

Benign Prostatic Hyperplasia

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THE STORY IN ONE SENTENCE

The prostate gland slowly grows larger with age and squeezes the urethra (the tube that carries urine out), so the bladder must push harder to empty.

“Traffic jam at the exit—bladder's fine, the road out is narrow.”

Normal anatomy — the body's job

The bladder is a stretchy, balloon-like bag that stores urine. When it is full, the detrusor muscle (the smooth muscle in the bladder wall) squeezes and pushes urine through the urethra—the tube that carries urine out of your body. The prostate gland is a small, walnut-shaped organ that wraps around the first part of the urethra, right where it exits the bladder.

BODY'S JOB

Store urine until you decide to go, then squeeze it out through a wide-open urethra in one smooth, strong stream.

1. Kidneys — Filter your blood nonstop and make urine—about 1 to 2 liters every day.
2. Ureters — Two thin tubes that carry urine down from each kidney to the bladder.
3. Bladder — Stretchy storage sac that holds 300 to 500 mL of urine (about a can of soda) until you are ready to go.
4. Bladder neck — The smooth-muscle ring at the bottom of the bladder that opens to let urine out.
5. Prostate — Walnut-sized gland wrapped around the first part of the urethra; adds fluid to semen but can squeeze the urethra if it grows.
6. Urethra — The tube that carries urine (and semen) out through the penis.
7. Outside — Smooth, strong stream—bladder empty, job done.

What breaks

The prostate slowly grows larger over years, especially in the transition zone (the inner part of the gland closest to the urethra). Because the prostate wraps around the urethra like a donut, the growing tissue squeezes inward and narrows the tube. At the same time, smooth muscle inside the prostate and bladder neck becomes tighter because of increased alpha-1 adrenergic tone (the 'fight-or-flight' chemical signal that tightens muscle), adding even more squeeze. So the urethra gets narrower from two directions—extra tissue pushing in and extra muscle tension clamping down.

CLINICAL TERM

Progressive benign prostatic hyperplasia (BPH) with enlargement of the transition zone, plus increased alpha-1 adrenergic tone at the bladder neck and prostatic urethra, resulting in bladder outlet obstruction.

How the body compensates

The bladder tries to push harder against the narrowed urethra. Over months and years, the detrusor muscle thickens (hypertrophy) like a bicep that lifts weights every day. Eventually that thickened muscle becomes overactive—twitchy and squeezing at the wrong times—and the bladder may not empty completely, leaving post-void residual (PVR) urine behind.

1. Detrusor muscle hypertrophy (bladder wall thickens) to generate higher pressure against outlet resistance.
2. The thickened, irritable detrusor becomes overactive and contracts on its own, even when the bladder is not full → urgency (sudden need to go), frequency (going many times a day), and waking at night to urinate (nocturia) (waking at night to urinate).
3. Incomplete bladder emptying → post-void residual urine stays behind after each trip to the bathroom.
4. Stagnant residual urine becomes a growth medium for bacteria → urinary tract infections (UTIs) and bladder stones (crystals that form when minerals sit too long) can develop.
5. Chronic high bladder pressure transmits backward through the ureters → hydronephrosis (swollen kidneys) and obstructive nephropathy (kidney damage from blocked urine flow).
6. Very late stage: the detrusor decompensates (loses contractile strength, like an overstretched rubber band) → chronic urinary retention, overflow incontinence (dribbling because the bladder is always full), and irreversible kidney injury.

Domino effect

1. Prostate tissue enlarges, especially the transition zone around the urethra. — Age-related hormonal changes—especially higher levels of dihydrotestosterone (DHT, a stronger form of testosterone)—drive benign (non-cancer) growth of prostate cells.
2. Urethral lumen (the open space inside the tube) narrows. — The enlarged gland compresses the urethra from all sides, like a tightening donut squeezing the hole in the middle.
3. Bladder outlet obstruction increases; bladder must generate higher pressure to void. — More resistance in the urethra means the detrusor muscle must work harder to push urine through the narrow spot.
4. Detrusor hypertrophy and overactivity develop. — Chronic workload causes the bladder muscle to thicken (like a gym muscle) and become irritable, firing involuntary squeezes even when you are not ready to go.
5. Lower urinary tract symptoms (LUTS) appear: weak stream, hesitancy (delay in starting), intermittency (stream stops and starts), urgency, frequency, waking at night to urinate (nocturia). — Obstructive symptoms (weak stream, hesitancy, intermittency) come from the narrow urethra; irritative symptoms (urgency, frequency, waking at night to urinate (nocturia)) come from the overactive, thickened bladder.
6. Incomplete emptying → rising post-void residual volume (urine left behind after voiding). — The detrusor cannot overcome outlet resistance completely, so urine is left behind in the bladder after each void.
7. Complications: recurrent UTIs, bladder stones, hydronephrosis, acute urinary retention (sudden complete blockage). — Stagnant urine sitting in the bladder grows bacteria (UTIs) and crystallizes into stones; chronic high pressure backs urine up into the kidneys (hydronephrosis); sudden complete blockage causes painful retention and may require emergency catheter placement.

Symptoms · what the patient feels, and why

- Weak urinary stream: The prostate wraps around the urethra (the tube that carries urine out of the body). When the prostate grows bigger, it squeezes the urethra like a hand squeezing a garden hose—the pipe gets narrower, so urine comes out slower even though the bladder pushes with normal force.
- Hesitancy (trouble starting to pee): The bladder is a muscle bag that squeezes to push urine out. Because the enlarged prostate has made the exit narrow, the bladder has to build up extra pressure—like winding up before a big throw—before it can force the first drops through the tight opening.

- Intermittent stream (stop-and-go flow): The detrusor (the bladder's squeezing muscle) gets tired while pushing against the tight squeeze. It pauses to rest, then squeezes again. This happens over and over because one long push isn't strong enough to empty through the narrow opening.
- Feeling that the bladder isn't empty after peeing: Even after the bladder squeezes as hard as it can, some urine is left behind (post-void residual) because the narrow exit makes it impossible to push everything out. That leftover urine sits at the bottom like water stuck in a kinked hose.
- Urinary frequency (peeing more than 8 times a day): Because the bladder never gets completely empty, it fills up again faster. The bladder starts to feel full when it is only half-filled with new urine plus the leftover from last time—so you have to go more often to stay comfortable.
- Waking at night to urinate (Nocturia) (waking up 2 or more times at night to pee): The detrusor (the bladder's muscle wall) becomes irritable and overactive from working so hard. At night it squeezes on its own without warning, and because leftover urine takes up space, even a small amount of new urine triggers the brain's 'I need to go' signal and wakes you up.
- Urgency (sudden, strong need to pee that is hard to hold): The overworked detrusor muscle starts to contract by itself—without you deciding to—and sends a loud 'go NOW' signal to your brain even when the bladder is not truly full. This is called detrusor overactivity.
- Blood in the urine (Hematuria) (blood you can see in the urine, making it pink or red): The enlarged prostate grows new, fragile blood vessels on its surface. Straining to pee or inflammation inside the prostate can break these tiny vessels, letting blood leak into the urine like a scratch that bleeds.
- Acute urinary retention (sudden complete inability to pee, even though the bladder is painfully full): The prostate swells enough—or the detrusor muscle gives out—so that the exit is completely blocked or the bladder cannot squeeze at all. No urine can get through, and the bladder stretches like an overinflated balloon, causing severe pain low in the belly.
- Overflow incontinence (constant dribbling of small amounts of urine without control): Chronic blockage fills the bladder so full that pressure inside rises higher than the internal urethral sphincter (the valve muscle that holds urine in) can hold. Urine leaks around the blockage drop by drop, like water spilling over the top of a dam.

Focused exam

- Palpable distended bladder above the pubic bone: The bladder is stretched tight with urine that cannot escape—a sign of acute urinary retention (sudden total blockage). Without immediate relief by catheter, back-pressure can climb up the ureters (the tubes from the kidneys) and damage the kidneys within hours to days.

- Digital rectal exam: smooth, symmetrically enlarged prostate without hard lumps or asymmetry: A smooth, rubbery, symmetrical prostate is the hallmark of benign prostatic hyperplasia (BPH, non-cancerous growth of the prostate). Hard nodules, one-sided enlargement, or a rock-hard gland raise suspicion for prostate cancer and require a biopsy to check for malignant cells.
- Post-void residual volume greater than 200 mL on bedside bladder ultrasound: More than 200 milliliters of urine sitting in the bladder after the patient tries to empty it confirms the bladder cannot overcome the obstruction caused by the enlarged prostate (AUA guidelines define abnormal incomplete emptying as PVR > 200 mL; 50–100 mL is borderline, and > 300 mL signals high risk of kidney damage).
- Weak, intermittent urinary stream with hesitancy (long delay before urine starts to flow): These obstructive symptoms occur because the urethra (the tube that carries urine out of the body) is squeezed by the overgrown prostate. The bladder must generate much higher pressure to push urine through the narrow channel, eventually exhausting the bladder muscle and leaving urine behind.
- Frequent nighttime urination (nocturia, typically 3 or more trips per night) and sudden strong urges to urinate: Chronic obstruction causes the bladder wall to thicken and become overactive (detrusor overactivity, like a muscle that twitches from overwork), triggering frequent, urgent signals even when the bladder is not truly full. Waking at night to urinate (Nocturia) also predicts higher risk of falls and fractures in older men.

Diagnostics · every test answers a question

- Post-void residual (PVR) volume by bladder ultrasound or catheter measurement → How much urine is trapped in the bladder after the patient tries to empty it completely?
- Urinalysis with microscopy → Is there infection, blood, glucose, or protein in the urine that could explain symptoms or complicate BPH?
- Serum prostate-specific antigen (PSA) → How large is the prostate likely to be, and what is the risk of prostate cancer?
- Serum creatinine and estimated glomerular filtration rate (eGFR) → Has chronic bladder outlet obstruction caused back-pressure damage to the kidneys?
- Uroflowmetry (measures the speed and pattern of urine flow during voluntary voiding) → How severely is urine flow restricted by the obstruction?
- AUA Symptom Index (AUASI) or International Prostate Symptom Score (IPSS) questionnaire → How much do the symptoms bother the patient, and how severe is the functional impact?

Treatment logic

- Watchful waiting with behavioral modifications: timed voiding every 2–3 hours during the day, limit fluids after 6 PM, reduce caffeine and alcohol, practice double voiding (urinate, wait 30 seconds, try again) → Reduces symptom bother and improves quality of life in men with mild symptoms (AUASI score < 8) without side effects or cost. Timed voiding prevents the bladder from overfilling, and fluid restriction reduces nighttime trips.
- Alpha-1 adrenergic blockers: tamsulosin 0.4 mg daily, alfuzosin 10 mg daily, silodosin 8 mg twice daily, doxazosin (titrated to 4–8 mg daily), or terazosin (titrated to 5–10 mg daily) → Relaxes smooth muscle in the prostate and bladder neck within 48 hours to 2 weeks, opening the urethral channel and improving urine flow rate and symptom scores by 30–40%. Works regardless of prostate size (AUA/EAU BPH guidelines).
- 5-alpha-reductase inhibitors (5-ARIs): finasteride 5 mg daily or dutasteride 0.5 mg daily → Blocks the enzyme that converts testosterone to dihydrotestosterone (DHT, the hormone that drives prostate cell growth), shrinking prostate volume by 20–30% over 6–12 months. Reduces risk of acute urinary retention by 50% and reduces risk of needing surgery by 50% over 4 years (MTOPS, CombAT trials). Most effective in men with prostate volume > 30–40 grams (roughly > 40 mL by ultrasound).
- Combination therapy: alpha-blocker (e.g., tamsulosin) plus 5-alpha-reductase inhibitor (finasteride or dutasteride) → Provides immediate symptom relief from the alpha-blocker while the 5-ARI shrinks the prostate over the next 6–12 months. Combination therapy reduces long-term clinical progression (worsening symptoms, acute retention, need for surgery) more than either drug alone (CombAT, MTOPS trials). Recommended for men with moderate-to-severe symptoms and prostate volume > 40 grams.
- Phosphodiesterase-5 (PDE5) inhibitor: tadalafil 5 mg once daily → Relaxes smooth muscle in the prostate, bladder, and urethra through increased cyclic GMP (a different mechanism from alpha-blockers), improving lower urinary tract symptom scores by 20–30%. Also treats erectile dysfunction if present. FDA-approved for both BPH and ED (2011).
- Anticholinergic (e.g., oxybutynin, tolterodine) or beta-3 agonist (mirabegron) added to alpha-blocker in men with persistent urgency and frequency despite obstruction relief → Calms overactive bladder muscle contractions (detrusor overactivity), reducing urgency, frequency, and waking at night to urinate (nocturia). Beta-3 agonists relax the bladder during filling without impairing voiding (fewer side effects than anticholinergics).

- Minimally invasive or surgical procedures: transurethral resection of the prostate (TURP), holmium laser enucleation of the prostate (HoLEP), prostatic urethral lift (UroLift), water vapor thermal therapy (Rezūm), or prostatic artery embolization (PAE) → Physically removes, vaporizes, compresses, or devascularizes (cuts off blood supply to) obstructing prostate tissue, opening the urethral channel. TURP and HoLEP are gold standards, improving peak flow rates by 100–200% and durably relieving obstruction with low reoperation rates (< 10% at 5 years; AUA BPH surgical guidelines). Reserved for men with moderate-to-severe symptoms refractory to medication, recurrent urinary retention, recurrent UTIs, bladder stones, or obstructive kidney injury.

Medications

Tamsulosin (Flomax) · Alpha-1A selective blocker

Blocks tiny switches called alpha-1A receptors that tell the muscle around the prostate and bladder neck to squeeze tight. When those switches are blocked, the muscle relaxes and the tube that carries urine out (the urethra) opens wider, so urine flows more easily.

1. Alpha-1A receptors sit on the surface of smooth muscle cells around the bladder neck and the part of the urethra inside the prostate.
2. A chemical signal called norepinephrine (made by nerves) normally locks onto these receptors and tells the muscle to squeeze tight.
3. When the prostate is enlarged (benign prostatic hyperplasia), that constant squeeze makes it hard for urine to get through.
4. Tamsulosin blocks the alpha-1A receptors so norepinephrine cannot attach.
5. The smooth muscle relaxes and loses its tight squeeze.
6. The urethra opens wider and urine flows out with much less pushing and straining.

SAFETY WATCH

Orthostatic low blood pressure (hypotension) (blood pressure drops when you stand up, making you dizzy or lightheaded). This happens most often after the first dose because alpha-1 receptors also sit on blood vessel walls. · Retrograde ejaculation (during orgasm, semen flows backward into the bladder instead of out through the penis). This is not dangerous but can affect fertility and feels different. · Intraoperative floppy iris syndrome (during cataract or other eye surgery, the iris does not stay firm and can wave around, making surgery harder). Always tell your eye surgeon you take or have taken tamsulosin.

Finasteride (Proscar) · 5-alpha-reductase inhibitor

Blocks an enzyme called 5-alpha-reductase that turns regular testosterone into a supercharged version called DHT (dihydrotestosterone). DHT is the main fuel that makes prostate cells grow. When you block the enzyme, DHT drops by about 70–90% and the prostate slowly shrinks by 20–30% over 6 to 12 months.

1. Prostate cells contain an enzyme called 5-alpha-reductase type II.
2. This enzyme grabs testosterone (the male hormone made by the testicles) and converts it into dihydrotestosterone (DHT), which is five times more powerful at making prostate cells grow.
3. DHT attaches to androgen receptors (special locks inside the nucleus of prostate cells) and tells the genes to make the cell grow and divide.
4. Finasteride blocks 5-alpha-reductase so it cannot make DHT.
5. DHT levels inside the prostate drop by 70–90%.
6. Without the growth signal, prostate cells slowly shrink. Over 6 to 12 months, the whole prostate shrinks by about 20–30%, which takes pressure off the urethra and lets urine flow better.

SAFETY WATCH

Lowers the blood test for prostate cancer (PSA, prostate-specific antigen) by about 50% after 6 months. Doctors know to multiply your PSA result by 2 when checking for cancer. Tell every provider you take finasteride so they read the number correctly. · Sexual side effects: lower sex drive (libido), trouble getting or keeping an erection (erectile dysfunction), and less fluid when you ejaculate. These happen in about 5–10% of men and usually get better if you stop the drug, but in rare cases they may last. · Teratogenic (causes severe birth defects in a male fetus). Pregnant people or people planning pregnancy must NEVER touch crushed or broken finasteride tablets because the drug can absorb through the skin and harm the baby.

Dutasteride (Avodart) · Dual 5-alpha-reductase inhibitor (type I and type II)

Blocks BOTH forms of the enzyme 5-alpha-reductase (type I and type II) that turn testosterone into DHT (dihydrotestosterone, the hormone that fuels prostate growth). This drops DHT even more than finasteride—by about 90–95%—so the prostate shrinks more over 6 to 12 months.

1. Prostate cells use two versions of the enzyme 5-alpha-reductase (type I and type II) to convert testosterone into DHT.
2. DHT is the main signal that tells prostate cells to grow and multiply.
3. Dutasteride blocks both type I and type II enzymes at the same time.

4. DHT levels inside the prostate drop by about 90–95% (more than finasteride, which only blocks type II).
5. Over 6 to 12 months, the prostate shrinks by roughly 25–30%, taking pressure off the urethra so urine can flow more easily.

SAFETY WATCH

Lowers PSA (prostate-specific antigen) by about 50% after 6 months. Doctors multiply your PSA by 2 when screening for prostate cancer. Always tell every provider you take dutasteride. · Sexual side effects: lower sex drive (libido), erectile dysfunction (trouble getting or keeping an erection), and reduced semen volume. These happen in about 5–10% of men. · Teratogenic (causes severe birth defects in a male fetus). Pregnant people must NEVER handle crushed or broken tablets because the drug absorbs through the skin and can harm the baby. · Long half-life (stays in the body for weeks after stopping). It takes about 3–6 months to fully wash out of your system.

Alfuzosin (Uroxatral) · Non-selective alpha-1 blocker (targets alpha-1A, alpha-1B, and alpha-1D)

Blocks all three types of alpha-1 receptors (the 'squeeze' switches) on smooth muscle at the prostate, bladder neck, and blood vessels. This relaxes the muscle around the urethra so urine flows more easily, but it also relaxes blood vessel walls so blood pressure can drop.

1. Alpha-1 receptors sit on smooth muscle cells around the bladder neck, prostate, and inside blood vessel walls.
2. Norepinephrine (a nerve signal) binds to these receptors and tells the muscle to squeeze.
3. Alfuzosin blocks all three subtypes of alpha-1 receptors (1A, 1B, and 1D).
4. The smooth muscle around the urethra relaxes so urine can flow out more easily.
5. Because alfuzosin also blocks receptors in blood vessels, those relax too and blood pressure can drop.

SAFETY WATCH

Orthostatic low blood pressure (hypotension) (dizziness or lightheadedness when standing up) because it relaxes blood vessels. This is more common than with tamsulosin because alfuzosin is less selective. · Must avoid in moderate or severe liver disease (Child-Pugh B or C) because the drug is broken down by the liver and levels can build up. · Avoid with strong CYP3A4 inhibitors (like ketoconazole, itraconazole, ritonavir) because they raise alfuzosin levels and increase the risk of low blood pressure and fainting. · QT prolongation (a change in the heart's electrical rhythm that can rarely lead to dangerous arrhythmias). Avoid in patients already at risk or taking other drugs that prolong the QT interval.

Doxazosin (Cardura) · Non-selective alpha-1 blocker (targets alpha-1A, alpha-1B, and alpha-1D)

Blocks all three types of alpha-1 receptors on smooth muscle at the prostate, bladder neck, and blood vessels. This relaxes the muscle around the urethra so urine flows better, and also relaxes blood vessel walls (which is why it is also used to treat high blood pressure).

1. Alpha-1 receptors sit on smooth muscle cells in the bladder neck, prostate, and blood vessel walls.
2. Norepinephrine (a nerve chemical) binds to these receptors and tells the muscle to squeeze tight.
3. Doxazosin blocks all three subtypes of alpha-1 receptors.
4. The muscle around the urethra relaxes and the tube opens wider so urine flows more easily.
5. At the same time, blood vessels relax so blood pressure drops (useful if you have high blood pressure too).

SAFETY WATCH

Orthostatic low blood pressure (hypotension) (sudden drop in blood pressure when standing up, causing dizziness or fainting), especially after the first dose or when the dose is increased. Always start at a low dose at bedtime. · More blood-pressure effect than tamsulosin because doxazosin is non-selective. Watch closely if you already take blood-pressure medicine. · Intraoperative floppy iris syndrome during cataract surgery. Tell your eye surgeon you take or have taken doxazosin.

Terazosin (Hytrin) · Non-selective alpha-1 blocker (targets alpha-1A, alpha-1B, and alpha-1D)

Blocks all three types of alpha-1 receptors on smooth muscle at the prostate, bladder neck, and blood vessels. This relaxes the muscle around the urethra so urine flows better, and also relaxes blood vessel walls (which is why it is also used to treat high blood pressure).

1. Alpha-1 receptors sit on smooth muscle cells in the bladder neck, prostate, and blood vessel walls.
2. Norepinephrine (a nerve signal) binds to these receptors and keeps the muscle tight.
3. Terazosin blocks all three subtypes of alpha-1 receptors.
4. The muscle around the urethra relaxes and urine flows more easily.
5. At the same time, blood vessels relax and blood pressure drops (helpful if you also have high blood pressure).

SAFETY WATCH

Orthostatic low blood pressure (hypotension) (sudden drop in blood pressure when standing up, causing dizziness or fainting), especially after the first dose or a dose increase. Always start at a low dose at bedtime and increase slowly. · More blood-pressure effect than tamsulosin because terazosin is non-selective. Monitor closely if you already take blood-pressure medicine. · Intraoperative floppy iris syndrome during cataract surgery. Tell your eye surgeon you take or have taken terazosin.

Silodosin (Rapaflo) · Alpha-1A selective blocker

Blocks alpha-1A receptors (the 'squeeze' switches) on smooth muscle at the prostate and bladder neck so the muscle relaxes and the urethra opens wider. It is very selective for alpha-1A so it has less effect on blood vessels and causes less dizziness than non-selective blockers.

1. Alpha-1A receptors sit on smooth muscle cells around the bladder neck and the part of the urethra inside the prostate.
2. Norepinephrine (a nerve signal) binds to these receptors and tells the muscle to squeeze tight.
3. Silodosin selectively blocks alpha-1A receptors so norepinephrine cannot attach.
4. The smooth muscle relaxes and the urethra opens wider.
5. Urine flows out with much less resistance.

SAFETY WATCH

Retrograde ejaculation (semen flows backward into the bladder instead of out) happens in about 20–30% of men taking silodosin—higher than with tamsulosin. This is not dangerous but can affect fertility. · Orthostatic low blood pressure (hypotension) (dizziness when standing up) is LESS common than with non-selective alpha blockers because silodosin has less effect on blood vessels. · Intraoperative floppy iris syndrome during cataract surgery. Tell your eye surgeon you take or have taken silodosin. · Avoid in severe kidney disease (CrCl less than 30 mL per minute) because the drug and its active form build up in the body.

Tadalafil (Cialis, daily low-dose 5 mg) · Phosphodiesterase-5 (PDE5) inhibitor

Blocks the enzyme phosphodiesterase-5 (PDE5) inside smooth muscle cells of the prostate, bladder, and urethra. This lets a messenger molecule called cyclic GMP (cGMP) build up. cGMP relaxes smooth muscle so the bladder neck and prostate loosen and urine flows more

easily. It also improves blood flow to the penis, which is why the same drug treats erectile dysfunction.

1. Nitric oxide (a natural gas signal in the body) triggers smooth muscle cells to make cyclic GMP (cGMP), a molecule that relaxes the muscle.
2. An enzyme called phosphodiesterase-5 (PDE5) normally breaks down cGMP so the muscle can tighten again.
3. Tadalafil blocks PDE5.
4. cGMP levels stay high and the smooth muscle around the prostate, bladder neck, and urethra stays relaxed.
5. The urethra opens wider and urine flows more easily.
6. At the same time, blood vessels in the pelvic area and penis relax, which can improve erections.

SAFETY WATCH

Headache, flushing (red, warm face), and upset stomach are common side effects because PDE5 inhibitors relax blood vessels throughout the body. · NEVER use with nitrates (nitroglycerin, isosorbide) or riociguat because the combination can cause a dangerous drop in blood pressure. · Use caution if you take alpha blockers (like tamsulosin) at the same time because both drugs lower blood pressure. Start tadalafil at the lowest dose and watch for dizziness. · Avoid in severe liver disease (Child-Pugh C) and severe kidney disease (CrCl less than 30 mL per minute) because the drug is not cleared well and levels build up. · Rare but serious: sudden vision loss (non-arteritic anterior blood-starved (ischemic) optic neuropathy, NAION) or sudden hearing loss. Stop the drug and get help right away if this happens.

Red flags

- Acute urinary retention: sudden inability to pee with severe pain low in the belly and a bladder you can feel as a hard bulge above the pubic bone—needs emergency catheter (thin tube) placed through the urethra to drain the bladder right away.
- Repeated bladder infections (cystitis) or fever with pain when peeing—leftover urine becomes a breeding ground for bacteria, and infection can climb to the kidneys (pyelonephritis) or spread into the blood (urosepsis), which is life-threatening.
- Blood in the urine (hematuria) that keeps coming back or does not go away—must check for bladder cancer, kidney stones, and prostate cancer with imaging and cystoscopy (camera inside the bladder).

- Rising creatinine on a blood test or swollen kidneys (hydronephrosis) on ultrasound or CT—means backup pressure from the blocked bladder is traveling up the ureters and damaging the kidneys (obstructive nephropathy), which can lead to kidney failure.
- Hard, lumpy, or uneven prostate felt during digital rectal exam, or PSA (prostate-specific antigen blood test) that jumps up fast or stays very high—raises strong concern for prostate cancer and requires MRI and biopsy.

Nursing priorities

1. Assess bladder fullness and pain BEFORE giving antihistamines or decongestants — These drugs can trigger sudden retention in a patient who already has a tight pipe.
2. Check post-void residual after any near-retention event — Confirms the bladder is emptying enough to go home safely.
3. Teach fluid and caffeine timing (fluid earlier, caffeine off after noon) — Makes nighttime urgency and sleep interruption more livable.
4. Teach first-dose orthostatic caution with alpha-blockers — Prevents falls, especially in older adults.
5. Report new fever, flank pain, or blood in pee immediately — These can mean an obstructive infection or kidney injury.

Sources

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