with POAG, but have yet to be examined in human trials.

DIAGNOSIS

POAG is generally diagnosed during routine eye examination, at which time most patients are asymptomatic. Diagnosis is based on changes to the structure of the ONH and loss of retinal nerve fibres, detected using slit-lamp biomicroscopy and/or digital imaging including optical coherence tomography. Although structural damage can occur up to 8 years before the loss of visual sensitivity, visual field assessment using perimetry should be used to diagnose and, in particular, to monitor the progression of glaucoma.

Almost 50% of cases of glaucoma are undiagnosed in the United States, Australia and Singapore — this figure increases to >95% in less-developed countries



QUALITY OF LIFE

Glaucoma-associated vision loss impairs quality of life by affecting the ability of patients to perform daily activities, such as reading, walking and driving. The risk of motor vehicle accidents is increased in patients with POAG, which leads many patients to cease driving. Balance and walking ability is also impaired, meaning patients with POAG have a higher risk of falls than healthy individuals.

Patients with a faster decline of vision loss report more severe reductions in quality of life than those with a slower decline of visual function