

Contents

<u>Definition</u>	2
<u>Prevalence and incidence</u>	3
<u>Signs and symptoms</u>	3
<u>Causes/risk factors</u>	4
<u>Pathophysiology (mechanism of disease)</u>	5
<u>Prognosis and complications</u>	6
<u>Diagnosis/detection</u>	6
<u>Pharmacological treatment</u>	7
<u>Non-pharmacological treatment</u>	9
<u>Patient support</u>	9
<u>Further resources</u>	9
<u>External websites</u>	9
<u>References</u>	10

Definition

Access **'What is glaucoma?'** on the Glaucoma UK website which provides an easy to read overview of glaucoma.

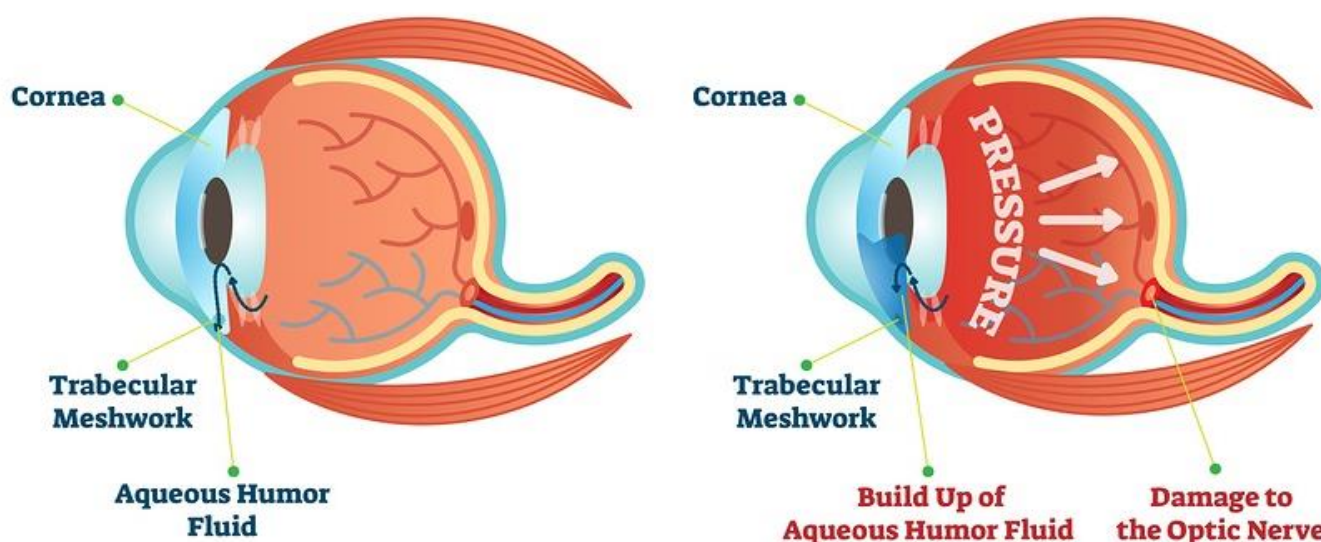
The NICE Clinical Knowledge Summary (CKS) **Glaucoma** defines glaucoma as 'a group of eye diseases that cause progressive optic neuropathy, and in which intraocular pressure (IOP) is a key modifiable factor. Glaucoma is commonly associated with raised IOP and is characterised by:

- visual field defects.
- changes to the optic nerve head such as pathological cupping or, as a late sign, pallor of the optic disc.

Ocular hypertension is where there is consistently or recurrently elevated IOP (greater than 21 mmHg) but with no signs of glaucoma.¹

The diagram below shows a normal eye (left) and an eye with glaucoma (right). The fluid inside the eye (aqueous humour) drains out through the pupil, around the iris and out of the eye through the trabecular meshwork (drainage channels).

When this process is disrupted the pressure inside the eye can cause damage to the optic nerve as described above. The optic disc is the area where the optic nerve enters the back of the eye. Pathological cupping and pallor of the optic disc describe changes that can be seen when the back of the eye is examined in a person with glaucoma. It should be noted that some people develop normotensive glaucoma – glaucoma with an IOP below 21 mmHg.²



As per NICE CKS **Glaucoma** there are several ways to classify glaucoma:

- *'age of onset: congenital, infantile, juvenile, or adult.*
- *cause: primary (no known cause), or secondary with a known underlying cause*
- *rate of onset: acute, subacute, or chronic*
- *the anterior chamber angle between the iris and cornea: being either open or closed.'*

Visit NICE CKS **Glaucoma, Classification** to learn more about the different types of open and closed angle glaucoma. A more detailed description of the anatomy of the eye and open and closed angle glaucoma can be found under **Anatomy and physiology**.

[Return to contents](#)

Prevalence and incidence

Ocular hypertension affects three to five percent of people in the UK over 40.³

Primary open-angle glaucoma (POAG) is the most common type of glaucoma.⁴ It is estimated to affect around 500,000 people in the UK and about 2 percent of people older than 40 years. Prevalence increases with age and affects about 8 percent of people over 80 years.

Primary angle closure glaucoma (PACG) affects around 130,000 people in the UK and about 0.4 percent of people over the age of 40. Again, prevalence increases with age and PACG affects around 0.94 percent of those over 70. PACG is three times more common in women than men.³

[Return to contents](#)

Signs and symptoms

Open-angle glaucoma and closed-angle glaucoma present differently.

Open-angle glaucoma

People with open-angle glaucoma are normally asymptomatic initially and unaware of early peripheral vision loss. If uncontrolled, open-angle glaucoma can cause more significant visual field loss and reduced central vision.

Closed-angle glaucoma

Acute angle closure is an ocular emergency and symptoms develop suddenly. As per the NICE CKS **Glaucoma** typical signs and symptoms of acute angle closure include:

- eye pain, often severe, and associated with headache, nausea, and vomiting caused by the pain
- a red eye
- impaired visual acuity and lights are seen surrounded by halos – caused by a hazy oedematous cornea
- semi-dilated and fixed pupil – classically the pupil becomes fixed in a vertically oval shape
- a tender, hard eye – when measured by an optometrist or ophthalmologist, the intraocular pressure (IOP) is typically very high.⁵

Information about the symptoms of sub-acute angle glaucoma and chronic PACG can be found in the NICE CKS **Glaucoma – How is primary angle closure glaucoma diagnosed?**

[Return to contents](#)

Causes/risk factors

Open-angle glaucoma

It is unclear why POAG develops; it is thought to be related to changes in the trabecular meshwork.⁶

Risk factors for POAG include:

- raised IOP
- increasing age
- family history and genetic factors
- black ethnicity
- corticosteroid use
- myopia (near-sightedness)
- **type 2 diabetes mellitus**
- **hypertension** and cardiovascular disease.⁷

Secondary causes of open angle glaucoma include: deposition of materials into the trabecular meshwork such as pigment, organelles, inflammatory cells and proteins, the formation of new blood vessels which block flow through the trabecular meshwork and corticosteroids increasing the resistance to outflow of the aqueous humour through the trabecular meshwork.⁶

Closed-angle glaucoma

PACG occurs in people who have an anatomical predisposition meaning that their iris pushes onto the trabecular meshwork.⁶

Risk factors for PACG include:

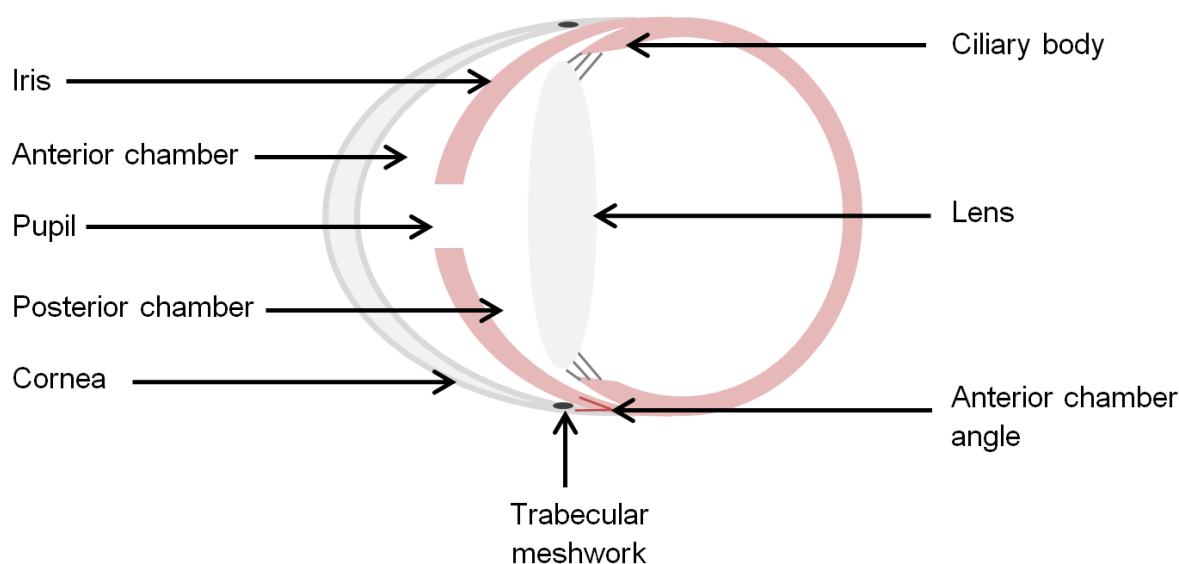
- increasing age
- female gender
- Asian ethnicity
- hyperopia (farsightedness)
- family history and genetic factors.⁷

Secondary causes of angle closure glaucoma can occur as a result of various rare eye conditions.⁶

More details about the causes and risk factors for glaucoma can be found on the NICE CKS **Glaucoma**, **Causes** and **Risk factors** pages.

[Return to contents](#)

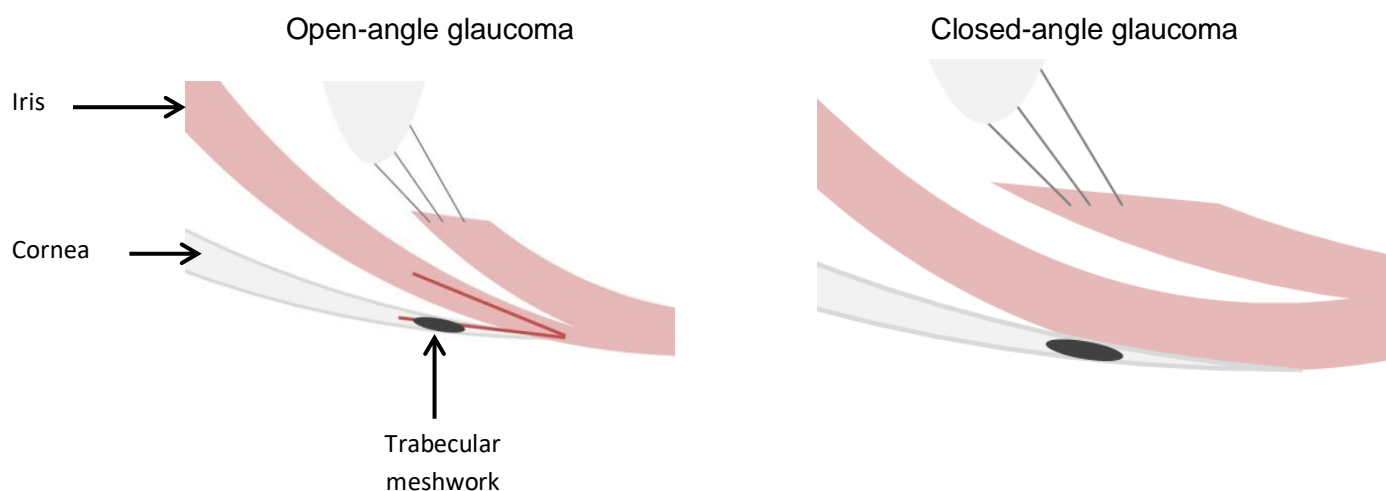
Pathophysiology (mechanism of disease)



In a healthy eye, aqueous humour (a clear fluid) is produced by the ciliary body behind the iris and secreted into the posterior chamber between the iris and lens. Secretion of aqueous humour is increased by stimulation of beta-2 receptors and decreased by stimulation of alpha-2 receptors of the sympathetic nervous system which are located on cells of the ciliary body. Carbonic anhydrase also plays a key role in its production.

Aqueous humour passes out of the pupil and into the anterior chamber, between the cornea and iris. It then passes out of the eye primarily via the trabecular meshwork. A small percentage drains through the venous circulation of the iris, ciliary body and other structures within the eye. This is known as uveoscleral outflow.²

If the anterior chamber angle (between the iris and cornea) is normal then the trabecular meshwork is exposed. If this angle is closed or partially closed then the trabecular meshwork can become covered by the iris.



When the aqueous humour is unable to drain, either due to a closed anterior chamber angle obstructing the trabecular meshwork, or the anterior chamber angle being open but the trabecular meshwork being unable to drain due to another reason, this can lead to glaucoma.

IOP keeps the eye spherical and is maintained by a balance between aqueous humour production and outflow. Raised IOP is the main risk factor for developing glaucoma as the raised pressure may damage the nerve fibres of the optic nerve. A pressure between 11 and 21 mmHg is considered normal however, some people develop glaucoma at a pressure below 21 mmHg and some people have pressures well above this level without showing signs of glaucoma.²

[Return to contents](#)

Prognosis and complications

The risk of ocular hypertension developing into glaucoma depends on the severity of the IOP, corneal thickness and age, among other factors. If appropriately treated the risk of glaucoma is reduced.

POAG usually progresses over several years and vision is lost peripherally first, and then may begin to affect central vision. Blindness develops in both eyes in around 5 to 10 percent of people. All vision loss associated with glaucoma is irreversible.

PACG more commonly leads to blindness, however if treatment is initiated early this improves outcomes. If a fellow eye (eye that is not affected during the initial development of PACG) is left untreated there is a 40 to 80 percent chance of an acute episode of angle closure over the following five to ten years.⁸

[Return to contents](#)

Diagnosis/detection

Signs of glaucoma and/or ocular hypertension may be picked up by optometrists during an eye test. In ocular hypertension and POAG the optometrist will usually refer the patient routinely to the ophthalmologist to make a formal diagnosis.^{9,10} Regular eye tests are available for certain groups of people, more information can be found on the **NHS Free NHS eye tests and optical vouchers** page.

Acute angle closure leading to rapid onset of symptoms is a medical emergency^{11,12} and can be suspected in a person with an acute painful red eye and the symptoms listed above, in this case urgent specialist ophthalmology assessment and treatment is required.¹³

For reference, sections 1.1 to 1.5 of NICE guidance **Glaucoma: diagnosis and management [NG81]** cover diagnosis and assessment of glaucoma in people aged 18 and over. In this guidance the clinical and cost effectiveness of treating people with an IOP above 21 mmHg but below 24 mmHg is discussed – see page 25 of 41.

In the following Fight for Sight video we meet Maureen and explore what it is like to receive a diagnosis of glaucoma.

Maureen's story living with glaucoma | Fight for Sight



[Return to contents](#)

Pharmacological treatment

The intention of glaucoma treatment is to maintain a person's visual function and related quality of life. Currently the only approach that has shown to be effective for maintaining visual function is reducing IOP.¹⁴

There are five main classes of medicines which are commonly used to treat glaucoma, all of which act to lower IOP.

- **Beta-blockers** are thought to reduce the amount of aqueous humour produced by the ciliary body and are effective in primary open-angle glaucoma. They are usually administered topically. Topical beta-blockers include betaxolol, levobunolol hydrochloride and timolol maleate.
- **Prostaglandin analogues and prostamides** increase uveoscleral outflow of aqueous humour and are used in ocular hypertension or open-angle glaucoma. This group includes the topical prostaglandin analogues latanoprost, tafluprost and travoprost and the synthetic prostamide, bimatoprost.
- **Sympathomimetics** or selective alpha2-adrenoceptor agonists are thought to reduce aqueous humour production. This group includes brimonidine and apraclonidine. Brimonidone is also thought to increase outflow via the uveoscleral pathway.
- **Carbonic anhydrase inhibitors** such as acetazolamide, brinzolamide, and dorzol inhibit carbonic anhydrase enzymes and reduce aqueous humour production. Acetazolamide is administered orally and brinzolamide and dorzolamide are used topically.
- **Miotics** such a pilocarpine act by opening the inefficient drainage channels in the trabecular meshwork.^{11,14,15}

For more information about the pharmacological treatment options for glaucoma visit:

- *British National Formulary (BNF) treatment summary **Glaucoma***
- Section 1.5 Treatment of NICE guidance **Glaucoma: diagnosis and management [NG81]**
- Section 3.3 – *Antiglaucoma drugs* of the European Glaucoma Society (EGS) **Terminology and guidelines for glaucoma**

Combination therapies and supporting people

If glaucoma is not controlled by monotherapy and a second eye drop is needed, a combination product should be offered before two separate eye drops. This helps people to use their eye drops as prescribed and helps to reduce washout of the first eye drop by the second, and exposure to preservatives.¹⁴

The EGS **Terminology and guidelines for glaucoma** covers combination therapies in section 3.3.1.2 *Add Second Drug/Combination Therapy*.

Supporting patients with using eye drops is a *Guidelines in practice* article which explores the importance of good eye drop technique and regular eye drop use. It links to the Moorfields eye hospital **Know Your Drops** campaign page which includes a video explaining how to use eye drops and links to patient information.

The IGA also offers the **Eye Drops and Dispensing Aids** booklet and a series of short films: **How to put drops in the eye**.

An IGA survey of over 1000 people with glaucoma showed that 45 percent had not been instructed how to use their eye drops, 91 percent were not assessed on their ability to use eye drops and 50 percent were not aware they should practise punctal occlusion (the process of gently pressing on the tear duct next to the nose after inserting the drop, this reduces systemic absorption and therefore potential side effects).^{16,17,18}

Providing information on how to use eye drops and the importance of punctal occlusion is an area where pharmacy professionals can work with patients to ensure that they are getting the most out of their eye drop treatments.

Eye irritation due to allergy, toxicity or inflammatory conditions can present as dry eye, burning/stinging, itching, irritation, tearing, foreign body sensation, red eye, and blurred vision. This group of symptoms indicates ocular surface disease (OSD),¹⁹ a term used to describe dry eye, eyelid disease, conjunctivitis and keratitis (inflammation of the cornea).²⁰

Preservatives in eye drops have been linked to ocular surface changes and referral to an ophthalmologist is recommended if an allergy to eye drops is suspected.^{21, 22}

NICE recommends preservative-free eye drops for:

- ocular hypertension if a person has an allergy to preservatives or people with clinically significant and symptomatic ocular surface disease, but only if they are at high risk of conversion to chronic open-angle glaucoma (COAG)
- COAG if there is evidence that the person is allergic to the preservative or has clinically significant and symptomatic ocular surface disease.¹⁵

The EGS **Terminology and guidelines for glaucoma** guideline also offers recommendations on the use of preservative free eye drops in Section 3.3.5 – *Local Toxicity of Topical IOP – Lowering Treatment The Role of Preservatives*.

[Return to contents](#)

Non-pharmacological treatment

Laser treatment and trabeculectomy (a surgical procedure to help fluid drain from the eye) are alternative treatments. For more information, visit NHS choices **Glaucoma, Treatment** page.

[Return to contents](#)

Patient support

Fight for Sight, the eye research charity, funds pioneering research to prevent sight loss and treat eye disease and offers information about the eye, eye conditions and patient stories.

National Eye Research Centre is a leading eye research charity that funds research into the causes of and treatments for eye disease, sight loss and blindness and also offers information about eye conditions.

Sightline is a charity which provides practical and emotional support to blind and partially sighted people through a telephone helpline.

The NHS has a dedicated **Glaucoma** page.

Glaucoma UK is a charity for people with glaucoma. They provide information, literature, advice and fund essential research to prevent unnecessary loss of sight through early detection, diagnosis and treatment. Their **campaign** page links to previous and upcoming campaigns and resources.

The **Royal National Institute of Blind People** offers a **glaucoma** information page and a **helpline** for those affected by sight loss.

[Return to contents](#)

Further resources

PrescQIPP has launched a **bulletin for eye preparation**, which, along with the supporting resources, reviews the place in therapy for eye products, in particular products for glaucoma and dry eye. It offers guidance on prescribing, self-care and cost-effective treatments. Cost comparison charts across a range of areas are available as supporting resources.

The College of Optometrists offers clinical management guidelines for optometrists; they contain information on diagnosis and management and also a lay summary which may be useful for explaining glaucoma in a person-centred way:

- **Glaucoma (primary open angle) (POAG)**
- **Primary Angle Closure/Primary Angle Closure Glaucoma (PAC / PACG)**
- **Glaucoma (steroid)**
- **Ocular hypertension (OHT)**

[Return to contents](#)

External websites

CPPE is not responsible for the content of any non-CPPE websites mentioned on this page or for the accuracy of any information to be found there.

All web links were accessed on 16 April 2021

[Return to contents](#)

References

1. National Institute for Health and Care Excellence. Clinical Knowledge Summaries. **Glaucoma. Definition.** June 2019.
2. National Institute for Health and Care Excellence. Clinical Knowledge Summaries. **Glaucoma. Anatomy and Physiology.** June 2019.
3. National Institute for Health and Care Excellence. Clinical Knowledge Summaries. **Glaucoma. Prevalence.** June 2019.
4. NHS. **Glaucoma. Overview.** February 2018.
5. National Institute for Health and Care Excellence. Clinical Knowledge Summaries. **Glaucoma, Primary angle closure glaucoma.** June 2019.
6. National Institute for Health and Care Excellence. Clinical Knowledge Summaries. **Glaucoma. Causes.** June 2019.
7. National Institute for Health and Care Excellence. Clinical Knowledge Summaries. **Glaucoma. Risk factors.** June 2019.
8. National Institute for Health and Care Excellence. Clinical Knowledge Summaries. **Glaucoma, Prognosis.** June 2019.
9. The College of Optometrists. **Glaucoma (primary open angle) (POAG).** May 2018.
10. The College of Optometrists. **Ocular hypertension (OHT).** May 2018.
11. British National Formulary. **Treatment summary – Glaucoma and ocular hypertension.** No date.
12. The College of Optometrists. **Primary Angle Closure / Primary Angle Closure Glaucoma (PAC / PACG).** May 2018.
13. National Institute for Health and Care Excellence. Clinical Knowledge Summaries. **Glaucoma. Scenario: Acute angle closure and angle closure glaucoma.** June 2019.
14. European Glaucoma Society. **Terminology and guidelines for glaucoma.** 4th ed. EU: SvetPrint; 2017.
15. National Institute for Health and Care Excellence. NICE guideline 81. **Glaucoma: diagnosis and management.** November 2017.
16. International Glaucoma Association. **IGA News Autumn 2014.**
17. International Glaucoma Association. **How to put drops in the eyes.** No date.
18. Mann E. **Glaucoma: treatment of open angle glaucoma and ocular hypertension.** *The Pharmaceutical Journal.* May 2015.
19. Stewart W C, Stewart J A, Nelson L A. **Ocular Surface Disease in Patients with Ocular Hypertension and Glaucoma.** *Current Eye Research.* 2011; 36(5): 391-398.
20. Leung E W, Medeiros F A, Weinreb R N. **Prevalence of Ocular Surface Disease in Glaucoma Patients.** *Journal of Glaucoma.* 2008; 17(5): 350-355.
21. Steven D W, Alaghband P, Lim K S. **Preservatives in glaucoma medication.** *British Journal of Ophthalmology* 2018; 0: 1-7.
22. National Institute for Health and Care Excellence. Clinical Knowledge Summaries. **Glaucoma. Scenario: primary open angle glaucoma and intraocular hypertension.** June 2019.

Fact sheet

Glaucoma

[Return to contents](#)

Last review: April 2021

Next review due: April 2022