

Open-Angle Glaucoma

The drain inside the eye gets clogged over time, so pressure builds and crushes the nerve that sends pictures to the brain—stealing vision from the edges inward without any pain.

THE ONE-LINE TRUTH

Slow, painless rise in eye pressure that quietly damages the optic nerve and side vision.

The chain — how it unfolds

1. Trabecular meshwork clogs and outflow resistance rises — why: Aging, genetics, and debris slowly damage the drain cells, so fluid cannot leave the eye fast enough and pressure climbs above the safe range (often above 21 mm Hg).
2. Intraocular pressure squeezes the optic nerve head — why: The optic disc (where the nerve exits the back of the eye) is the weakest spot in the wall of the eyeball, so rising pressure bends and crushes the nerve fibers and the tiny blood vessels that feed them.
3. Optic nerve axons lose their blood supply and die — why: Squeezed capillaries cannot deliver oxygen; the long nerve fibers (axons) that run from the retina to the brain starve and break apart.
4. Retinal ganglion cell bodies die — why: When an axon is cut or crushed, the whole nerve cell dies—so patches of retina go dark and stop sending signals to the brain.
5. Peripheral visual field shrinks inward (tunnel vision) — why: The nerve fibers that serve side vision die first (they enter the optic nerve at the edges), so you lose the ability to see things off to the side, above, or below—while straight-ahead vision may still be clear.
6. Optic disc develops cupping — why: As nerve fibers disappear, the center of the optic disc caves inward like a bowl (increased cup-to-disc ratio), which an eye doctor can see during an exam.
7. Central vision is eventually lost if untreated — why: In late-stage disease, the pressure and starved blood flow (ischemia) (not enough blood reaching the tissue to feed it) finally destroy the central nerve fibers, and you go blind.

- 8.** Damage is permanent and progressive without treatment — why: Dead nerve cells never regenerate, and rising pressure continues to kill more fibers every month—so early detection and pressure-lowering treatment (drops, laser, or surgery) are the only way to save the vision that remains.

Spot it — what you see, and why

- Gradual loss of side vision (peripheral vision): Pressure inside the eye builds up slowly over months or years. This pressure squeezes and damages the optic nerve (the cable that sends pictures from your eye to your brain). The nerve fibers on the outer edges die first, so you lose the ability to see things off to the side while your straight-ahead vision stays clear for a long time.
- Tunnel vision in late stages: As more and more optic nerve fibers die from ongoing pressure, the only vision left is a small circle straight ahead—like looking through a narrow tube. The outer ring of nerve fibers is already gone, so only the center bunch is still working.
- No pain: In open-angle glaucoma, the drain inside the eye clogs very slowly and gently. There is no sudden spike in pressure and no stretching of pain-sensing nerves in the eye, so you feel nothing even as damage builds.
- Difficulty seeing in dim light or at night: When part of the optic nerve is damaged, your brain gets fewer signals about contrast and detail. In low light, your eye needs every nerve fiber to build a clear picture—so any loss makes dim scenes look muddled or hard to read.
- Trouble adjusting when moving from bright to dark spaces: A healthy optic nerve quickly sends updated brightness signals to your brain. When nerve fibers are dying, the communication slows down and becomes patchy, so your brain takes longer to make sense of the new light level.
- Increased cup-to-disc ratio on eye exam: The optic nerve head (where the nerve enters the back of the eye) has a small dip in the center called the cup. Chronic high pressure kills nerve tissue around the cup, making the dip larger and deeper—like a pothole getting bigger. An eye doctor measures this change to track damage.

Red flags — act now

- Sudden, severe eye pain (suggests acute angle-closure glaucoma instead, which is an emergency)
- Rapid vision loss over days or weeks (open-angle glaucoma is slow; fast loss means something else urgent is happening)
- Severe headache with nausea and vomiting along with blurry vision (points to sudden high pressure or another brain/eye emergency)

- Only one eye affected and vision dropping quickly (open-angle is usually both eyes; one-sided fast loss suggests stroke, detachment, or infection)

Treat it — in this order

1. Prostaglandin analog eye drops (latanoprost, travoprost, bimatoprost, tafluprost) once nightly → These drops increase fluid drainage out of the eye through a second pathway (the uveoscleral route, which bypasses the clogged main drain). They lower eye pressure by about 25-35%, which is the biggest drop of any single medicine. Lower pressure slows or stops nerve damage.
2. Beta-blocker eye drops (timolol, betaxolol, levobunolol) once or twice daily → These drops slow down the eye's production of fluid (aqueous humor), so less fluid builds up. They lower IOP by about 20-25%. Often added when a prostaglandin alone is not enough.
3. Alpha-agonist eye drops (brimonidine, apraclonidine) two or three times daily → These drops both reduce fluid production and slightly increase drainage. They lower IOP by about 15-20%. Brimonidine may also protect the optic nerve directly (neuroprotection), though this is still being studied.
4. Carbonic anhydrase inhibitor eye drops (dorzolamide, brinzolamide) two or three times daily → These drops reduce fluid production by blocking an enzyme (carbonic anhydrase) in the eye. They lower IOP by about 15-20%. They are often combined with a beta-blocker in one bottle for convenience.
5. Selective laser trabeculoplasty (SLT) → A laser gently heats the trabecular meshwork (the drain) to wake it up and make it work better. It lowers IOP by about 20-30% and can replace or reduce the number of eye drops. The effect lasts 1-5 years on average, and the laser can be repeated if pressure goes back up.

Your top priorities at the bedside

1. Teach the patient how to put in eye drops correctly and build a daily routine so no doses are missed. — why: The drops are the main treatment to lower eye pressure and protect what vision is left. But studies show about half of patients don't use them correctly or regularly. If drops are used wrong (like touching the dropper to the eye, not waiting between different drops, or missing days), the pressure stays high and the optic nerve keeps getting damaged. Walking through the technique step-by-step and linking it to something they already do every day (like brushing teeth) makes it far more likely to happen.

2. Check if the patient can afford their medications and help connect them to assistance programs if cost is a barrier. — why: Glaucoma drops can be expensive, and many patients skip doses or stop altogether because they cannot pay for refills. When treatment stops, eye pressure climbs back up and more permanent vision loss happens. Asking directly and non-judgmentally about cost, then helping find manufacturer programs, generic options, or community resources keeps the treatment plan from failing.
3. Assess the patient's current vision and daily safety, especially fall risk and ability to drive or move around safely. — why: Glaucoma takes away side vision first, which means the patient might not see curbs, steps, or cars coming from the side. This raises the risk of falls, car crashes, and injuries. Understanding what they can and cannot see helps us teach compensation strategies (like turning the head more, using better lighting, removing tripping hazards at home) and refer for low-vision services or occupational therapy when needed.