

## ENTITLEMENT ELIGIBILITY GUIDELINES

# GLAUCOMA

**MPC** 00640  
**ICD-9** 365.1 and 365.2

### DEFINITION

Glaucoma is a medical condition referring to a group of diseases that has certain common features. These include an intraocular pressure (IOP) too high for the continued health of the eye, cupping and atrophy of the optic nerve head, and loss of visual field.

### DIAGNOSTIC STANDARD

A diagnosis by a qualified ophthalmologist is required. Investigations should include visual acuity, visual field (perimetry), and intraocular pressure (tonometry). Reports must be submitted with the application.

### ANATOMY AND PHYSIOLOGY

The eye has 3 chambers, the anterior chamber in front of the iris, the posterior chamber between the iris and the lens, and the vitreous chamber behind the lens.

Intraocular pressure is maintained by a balance between inflow and outflow of the aqueous humour, the fluid which nourishes the transparent structures of the eye. Aqueous humour is produced and secreted by the ciliary body, a gland behind the iris of the eye. Aqueous humour enters the anterior chamber of the eye through the pupil, and leaves by passing through the trabecular meshwork in the iridocorneal angle of the anterior chamber and back into venous circulation through the canal of Schlemm.

A number of classification schemes for the glaucomas have been proposed. They are based on the age of the person (infantile, juvenile, adult), the site of obstruction to aqueous outflow (pre-trabecular, trabecular, post-trabecular), the tissue principally involved (e.g. glaucoma caused by diseases of the lens), and etiology. Although each of these systems has value, the classification scheme that separates angle closure from open angle glaucoma has been used most widely, because it focuses on pathophysiology and points to proper clinical management.

A classification outline for open angle, angle closure, combined-mechanism and childhood glaucoma follows:

**A. Open angle glaucoma**

1. Primary open angle glaucoma
  - Optic nerve damage and visual field loss associated with increased intraocular pressure (IOP)
  - Trabecular obstruction, cause not known
2. Glaucoma suspect
  - Normal optic disc and visual field associated with elevated IOP
  - Suspicious optic disc and/or visual field with normal IOP
3. Normal-tension glaucoma
  - Optic nerve damage and visual field loss associated with normal IOP
4. Secondary open angle glaucoma
  - Increased resistance to trabecular meshwork outflow associated with another condition, e.g. pigmentary glaucoma, phacolytic glaucoma, steroid-induced glaucoma, ocular inflammation
  - Increased post-trabecular resistance secondary to elevated episcleral venous pressure, e.g. carotid-cavernous sinus fistula

**B. Angle closure glaucoma**

1. Primary angle closure glaucoma with relative pupillary block
  - aqueous humor from posterior chamber to anterior chamber restricted; peripheral iris in contact with trabecular meshwork
2. Primary angle closure glaucoma without pupillary block, e.g. plateau iris
3. Secondary angle closure glaucoma with pupillary block, e.g. swollen lens, secluded pupil
4. Secondary angle closure glaucoma without pupillary block
5. Posterior pushing mechanism: lens iris diaphragm pushed forward, e.g. posterior segment tumor, scleral buckling procedure, uveal effusion
6. Anterior pulling mechanism: anterior segment process pulling iris forward to form peripheral anterior synechiae, e.g. iridocorneal endothelial syndrome, neovascular glaucoma, inflammation

**C. Combined-mechanism glaucoma**

1. A combination of two or more forms of glaucoma, e.g. open angle glaucoma in a person who develops secondary angle closure following a scleral buckling procedure

**D. Childhood glaucoma**

1. Primary congenital/infantile glaucoma

2. Juvenile
3. Glaucoma associated with congenital anomalies
  - associated with ocular disorders, e.g. anterior segment dysgenesis, aniridia
  - associated with systemic disorders, e.g. rubella, Lowe's syndrome
4. Secondary glaucoma in infants and children, e.g. glaucoma secondary to retinoblastoma or trauma

## CLINICAL FEATURES

Glaucoma is a leading cause of blindness.

An intraocular pressure (IOP) of 21 mmHg or greater is associated with glaucoma. This increased pressure may damage the optic nerve, the head of which is known as the optic disc. The disc may develop cupping and pallor which results in "blind spots" or scotomata in the person's field of vision. These scotomata may enlarge, coalesce and eventually lead to blindness.

An IOP of 21 mmHg or more does not necessarily mean a diagnosis of glaucoma, as the person would also need to demonstrate a glaucomatous visual field defect or optic nerve damage. There are individuals with an IOP over 21 who never develop glaucoma, as well as people with sensitive eyes who develop glaucomatous visual field defects with pressures less than 21.

While the peripheral visual field gradually shrinks, fixation remains unaffected until the late stages of the disease.

Open angle glaucoma is the most common type of glaucoma, occurring in approximately two per cent of people over 40 years of age. It is asymptomatic in its early stages, but if left untreated blindness can eventually result. It does not usually cause pain or a red eye, and has only moderately elevated intraocular pressure. It is bilateral, insidious in onset, and slowly progressive. By the time the person notices visual loss, there is substantial irreversible damage to peripheral vision.

Angle closure glaucoma occurs when the iridocorneal angle is closed off and IOP rises to very high levels. It may cause pain and blurring of vision.

Congenital glaucoma is due to a congenital malformation of the iridocorneal angle. Most of these cases are diagnosed in the first three months of life.

## PENSION CONSIDERATIONS

### A. CAUSES AND/OR AGGRAVATION

**THE TIMELINES CITED BELOW ARE NOT BINDING. EACH CASE SHOULD BE ADJUDICATED ON THE EVIDENCE PROVIDED AND ITS OWN MERITS.**

**NOTE:** THERE ARE MANY CAUSES OF, AND AGGRAVATION FACTORS IN, GLAUCOMA. THE FOLLOWING REPRESENT ***THE MOST COMMONLY SEEN*** BY VETERANS AFFAIRS CANADA:

1. Idiopathic

- The cause may be unknown.

2. Uveitis

- Uveitis is an inflammation of the vascular middle coat of the eyeball, consisting of the iris, ciliary body and choroid.
- It would have occurred within a few months prior to clinical onset or aggravation of angle closure glaucoma or open angle glaucoma.

3. Significant trauma to the affected eye

- Significant trauma would include a penetrating injury, blunt trauma, radiation injury, or acid burn injury to the affected eye which results in intraocular inflammation, bleeding, or other tissue injury.
- It would have occurred within several months prior to clinical onset or aggravation of angle closure glaucoma or open angle glaucoma.

4. Keratoplasty or other intraocular surgery of the affected eye

- Keratoplasty is an operative procedure in which the entire thickness of the cornea is removed and replaced by donor tissue.
- It would have occurred at any time prior to clinical onset or aggravation of angle closure glaucoma or open angle glaucoma.

5. Pseudoexfoliation

- Pseudoexfoliation occurs when deposits of unknown origin and

- composition are seen on the lens surface, posterior iris surface, ciliary processes, and zonule, in the trabecular meshwork, and loose in the anterior chamber.
- It would have occurred at any time prior to clinical onset or aggravation of angle closure glaucoma or open angle glaucoma.

6. Iridocorneal endothelial (ICE) syndrome

- Iridocorneal endothelial syndrome is a syndrome in which there is a character abnormality of the corneal endothelium.
- It should be present at time of clinical onset or aggravation of angle closure glaucoma or open angle glaucoma.

7. For angle closure glaucoma only

- cataract of the affected eye at time of clinical onset
- anterior subluxation or dislocation of the lens of the affected eye at time of clinical onset
- undergoing treatment with a drug that can cause mydriasis or miosis or a drug reported in medical literature to have caused acute angle closure glaucoma, where that treatment has occurred within the 24 hours before clinical onset. These drugs would include, but are not limited to:
  - atropine
  - sympathomimetics (e.g. salbutamal)
  - scopolomine

8. For open angle glaucoma only

- for *ghost cell glaucoma only* - Ghost cell glaucoma is an open angle glaucoma which results from obstruction to aqueous outflow by degenerated red blood cells devoid of hemoglobin (ghost cells) in the aqueous humor. A vitreal hemorrhage, hyphema or intraocular surgery involving the affected eye would have occurred prior to clinical onset.
- for *phacolytic glaucoma only* - Phacolytic glaucoma is a form of open angle glaucoma resulting from leakage of lens protein into the aqueous humor from a cataract. A cataract involving the affected eye would have

occurred prior to clinical onset.

9. Neovascularization of the iridocorneal angle of the affected eye

- A condition which may give rise to neovascularization of the iridocorneal angle would include, but is not limited to, one of the following conditions:
  - central retinal vein obstruction of the affected eye
  - diabetic retinopathy of the affected eye
  - ipsilateral carotid artery occlusive disease of the affected eye
  - retinal detachment of the affected eye

10. Familial

- There is usually a strong family predisposition to glaucoma.

11. Inability to obtain appropriate medical treatment

**Exclusion:**

Despite research efforts to date, there is a lack of sufficient evidence at this time to establish for pension purposes a relationship between glaucoma and avitaminosis.

**B. MEDICAL CONDITIONS WHICH ARE TO BE INCLUDED IN ENTITLEMENT/ASSESSMENT**

**C. COMMON MEDICAL CONDITIONS WHICH MAY RESULT IN WHOLE OR IN PART FROM GLAUCOMA AND/OR ITS TREATMENT**

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