

Angle-Closure Glaucoma (Closed-Angle)

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THE STORY IN ONE SENTENCE

The drain inside your eye suddenly slams shut, pressure shoots sky-high in minutes, and the nerve that carries pictures to your brain can die within hours.

“Your iris flips forward like a curtain and blocks the drain — fluid piles up, pressure spikes fast, and without emergency care you can lose your sight before the day is over.”

Normal anatomy — the body's job

Your eye constantly makes a clear, watery fluid called aqueous humor in a ring of tissue (the ciliary body) that sits just behind your iris. That fluid flows forward through the pupil, circles around the front of the eye to feed the lens and cornea with nutrients and oxygen, and then drains out through a spongy mesh (the trabecular meshwork) tucked into the drainage angle — the corner where your iris meets the cornea. From there it empties into a tiny canal (Schlemm's canal) and flows into small veins, like water leaving a sink. The balance between how much fluid is made and how much drains out keeps the pressure inside your eye steady and safe.

BODY'S JOB

Aqueous humor delivers food and oxygen to parts of the eye that have no blood vessels of their own — especially the lens (the clear disk that focuses light) and the inside layer of the cornea (the clear window at the front). The drainage system keeps the amount of fluid inside balanced so pressure stays in the normal range, usually between 10 and 21 millimeters of mercury. Steady pressure holds your eyeball in the right shape and protects the delicate optic nerve at the back, which carries all the visual signals to your brain.

1. The ciliary body (a ring of tissue behind the iris) makes fresh aqueous humor every minute of the day.
2. Fluid flows forward through the pupil (the round opening in the center of your iris).

3. Fluid spreads around the front chamber of the eye, bathing the lens and the inner cornea with nutrients and oxygen.
4. Fluid drains out through the trabecular meshwork, a spongy mesh in the drainage angle where the iris and cornea meet at the edge.
5. Drainage empties into Schlemm's canal (a tiny circular channel), which funnels fluid into small veins that carry it away, keeping pressure balanced.

What breaks

Your iris suddenly moves forward or gets pushed hard against the drainage angle, slamming the exit shut. Fluid keeps being made at the normal rate but cannot leave, so pressure inside the eyeball climbs fast — like covering the drain in a sink while the tap is still running full blast.

CLINICAL TERM

Acute primary angle closure: rapid, complete obstruction of the trabecular meshwork caused by apposition (touching and pressing) of the peripheral iris against the cornea at the drainage angle, blocking aqueous outflow and causing a sudden spike in intraocular pressure.

How the body compensates

The eye has almost no way to lower pressure once the drain is completely blocked. The ciliary body keeps making fluid at the same steady rate — it has no automatic shut-off — so pressure climbs fast until the eyeball itself stretches and tissues begin to die. The few responses the eye does mount are weak and do not come close to fixing the problem.

1. {'step': 'A small amount of fluid tries to squeeze out through a backup route called uveoscleral outflow, draining through the ciliary muscle and out through the white of the eye (the sclera).', 'limit': 'This route only handles about 10 percent of normal drainage — nowhere near enough to keep up with the fluid still being made. Pressure keeps climbing.'}
2. {'step': 'The cornea swells with water (corneal swelling (edema)) because the high pressure forces fluid into the corneal layers faster than the pump cells on the inside (the endothelium) can clear it out. Your vision turns blurry and you see rainbow halos around lights.', 'limit': 'Corneal swelling is a distress signal — a visible sign of damage — not a mechanism that lowers pressure or fixes the blockage.'}

3. {'step': 'Pain receptors in the eye and around the eye socket fire intensely because the sudden stretch of the eyeball activates nerve endings, and branches of the trigeminal nerve (cranial nerve V, the main nerve that senses the face and eye) send alarm signals to the brain.', 'limit': 'Pain is your body's urgent warning to get help immediately; it does not open the drain or drop the pressure.'}
4. {'step': 'Blood vessels on the surface of the eye dilate and turn the white part (the sclera) bright red because the eye is irritated and blood-starved (ischemic) (not getting enough oxygen).', 'limit': 'Redness shows the eye is in serious trouble, but it does not reopen the angle or relieve the pressure.'}
5. {'step': 'The nausea center in your brainstem can fire because the trigeminal nerve pathways overlap with the vagus nerve (cranial nerve X, which controls nausea), so severe eye pain can make you feel sick and vomit.', 'limit': 'Nausea is a side effect of the pain signals crossing in the brain; it has no role in fixing the eye problem.'}

Domino effect

1. The iris bows forward and its outer edge presses flat against the trabecular meshwork, blocking the drainage angle completely. — Fluid trapped behind the iris (often by pupillary block — the iris sticking to the lens) pushes the iris forward like air inflating a sail. The peripheral iris then touches the inside corner of the cornea and seals off the drain, so aqueous humor has nowhere to go.
2. Intraocular pressure spikes — often shooting above 40 to 60 millimeters of mercury within minutes to hours (normal is 10 to 21). — The ciliary body keeps making fluid at the usual rate (about 2 microliters per minute), but now the drain is shut. Fluid piles up inside a closed space, and pressure climbs fast — like blowing air into a balloon that cannot let any out.
3. The cornea swells and turns cloudy (corneal swelling (edema)), and you see rainbow halos around lights. — High pressure forces water into the layers of the cornea faster than the pump cells (the corneal endothelium) can clear it. The swelling scatters light, making your vision foggy and creating colorful halos — a hallmark sign.
4. You feel crushing pain in and around the eye, a severe headache on that side of your head, and often nausea and vomiting. — The sudden stretch activates pain fibers in the eye itself and along the branches of the trigeminal nerve (cranial nerve V), which supplies sensation to the eye, forehead, and face. Those nerve signals cross paths in the brainstem with the vagus nerve (cranial nerve X), which controls the stomach, so the brain's vomiting center can fire even though the problem is in the eye.

5. The pupil becomes mid-dilated (partially open), often oval instead of round, and does not react to light (a fixed pupil). — The iris muscle is stunned by starved blood flow (ischemia) (the high pressure crushes the tiny blood vessels feeding it, so it runs out of oxygen) and cannot squeeze or relax normally. A stuck, misshapen pupil is a classic exam finding.
6. The optic nerve at the back of the eye starts to die (ischemic optic neuropathy). — The skyrocketing pressure squeezes the small arteries that feed the optic nerve head (where the nerve enters the eyeball). Without blood flow, the nerve fibers run out of oxygen and begin to die within hours — just like a stroke in the brain.
7. Vision loss becomes permanent if the pressure is not lowered within hours. — Once optic nerve fibers are dead, they never grow back or regenerate. You lose that part of your visual field forever — often starting with peripheral (side) vision and progressing to total blindness in that eye if untreated.
8. Without emergency treatment, the entire eye can go blind and develop chronic pain, inflammation inside the eye (uveitis), or scarring. — Prolonged high pressure kills the whole optic nerve and damages other structures (the retina, the iris, the lens). The dying tissue can trigger ongoing inflammation, and scar tissue can form, leading to a painful, blind eye that may eventually need to be removed or treated with ongoing drops to control pain.

Symptoms · what the patient feels, and why

- Sudden, severe eye pain: The pressure inside the eye shoots up very fast when the drain for eye fluid suddenly gets blocked (the iris flops forward and covers the drainage angle like a curtain falling over a gutter). This stretches the eyeball's wall and presses on pain nerves inside, causing intense aching or throbbing pain.
- Blurry vision or sudden vision loss: When pressure inside the eye spikes (sometimes above 40 or 50 mmHg, much higher than the normal 10–21), it squeezes the optic nerve (the cable that carries pictures from your eye to your brain) and cuts off blood flow to the retina (the thin sheet at the back of the eye that captures light). Without enough blood and oxygen, the nerve and retina can't work right, so vision gets foggy or goes dark.
- Seeing halos or rainbow rings around lights: The high pressure makes the cornea (the clear dome on the front of your eye) swell with fluid, like a sponge soaking up water. The swollen cornea scatters light into colors as it passes through, creating rainbow rings around lamps, headlights, or any bright source.
- Red eye: The sudden pressure increase makes tiny blood vessels on the white part of the eye (the conjunctiva and episclera) swell and leak, turning the eye pink or red. It's like when your skin gets red and puffy after you bump it—the blood vessels open up in response to stress and injury.

- **Headache (often on the same side as the affected eye):** Pain nerves from the eye share the same pathways to the brain as nerves from your forehead and temples (they all feed into the trigeminal nerve, the face's main feeling nerve). When the eye hurts badly, the brain sometimes reads that signal as head pain too, especially around the eyebrow and temple on that side.
- **Nausea and vomiting:** The eye pain is so intense that it triggers the vagus nerve (a big nerve that runs from your brain down to your stomach and gut). This nerve tells your stomach to feel sick and sometimes makes you throw up, even though the problem is in your eye, not your belly.
- **Hard, firm eyeball when gently touched:** The fluid trapped inside the eye has nowhere to go, so pressure builds up like air in an over-inflated basketball. When a doctor carefully presses on the closed eyelid, the eyeball feels rock-hard instead of soft and springy like a normal eye.
- **Pupil that is mid-sized and does not respond to light:** The high pressure damages or stuns the iris sphincter (the tiny ring muscle that makes the pupil shrink in bright light). The pupil gets stuck in a middle size—neither wide nor pinpoint—and won't shrink when you shine a light at it, unlike a healthy eye that tightens right away.
- **Cloudy or steamy-looking cornea:** The cornea (the clear window at the front of the eye) swells because the high pressure pushes fluid into its layers, a process called corneal swelling (edema) (puffiness of the cornea). The extra fluid makes the cornea look hazy or cloudy, like a foggy windshield, instead of crystal clear.

Focused exam

- **Intraocular pressure (IOP) measurement using tonometry:** Normal eye pressure is 10–21 mmHg. In angle-closure glaucoma, pressure jumps above 30 mmHg (often 40–80 mmHg) because the fluid inside the eye cannot drain out—like a sink with a clogged drain that keeps filling. This high pressure crushes the optic nerve (the cable that carries vision signals to your brain), which can destroy vision permanently within hours if not treated.
- **Pupil reaction to light:** The pupil (the black circle in the center of your eye) normally gets smaller when you shine a light on it. In angle-closure glaucoma, the pupil is often stuck mid-size (4–6 mm) and barely reacts or does not react at all—like a broken dimmer switch. This happens because the high pressure and inflammation inside the eye paralyze the iris sphincter (the ring of muscle that squeezes the pupil smaller).

- **Slit-lamp examination of the anterior chamber (front space of the eye):** The doctor looks at the space between the clear front window of your eye (the cornea) and the colored part (the iris). In angle-closure glaucoma, this space is very shallow—like a room with a low ceiling—because the iris is pushed forward and blocks the drain holes in the corner (the trabecular meshwork, the filter that lets fluid out). A shallow chamber makes angle closure much more likely and confirms the diagnosis.
- **Corneal swelling (edema) (swelling of the clear front window of the eye):** When eye pressure spikes, the cornea swells with extra water because its pump cells (the endothelium, a single layer of cells on the back surface of the cornea) cannot keep up—like a sponge soaking up water. This makes the cornea look cloudy or hazy and causes the patient to see halos around lights. Corneal swelling (edema) is a red flag that pressure has been dangerously high.
- **Gonioscopy (using a special mirror lens to look at the drainage angle):** This test lets the doctor see the angle where fluid drains out of the eye (the trabecular meshwork, located in the corner where the iris meets the cornea). In angle-closure glaucoma, the iris is pushed up against this drain and blocks it—like a shower curtain sucked against a drain. Seeing a closed or very narrow angle (less than 10 degrees or no trabecular meshwork visible) confirms the diagnosis and tells the doctor that pressure will not come down until the blockage is relieved.
- **Red eye (conjunctival injection) and ciliary flush (ring of redness around the cornea):** The blood vessels on the white part of the eye (the conjunctiva) and around the cornea get swollen and red because the high pressure and inflammation irritate them. A ring of deep red around the cornea (ciliary flush) is especially important—it tells you the problem is inside the eye, not just on the surface like pink eye. This is a warning sign of serious eye disease like angle-closure glaucoma or uveitis (inflammation inside the eye).

Diagnostics · every test answers a question

- **Intraocular pressure (IOP) measurement by applanation tonometry** → Is the pressure inside the eye dangerously high right now?
- **Gonioscopy with a Goldmann or Zeiss gonioscope** → Is the drainage angle open or closed, and how much of it is blocked?
- **Visual acuity testing (reading an eye chart at 20 feet)** → How much vision has the patient already lost?
- **Dilated funduscopy (ophthalmoscopy) to examine the optic nerve head** → Is the optic nerve swollen, pale, or permanently damaged?
- **Anterior-segment optical coherence tomography (AS-OCT) or ultrasound biomicroscopy (UBM)** → Why is the angle closed—what anatomical problem is pushing the iris forward?

- Slit-lamp examination for cells and flare in the anterior chamber → Is there inflammation inside the eye that could be causing or worsening the high pressure?
- Visual field testing (perimetry) → Has the patient lost any side vision from optic nerve damage?
- Optical coherence tomography (OCT) of the optic nerve head and retinal nerve fiber layer → How much nerve tissue has been lost, and is the nerve fiber layer thinned?

Treatment logic

- Intravenous hyperosmotic agent: mannitol 20% IV (1–2 g/kg over 30–60 minutes) OR oral glycerin 50% solution (1–1.5 g/kg) → These medicines pull water out of the eye by creating a concentration difference in the blood—like adding salt to a sponge to make it release water. Mannitol and glycerin shrink the vitreous gel (the clear jelly that fills the back of the eyeball), which drops the pressure fast, usually within 30–60 minutes. This buys time to do definitive treatment such as laser iridotomy. Mannitol is given in the hospital through an IV; glycerin can be given by mouth in the office or emergency department.
- Topical beta-blocker (timolol 0.5% eye drops, one drop twice) and topical alpha-2 agonist (apraclonidine 1% or brimonidine 0.2%, one drop) → Beta-blockers reduce the amount of clear fluid (aqueous humor) your eye makes inside the ciliary body (the ring of tissue behind the iris that produces eye fluid), by blocking beta receptors on the cells that make fluid. Alpha-2 agonists (apraclonidine and brimonidine) also reduce fluid production and help fluid drain out through a backup route called the uveoscleral pathway (through the wall of the eye). Together they lower pressure by 20–30% within 1–2 hours—like turning down the faucet and opening a second drain at the same time.
- Carbonic anhydrase inhibitors: oral acetazolamide 500 mg OR topical dorzolamide 2% or brinzolamide 1% eye drops → These medicines block an enzyme called carbonic anhydrase inside the ciliary body, which the cells need to make aqueous fluid. Blocking this enzyme cuts fluid production by 40–50%. Oral acetazolamide works faster and stronger than the eye drops and begins lowering pressure within 2 hours—like shutting off most of the water supply to a backed-up sink. The eye drops (dorzolamide or brinzolamide) are weaker but still help and can be used if the patient cannot take pills.
- Topical pilocarpine 1–2% eye drops (one drop every 15 minutes for two doses, then every 6 hours), given ONLY after IOP drops below 40 mmHg → Pilocarpine is a cholinergic agonist that forces the pupil to constrict (get very small) by stimulating the iris sphincter muscle. This pulls the peripheral iris away from the trabecular meshwork (the drain) and can help open the angle—like pulling back a curtain. It also makes the ciliary muscle contract, which stretches and opens the trabecular meshwork to help fluid escape.

- Topical corticosteroid eye drops (prednisolone acetate 1%, one drop every 1–2 hours initially, then tapered) → Steroid eye drops reduce inflammation inside the eye triggered by the high pressure and the blocked angle. Inflammation causes pain, redness, and makes the angle tissues sticky and swollen, which can keep the angle closed even after pressure drops. Steroids (such as prednisolone acetate) calm this inflammation by blocking inflammatory signals like prostaglandins and cytokines inside the eye.
- Laser peripheral iridotomy (LPI) using Nd:YAG or argon laser → The ophthalmologist uses a laser to burn a tiny hole (about 0.2–0.5 mm) through the peripheral iris (the outer edge, away from the pupil). This creates a permanent shortcut for aqueous fluid to flow from the posterior chamber (the space behind the iris) directly into the anterior chamber (the space in front of the iris), so fluid no longer builds up behind the iris and pushes it forward. The iris falls back, the angle opens, and pressure drops to normal—like punching a relief hole in a dam. This is the definitive treatment for pupillary block angle closure (the most common type) and prevents future attacks.
- Phacoemulsification with intraocular lens implantation (cataract surgery) → The surgeon removes the natural lens of the eye (which is often large and contributes to the narrow angle) through a small incision, breaks it into pieces using ultrasound (phacoemulsification), and replaces it with a thin artificial lens (intraocular lens, or IOL). Removing the thick natural lens makes much more room inside the front of the eye, so the iris falls back and the angle opens wide and stays open permanently. This is the most definitive treatment for angle closure caused by a large or forward lens (phacomorphic glaucoma) and for patients with narrow angles and cataracts. Pressure usually drops to normal and stays there long-term without needing daily eye drops. It also improves vision if the lens was cloudy.

Medications

Timolol (eye drops) · Beta-blocker (for the eye)

Timolol slows down the cells inside your eye that make the clear fluid (aqueous humor, the watery liquid filling the front of your eye). When those cells slow down, they make less fluid. With less new fluid being made every minute, the pressure inside your eye drops.

1. Timolol blocks tiny switches called beta-2 receptors on the cells in the ciliary body.
2. When those switches are blocked, the cells get a slow-down signal.
3. The cells then make less aqueous humor (the clear fluid).
4. With less fluid being made, the pressure inside your eye slowly falls over the next hour or two.
5. Lower pressure protects the optic nerve (the cable at the back of your eye that carries sight signals to your brain) from being squeezed and damaged.

SAFETY WATCH

Can slow your heart rate or make asthma worse, because a small amount of the drop gets absorbed into your bloodstream and reaches your heart and lungs. · Tell your doctor if you have asthma, COPD (chronic obstructive pulmonary disease, a lung condition that makes breathing hard), a slow heart rate (bradycardia), or heart failure before using timolol. · May hide the shaking and fast heartbeat that warn you of low blood sugar if you have diabetes. · If you take other heart or blood pressure medicines (like calcium channel blockers or other beta-blockers), your doctor needs to know — the effects can add up and slow your heart too much.

Brimonidine (eye drops) · Alpha-2 agonist

Brimonidine works two ways at once: it tells the ciliary body to make less fluid, and it helps the fluid that is already there drain out through a back route called the uveoscleral pathway (think of it as a side door for fluid to leave the eye, seeping out through the wall). Both actions lower the pressure.

1. Brimonidine turns on alpha-2 receptors on cells in the ciliary body, telling them to slow down fluid production.
2. At the same time, it relaxes the tissues around the uveoscleral pathway, making it easier for fluid to drain out through that side route.
3. Both effects — less fluid coming in and more fluid leaving — work together to lower the pressure inside your eye within one to two hours.

SAFETY WATCH

Can cause redness in the white of your eye, dry mouth, tiredness, or a strange metallic taste. · Should NOT be used in babies or very young children (under age 2) because it can cause dangerous sleepiness, weak breathing, and a drop in body temperature. · Rarely can cause an allergic reaction with swollen, red, itchy eyelids after a few weeks of use. · Tell your doctor if you take medicines for depression (especially MAO inhibitors or tricyclic antidepressants) — brimonidine can interact with them and cause dangerous blood pressure swings.

Dorzolamide or Brinzolamide (eye drops) · Carbonic anhydrase inhibitor (topical, meaning directly in the eye)

These drops block an enzyme called carbonic anhydrase that the ciliary body needs to make fluid efficiently. This enzyme helps the cells pump water and salts into the fluid. When the enzyme is blocked, the cells make much less aqueous humor, so pressure drops.

1. Dorzolamide or brinzolamide soaks through your cornea and enters the ciliary body cells.

2. It blocks carbonic anhydrase II (the enzyme that helps the cell move water and bicarbonate, a type of salt, into the fluid).
3. Without that enzyme working well, the cell cannot make as much aqueous humor.
4. Less fluid production means lower pressure inside your eye, usually within an hour or two.

SAFETY WATCH

Can cause a bitter or sour taste in your mouth right after you put the drop in (the drop drains through your tear duct into your throat). · May sting or blur your vision for a few minutes after you use it. · Should NOT be used if you are allergic to sulfa drugs (sulfonamides), because these drops are chemically related to sulfa antibiotics. · Rarely can cause redness, a feeling like something is in your eye, or blurred vision that lasts longer.

Acetazolamide (pill or IV) · Carbonic anhydrase inhibitor (systemic, meaning it works throughout your whole body)

Acetazolamide is a pill (or IV) version of the enzyme blocker. It travels through your bloodstream to the ciliary body and shuts down carbonic anhydrase all over your body. This dramatically slows fluid production in your eye and quickly lowers pressure — it is the fastest medical way to bring down dangerously high eye pressure during an acute angle-closure attack.

1. You swallow the pill (usually 500 mg) or get it through an IV; the drug enters your bloodstream within minutes.
2. Acetazolamide reaches the ciliary body and blocks carbonic anhydrase II.
3. With the enzyme blocked, the cells can barely make any new aqueous humor.
4. Pressure inside your eye drops quickly — often within one to two hours.
5. At the same time, your kidneys lose the enzyme too, so you pee out extra bicarbonate, salt, water, and potassium, which lowers fluid volume everywhere in your body slightly.

SAFETY WATCH

Makes you pee a lot and can wash out potassium from your body, so your doctor may check your potassium level or tell you to eat potassium-rich foods (bananas, oranges, potatoes, spinach). · Can cause tingling (paresthesias) in your fingers, toes, or around your mouth (this is harmless but annoying — it happens because your blood becomes slightly more acidic). · May make fizzy drinks (soda, sparkling water) taste flat. · Should NOT be used if you have a sulfa allergy, severe kidney disease, or very low potassium or sodium in your blood. · Rarely can cause kidney stones if used long-term (it makes your urine more alkaline, which can form calcium phosphate stones) — drink plenty of water. · Can cause tiredness, confusion, or loss of appetite, especially in older adults. · Not safe to use long-term during pregnancy (Category C); only used in emergencies.

Pilocarpine (eye drops) · Cholinergic agonist (also called a muscarinic agonist, meaning it mimics the nerve signal acetylcholine)

Pilocarpine makes your pupil shrink and pulls the iris away from the drain in the angle. In angle-closure, the iris was blocking the drain like a curtain over a window; pilocarpine yanks the curtain back, reopening the drain and letting fluid flow out. Pressure drops because fluid can escape again through the trabecular meshwork (the spongy drain tissue).

1. Pilocarpine turns on muscarinic receptors (tiny switches) on the muscle fibers in your iris.
2. The circular sphincter muscle squeezes hard, making your pupil very small (pinpoint, often just 1 or 2 millimeters wide).
3. As the iris bunches up toward the center, it pulls away from the drainage angle at the edge of the front chamber.
4. The angle opens, and the trabecular meshwork (the spongy drain at the angle) is no longer blocked by the root of the iris.
5. Fluid can now flow out of your eye through the drain again, and pressure falls over 15 to 30 minutes.

SAFETY WATCH

Causes dim, blurry vision and makes it very hard to see in the dark because your pupil is tiny and cannot open to let in light. · Can cause a dull, aching pain across your forehead or behind your eyebrow (brow ache) because the eye muscles are working hard and the ciliary muscle is tight. · Young people (under 40) may notice their distance vision gets blurry because the ciliary muscle tightens too much, pulling on the lens. · Should be used carefully if you have a history of retinal detachment (the light-sensing layer at the back of your eye peeling away) — the pulling on eye structures can very rarely trigger it. · Do NOT drive at night or in dim light while using pilocarpine — your pupil will not open to let in enough light, and you will not see well. · Rarely can cause more sweating, stomach cramps, or diarrhea because it turns on the same receptors in other parts of your body (your gut and sweat glands).

Mannitol (IV infusion) · Osmotic diuretic (a drug that pulls water out of tissues by osmosis and makes you pee it out)

Mannitol is a sugar solution given through an IV. It stays in your bloodstream and acts like a sponge, pulling water out of your eye (and your brain) into your blood by osmosis (water always moves toward the side with more dissolved particles). Your kidneys then pee out that extra water. Your eye shrinks slightly, and pressure drops fast — often within 30 minutes. Mannitol is saved for severe, urgent cases when pressure is dangerously high (often above 50 mmHg) and other drugs are not enough.

1. Mannitol (usually 1 to 2 grams per kilogram of your body weight, as a 20% solution) is infused into a vein over 30 to 45 minutes.
2. It circles in your blood but cannot cross into your eye or brain easily, so it stays in your bloodstream.
3. Because mannitol is very concentrated in the blood, water is pulled from your eye and brain tissues into the bloodstream by osmosis.
4. Your eye deflates slightly as water leaves, and pressure drops fast — often by 10 to 20 mmHg or more within 30 to 60 minutes.
5. Your kidneys filter out the mannitol and the extra water, so you pee a large volume (often a liter or more) within the next hour.

SAFETY WATCH

Must be given in a hospital or emergency room with an IV line and close monitoring. · You will pee a lot and suddenly — a urinary catheter (a tube into your bladder) is often placed to measure how much you pee. · Can cause dehydration or electrolyte imbalances (salts in your blood like sodium and potassium getting out of balance) if fluid is not replaced carefully. · Should NOT be used if you have severe heart failure (the sudden shift of a liter or more of fluid into your bloodstream can overload your heart and cause pulmonary edema (fluid flooding the lungs), fluid in your lungs) or severe kidney failure (you cannot pee out the mannitol, and it builds up). · Rarely, if the concentration is too high or the infusion too fast, mannitol can crystallize in the IV tubing or in your kidneys, causing kidney damage (acute tubular tissue death (necrosis)). · Can cause a sudden headache or nausea as fluid shifts in your brain. · Check kidney function and electrolytes before and after giving mannitol.

Apraclonidine (eye drops) · Alpha-2 agonist (similar to brimonidine, but used more for short-term jobs)

Apraclonidine lowers eye pressure by cutting down how much fluid the ciliary body makes and by helping drainage through the uveoscleral (side) pathway. It works very much like brimonidine. Doctors often use it for short-term jobs, such as stopping pressure spikes right after laser procedures (like laser iridotomy) or during the acute attack as an extra tool.

1. Apraclonidine turns on alpha-2 receptors on the ciliary body cells, slowing down fluid production.
2. It also opens up flow through the uveoscleral route by relaxing tissues around that pathway.
3. Pressure drops within one to two hours and lasts several hours (usually three to five hours).

SAFETY WATCH

Can cause redness in the white of your eye, dry mouth, and tiredness (similar to brimonidine). · High rate of allergy with long-term use (red, swollen, itchy eyelids develop after weeks to months), so it is usually used only short-term (a few days to a week). · Should NOT be used in babies or very young children (same risk as brimonidine). · Can raise your upper eyelid slightly (making your eye look a bit more open and wide) — this is called eyelid retraction and goes away when you stop the drop. · Not usually used as a daily long-term medicine; brimonidine or other drugs are better for that.

Glycerin (oral liquid) · Osmotic agent (oral, meaning you drink it instead of getting an IV)

Glycerin is a thick, sweet, syrupy liquid you drink. It is absorbed into your bloodstream from your stomach and stays there. Just like mannitol, it pulls water out of your eye by osmosis (water moves toward the glycerin in your blood). Your eye shrinks, and pressure drops.

Glycerin is sometimes used in the emergency room when IV access is hard to get or mannitol is not available, but it is not used as much anymore because mannitol IV works faster and more reliably.

1. You drink the glycerin solution (usually about 1 to 1.5 grams per kilogram of your body weight, often mixed with juice or lemon to hide the very sweet taste).
2. It is absorbed into your bloodstream from your stomach and intestines over 15 to 30 minutes.
3. Glycerin raises the osmotic pull of your blood, drawing water out of your eye and into your bloodstream.
4. Pressure drops within 30 to 60 minutes, often by 10 to 20 mmHg or more.
5. Your kidneys filter it out (glycerin is broken down into sugar and used for energy or peed out), and you will pee more.

SAFETY WATCH

Tastes very sweet and thick, which can be hard to swallow and often causes nausea or vomiting. · Can raise blood sugar (because glycerin is broken down into glucose), so use carefully in people with diabetes — check blood sugar after giving it. · Can cause headache, thirst, or a feeling of fullness in your stomach. · Not used much anymore in the United States — mannitol IV is preferred in most hospitals because it works faster, is easier to control, and does not cause nausea as often.

Latanoprost (or other prostaglandin analogs: travoprost, bimatoprost, tafluprost) · Prostaglandin analog (a drug that mimics a natural signal in your body called prostaglandin F2-alpha)

Latanoprost copies a natural signal in your body called prostaglandin F2-alpha. It opens up the uveoscleral pathway (the side drain through the wall of your eye, running between muscle bundles in the ciliary body and through the sclera, the white outer coat). With that side drain more open, more fluid flows out, and pressure drops. Latanoprost is NOT used during the acute attack, but doctors often start it after the attack is controlled (after laser iridotomy) to keep pressure low long-term and protect your optic nerve.

1. Latanoprost (a prodrug, an inactive form) is absorbed through your cornea into the front of your eye.

2. Enzymes inside your eye (esterases) convert it into the active form, latanoprost acid.
3. Latanoprost acid binds to prostaglandin F (FP) receptors on cells lining the uveoscleral pathway.
4. This triggers remodeling of the extracellular matrix (the scaffolding of proteins and fibers between cells), loosening the spaces and widening the channels.
5. More fluid flows out through the uveoscleral route, and pressure drops steadily over 12 to 24 hours, reaching its lowest point about 12 hours after you put the drop in.

SAFETY WATCH

Can slowly darken the colored part of your eye (the iris) over months to years — brown pigment (melanin) increases, especially if you have green, hazel, or blue-green eyes. This change is usually permanent and may not reverse if you stop the drop. · Can make your eyelashes grow longer, thicker, and darker (this is why a version of this drug, bimatoprost, is sold as a cosmetic under the brand name Latisse). · May cause mild redness in the white of your eye or a feeling like something is in your eye. · Use only once a day, at bedtime, because it works best with that timing (the peak effect is 12 hours later, during the day when you are awake and pressure tends to be higher). · Can very rarely darken the skin around your eye (periorbital hyperpigmentation) if the drop runs onto your eyelid or cheek — wash your face and hands after putting it in. · Rarely can reactivate herpes simplex keratitis (a viral infection of the cornea) if you have a history of it. · Safe to use long-term; very effective at lowering pressure (often by 25 to 30 percent).

Red flags

- Sudden severe eye pain with vision loss (permanent blindness can happen within hours if pressure is not brought down)
- Headache with nausea and vomiting along with eye redness (not just a stomach bug—this triad means possible angle closure)
- Eye feels rock-hard to gentle touch through the eyelid (sign of dangerously high pressure)
- Pupil is mid-sized, oval, and does not shrink in bright light (shows the iris sphincter muscle is damaged by pressure)
- Symptoms in someone over 60, far-sighted, or of East Asian or Inuit descent (these groups have narrower drainage angles and higher risk)

Nursing priorities

1. Get the patient to an eye doctor (ophthalmologist) immediately, or call for emergency help if one is not available right away. — The pressure inside the eye is crushing the nerve that sends signals to the brain. If the pressure stays high for more than a few hours, the nerve dies permanently and the person goes blind in that eye. This is a true eye emergency, just like a heart attack is a heart emergency. Every minute counts.
2. Keep the patient calm, in a comfortable position (sitting up is often easier), and dim the lights in the room. — Bright lights make the pupil shrink, which sounds good, but the sudden change can sometimes make the blockage worse at first. Dim lights are gentler. Sitting up can also help fluid drain a bit better. Staying calm keeps blood pressure steady, which helps prevent the eye pressure from climbing even higher.
3. Do not give the patient anything to eat or drink until the doctor says it is okay. — The patient might need emergency surgery or a laser procedure to open the drain and save the eye. If there is food or liquid in the stomach, anesthesia (medicine to make them sleep or numb) is more dangerous because the person could vomit and breathe it into the lungs. Keeping the stomach empty keeps them safe if they need a procedure quickly.
4. Give prescribed eye drops or medicine exactly as the doctor orders, and watch for the patient to urinate after certain medicines. — The doctor will use special eye drops to make the pupil smaller (so the iris moves away from the drain) and other drops or pills to pull fluid out of the eye fast. Some pills (like acetazolamide or mannitol) make the kidneys work harder and push out a lot of urine. We watch to make sure the patient is urinating, which tells us the medicine is working and fluid is leaving the body.
5. Check and write down the patient's pain level, vision changes, and any nausea every 15 to 30 minutes. — These signs tell us if the pressure is going down or if it is still dangerously high. If pain gets worse, vision gets darker, or nausea increases, the treatment is not working fast enough and the doctor needs to know immediately so they can try something stronger or do surgery right away.
6. Teach the patient that the other eye is also at risk and needs to be checked and possibly treated to prevent the same emergency. — If one eye has the shape and anatomy that caused this blockage, the other eye almost always has the same shape. Doctors often do a small laser procedure on the healthy eye as prevention, making a tiny hole in the iris so fluid has an escape route. Knowing this helps the patient protect their vision in both eyes for life.

Sources

- American Academy of Ophthalmology Preferred Practice Pattern
- European Glaucoma Society

- National Eye Institute
- UpToDate

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