

Diabetes Insipidus

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THE STORY IN ONE SENTENCE

The body cannot hold onto water because either the brain stops making ADH (the hormone that tells kidneys to keep water in) or the kidneys ignore the ADH signal.

“Diabetes insipidus means the kidneys dump huge amounts of water every day, leaving the blood salty and the person desperately thirsty all the time.”

Normal anatomy — the body's job

The hypothalamus (a control center deep in your brain) makes ADH, stores it in the pituitary gland (a pea-sized gland just below), and releases it into your blood when your blood starts to get too salty or thick. ADH travels to the kidneys and tells them to pull water back into your body.

BODY'S JOB

ADH acts like a dimmer switch for your kidneys. When your blood gets even 1 or 2 percent saltier than normal, special sensors in your hypothalamus (the brain's main control room for water, temperature, and hunger) notice and tell the pituitary gland to release ADH. ADH travels to tiny tubes in the kidneys (the collecting ducts, the final hallway where pee is made) and opens up water channels (called aquaporin-2, tiny gates in the tube walls). Water then flows back into your bloodstream instead of leaving as pee. This keeps your blood at the perfect thickness, protects your cells from shrinking or swelling, and keeps your brain safe. At the same time, thirst kicks in so you drink a little more. Together, ADH and thirst keep your body's salt and water balanced every single day.

1. Blood gets a little salty (maybe you sweat, or you have not had water for a while).
2. Sensors (osmoreceptors, salt-level detectors) in the hypothalamus (deep in your brain) notice the blood is thick.
3. The hypothalamus tells the pituitary gland (a pea-sized gland right below it) to release ADH into the blood.

4. ADH flows through your bloodstream to the kidneys.
5. In the kidneys, ADH unlocks water channels (called aquaporin-2, tiny gates) in the walls of the collecting ducts (the final tube where pee is made).
6. Water rushes back into the bloodstream through those open channels, so less water leaves as pee.
7. Your pee becomes darker and more concentrated, and your blood stays at the right thickness.
8. At the same time, your hypothalamus turns on thirst so you drink a little water, and balance is restored.

What breaks

Either the brain (hypothalamus or pituitary gland) gets damaged and stops making ADH, or the kidneys get damaged and cannot respond to ADH anymore. Without that signal, the kidneys let water pour out all day long.

CLINICAL TERM

Central diabetes insipidus: the hypothalamus or pituitary gland is damaged (by surgery, tumor, trauma, infection, or autoimmune attack) and stops making or releasing ADH.

Nephrogenic diabetes insipidus: the kidneys are damaged (by drugs like lithium, genetic mutations in the ADH receptor or aquaporin-2 water channel, chronic kidney disease, high blood calcium, or low blood potassium) and cannot respond to ADH even when it is present. Gestational diabetes insipidus: the placenta makes an enzyme (vasopressinase) that breaks down ADH during pregnancy. Primary excessive thirst (polydipsia): the thirst center is broken or the person drinks huge amounts by habit, suppressing ADH and causing large urine output.

How the body compensates

As long as the person can drink freely and their thirst center works, the body keeps up by making them drink huge amounts all day and night. But if they cannot drink, or if the thirst center fails, blood salt climbs fast and the brain is in danger.

1. {'what': 'The kidneys pour out water every hour, so blood salt (sodium) starts to rise.', 'why': 'Without ADH, the collecting ducts stay closed to water reabsorption, so huge volumes of dilute pee (3 to 20 liters per day, sometimes more) leave the body. Sodium and other particles stay behind in the blood, making it saltier and thicker.'}

2. {'what': 'The hypothalamus senses blood getting salty and turns on intense thirst.', 'why': 'The hypothalamus has osmoreceptors (salt sensors) that detect even a 1 to 2 percent rise in blood thickness (osmolality, normally 275 to 295 mOsm/kg). Thirst is the body's emergency alarm to pull in more water and dilute the blood back down.'}
3. {'what': 'The person drinks constantly (polydipsia) and pees constantly (polyuria), staying roughly balanced.', 'why': 'As long as they can drink freely, water in equals water out, and blood sodium stays near normal (135 to 145 mEq/L). But they are exhausted, cannot sleep through the night (they wake up every 1 to 2 hours to pee and drink), and must live near bathrooms and water.'}
4. {'what': 'If the person cannot drink (surgery, unconscious, no water access, broken thirst center, infant who cannot ask for water), blood salt shoots up fast.', 'why': 'Water keeps pouring out as pee, but no water is coming in. Blood sodium climbs above 145 (hypernatremia), then 150, then higher. Cells all over the body shrink as water is pulled out of them to try to dilute the blood.'}
5. {'what': 'High blood salt shrinks brain cells and causes confusion, irritability, muscle twitching, seizures, coma, and brain bleeding.', 'why': 'Brain cells shrink when the blood around them gets too salty. Shrinking pulls tiny blood vessels (bridging veins) apart and can cause bleeding inside the skull (subdural or subarachnoid bleeding (hemorrhage)). Neurons misfire when they shrink, leading to seizures.'}
6. {'what': 'The danger zone: correcting blood salt too fast after it has been high for a while can cause brain swelling (cerebral swelling (edema)) and death.', 'why': 'If you drop the blood sodium too quickly, water rushes into brain cells faster than they can adjust, and the brain swells inside the rigid skull. The swelling can cause herniation (brain tissue squeezes through openings in the skull) and death. Go slow: lower sodium by no more than 10 to 12 mEq/L in 24 hours, and no more than 18 mEq/L in 48 hours (Adrogué-Madias formula).'}

Domino effect

1. The hypothalamus or pituitary gland stops making ADH, or the kidneys stop responding to it. — This is the single broken piece. Without ADH telling the kidneys to hold onto water, the system that keeps blood thickness steady is gone. In central DI, the hypothalamus (the ADH factory) or pituitary (the storage and release site) is damaged. In nephrogenic DI, the kidneys' ADH receptors (AVPR2, the docking site for ADH on collecting duct cells) or water channels (aquaporin-2, the gates that let water back into the blood) are broken.
2. The kidneys pour out huge amounts of dilute, pale pee all day and night (polyuria: 3 to 20 liters per day, sometimes more). — The collecting ducts stay closed to water because ADH is missing or ignored. Water that should be pulled back into the blood flows straight into the bladder as pee. The pee looks almost like water because it is so dilute (specific gravity less than 1.005, urine osmolality under 200 mOsm/kg, normal is 500 to 800).

3. Blood starts to get salty and thick (rising serum osmolality and sodium). — Water is leaving the body, but sodium and other particles stay behind, so the blood gets more concentrated, like soup left on the stove too long. Serum osmolality rises above 295 mOsm/kg, and sodium climbs above 145 mEq/L if the person cannot drink enough.
4. The hypothalamus senses the salty blood and turns on intense thirst (polydipsia). — The hypothalamus has osmoreceptors (salt sensors) that scream 'drink now' when blood gets even a little too thick. Thirst is the body's emergency backup to replace the lost water. People describe it as an unstoppable urge to drink, and they often prefer ice-cold water.
5. The person drinks huge amounts of water all day, matching what they are peeing out. — As long as they can drink freely, water in equals water out, so blood sodium stays near normal. But they are exhausted from constant drinking and peeing (every 1 to 2 hours, day and night), cannot sleep through the night, and must plan life around bathrooms and water access.
6. If the person cannot drink (unconscious, after surgery, no water nearby, broken thirst center, infant), blood sodium climbs fast (hypernatremia, over 145 mEq/L). — Water keeps pouring out as pee, but no water is coming in. Sodium rises above 145, then 150, then higher (can reach 160 to 180 in severe cases). Cells all over the body shrink as water is pulled out to try to dilute the blood. This is life-threatening.
7. Brain cells shrink, causing confusion, lethargy, irritability, muscle twitching, weakness, seizures, and coma. — When brain cells (neurons and glia, the supporting cells) shrink, they pull away from the skull and can tear tiny blood vessels (bridging veins), causing bleeding inside the skull (subdural or subarachnoid bleeding (hemorrhage)). Neurons misfire when they shrink, leading to seizures. Severe high sodium (hypernatremia) (over 160 mEq/L) carries high risk of permanent brain damage or death.
8. If you correct the high sodium too fast, the brain swells (cerebral swelling (edema)) and herniates, causing death. — After cells have adjusted to high salt by making new particles inside (idiogenic osmoles, emergency molecules the cell builds to pull water back in) to pull water back in, dropping sodium too quickly makes water rush into cells faster than they can get rid of those particles. The brain swells inside the rigid skull, leading to herniation (brain tissue squeezes through openings) and death. Always correct slowly: lower sodium by no more than 10 to 12 mEq/L in 24 hours, and no more than 18 mEq/L in 48 hours. Use the Adrogé-Madias formula to calculate how much free water or desmopressin to give.

Symptoms · what the patient feels, and why

- **Very large amounts of watery pee (polyuria):** Without enough vasopressin (also called ADH, the hormone made by the hypothalamus and stored in the pituitary that tells the kidneys to hold onto water), the kidneys let huge amounts of water escape—often 3 to 20 liters a day, which is like filling a big soda bottle ten times or more. The kidneys simply flush the water out instead of pulling it back into the bloodstream.
- **Pee is very pale or clear:** Because the kidneys are dumping so much water without pulling it back, the urine stays extremely diluted—it looks almost like water because very little waste is concentrated in it. Normally the kidneys concentrate urine to save water, but here that brake is missing.
- **Extreme thirst (polydipsia):** The body loses so much water through urine that the blood becomes more concentrated and salty. Special sensors in the hypothalamus (the brain's control center for thirst and water balance) detect this and fire up an intense thirst signal to make the person drink constantly and try to replace the lost fluid.
- **Getting up many times at night to pee (nocturia):** The kidneys keep dumping water around the clock, so the bladder fills repeatedly even during sleep. This forces the person to wake up multiple times every night to urinate, which wrecks sleep and leaves them exhausted.
- **Dry mouth and dry skin:** With constant water loss, the body becomes mildly dehydrated—tissues have less fluid than they need, so the mouth feels sticky and dry, and the skin loses its usual moisture and bounce.
- **Fatigue and weakness:** Repeated trips to the bathroom, broken sleep night after night, and the body's constant effort to keep up with fluid shifts all drain energy, leaving the person feeling tired, weak, and worn out.
- **High sodium level in the blood (hypernatremia):** When the kidneys lose too much water but keep most of the sodium, the blood becomes saltier than normal—like a pot of soup boiling down so the salt taste gets stronger. This high sodium can damage cells if it climbs too high.
- **Confusion or irritability:** If the blood sodium climbs too high or dehydration worsens, brain cells shrink slightly because water moves out of them to try to balance the salt. This disrupts the normal electrical signals between neurons (the brain's messenger cells), causing mental fog, confusion, or a short temper.
- **Weight loss:** Constant water loss and the body's struggle to keep up with fluid balance can lead to losing weight, especially if the person cannot drink enough to match what they are peeing out. The body is literally losing water weight day after day.
- **Constipation:** When the body is mildly dehydrated, the colon (the last part of the intestine) pulls extra water out of stool to try to save fluid for the bloodstream. This leaves the stool dry and hard, making it difficult to pass.

Focused exam

- Urine output over 3 liters per day in adults (or more than 40 mL per kilogram per day in children), and the urine looks pale or clear like water: When ADH (the hormone made in the brain that tells kidneys to hold onto water) is missing or the kidneys cannot hear its signal, the kidneys dump huge amounts of thin, watery urine. This flood of pale pee is the hallmark of diabetes insipidus and separates it from other causes of peeing often, like a bladder infection or nervousness.
- Extreme thirst (polydipsia) that wakes the patient at night and makes them drink constantly, sometimes 5 to 20 liters a day: The hypothalamus (the brain's control center for thirst and water balance) senses that water is leaving the body too fast and cranks up thirst to try to keep the blood from getting thick. This intense, never-ending thirst is the body's emergency alarm saying 'we are losing water faster than we can replace it.'
- Serum sodium above 145 mEq/L (hypernatremia), especially if the patient cannot reach water or is too young or confused to drink: When water leaves the body but salt stays behind, salt gets concentrated in the blood. High sodium is dangerous because it can shrink brain cells, cause confusion, seizures, and even coma if it climbs too fast or too high.
- Dry mucous membranes (mouth, lips), poor skin turgor (skin tents and returns slowly when pinched), sunken eyes, and decreased tears: These are physical signs that the body is running low on water. In diabetes insipidus, water loss can be so massive that even drinking gallons cannot keep up, and the tissues start to dry out.
- Waking at night to urinate (Nocturia) (waking two or more times per night to urinate) and sometimes bedwetting in children who were previously dry at night: Because the kidneys keep making thin urine around the clock without any off switch, the bladder fills fast even during sleep. Waking up to pee multiple times every night is a strong clue that water loss is not under normal hormonal control.
- Normal blood glucose and no other clear cause of increased urination like diuretic pills, high calcium, or a urinary tract infection: Diabetes insipidus and diabetes mellitus (the sugar kind) both cause lots of urination, but only diabetes mellitus spills glucose into urine and raises blood sugar. A normal glucose tells you the kidneys are dumping water for a different reason—likely a problem with ADH, the water-holding hormone.

Diagnostics · every test answers a question

- Serum sodium and serum osmolality → Is the blood too concentrated because the body is losing water faster than salt?
- Urine osmolality and urine specific gravity → Is the urine abnormally thin and watery even though the blood is concentrated?

- Water deprivation test (done in the hospital with close monitoring) → Can the kidneys concentrate urine when the body is deprived of water, or do they keep making thin urine no matter what?
- Desmopressin (DDAVP) challenge after water deprivation → If we give synthetic ADH, do the kidneys suddenly start concentrating urine, or do they still ignore the signal?
- MRI of the brain (focused on the pituitary gland and hypothalamus) → Is there a tumor, injury, bleed, or swelling in the pituitary gland or hypothalamus that destroyed the cells that make ADH?
- 24-hour urine volume measurement → How much urine is the patient actually making over a full day?
- Serum calcium and serum glucose → Are there other problems (high calcium or high sugar) that could cause the kidneys to dump water?
- Medication review (especially lithium, amphotericin B, demeclocycline, or ifosfamide) → Is the patient taking a drug known to damage the kidney's ability to respond to ADH?

Treatment logic

- Desmopressin (DDAVP) by nasal spray, tablet, or injection → Replaces the missing ADH hormone so the kidneys can finally hold onto water. Urine output drops, urine becomes more concentrated, and serum sodium returns to normal. Thirst disappears because the body is no longer losing gallons of water every day.
- Free access to water and careful matching of fluid intake to urine output → Prevents dangerous dehydration and high sodium by letting the patient drink as much as they lose. Keeps the blood from getting too thick and salty while the root cause is being diagnosed and treated.
- Thiazide diuretic (like hydrochlorothiazide) plus a low-sodium diet for nephrogenic diabetes insipidus → Paradoxically, a diuretic (water pill) reduces urine output by causing mild volume depletion, which makes the kidneys reabsorb more water upstream in the proximal tubule (the first part of the kidney's filter tube) before urine ever reaches the broken collecting duct. A low-salt diet reduces the load of salt the kidneys have to filter, so less water is needed to wash it out.
- Stop lithium or other nephrotoxic drugs immediately if they are the cause → Removes the toxin that damaged the kidney's ability to respond to ADH in the collecting duct. Over weeks to months, the kidneys may slowly regain function and urine output may decrease as the collecting duct cells heal.

- Amiloride (a potassium-sparing diuretic) for lithium-induced nephrogenic diabetes insipidus → Blocks lithium from entering kidney collecting duct cells through the epithelial sodium channel (the same channel ADH uses), protecting the cells from further damage and reducing urine output. Works specifically when lithium is the culprit.
- Frequent serum sodium monitoring and DDAVP dose titration → Prevents overcorrection that can swing sodium too low (hyponatremia, sodium below 135 mEq/L), which causes confusion, seizures, and brain swelling. Keeps sodium in the safe zone (135–145 mEq/L) as treatment takes effect.
- Treat the underlying cause (remove pituitary tumor, stop brain inflammation with steroids, correct high calcium) → Fixes the root problem that destroyed ADH production in the hypothalamus and pituitary or blocked ADH signaling in the kidney. If a tumor is removed or inflammation is controlled, central diabetes insipidus may resolve completely. If high calcium is corrected, nephrogenic DI may improve.

Medications

Desmopressin (DDAVP) · Synthetic vasopressin analog (ADH replacement)

Desmopressin is a lab-made copy of ADH, the hormone that tells your kidneys to hold onto water. In central diabetes insipidus, your brain doesn't make enough ADH, so your kidneys let too much water escape as urine. Desmopressin acts just like the missing hormone—it tells your kidneys to pull water back from the urine, which makes the urine more concentrated and cuts down how much you pee.

1. You take desmopressin by mouth, nasal spray, or injection.
2. It travels through your blood to your kidneys.
3. It locks onto V2 receptors on the cells lining the collecting ducts.
4. This signal tells the cell to move aquaporin-2 channels (tiny water gates) to the inner wall of the duct.
5. Water from the urine flows through these gates back into your bloodstream.
6. Less water leaves your body as urine, so urine volume drops and becomes more concentrated.
7. Your blood stays at a normal water level, and you feel less thirsty.

SAFETY WATCH

Water overload (hyponatremia—low blood sodium): If you drink too much water while on desmopressin, your blood sodium can drop dangerously low. Watch for headache, confusion, nausea, or seizures. Your doctor will check your sodium level regularly. · Dose timing: Take it at the same time each day. Follow the fluid limits your doctor sets, especially at night. · Nasal spray absorption: A cold or stuffy nose can block absorption. Tell your doctor if the medicine stops working during a cold. · Not for nephrogenic DI: Using desmopressin when the kidneys can't respond wastes the medicine and risks water overload. · Infants and young children: Dosing must be very precise. Too much can cause seizures from low sodium.

Hydrochlorothiazide (HCTZ) · Thiazide diuretic (water pill)

In nephrogenic diabetes insipidus, the kidneys ignore ADH, so you pee out huge amounts of dilute urine. Hydrochlorothiazide seems backward—it's a diuretic, which usually makes you pee more—but in nephrogenic DI it actually REDUCES urine volume. It works by making your body mildly low on salt and water. This triggers the kidney to pull more water back earlier in the tubule, before the broken ADH step. This offsets some of the water loss and cuts urine output by 30 to 50 percent.

1. You take hydrochlorothiazide by mouth once or twice a day.
2. It blocks the sodium-chloride cotransporter (NCC) in the distal tubule, so salt and water are lost in the urine.
3. This creates mild volume depletion—your blood volume drops a little.
4. Your body senses this and turns on the renin-angiotensin-aldosterone system (RAAS), which makes the proximal tubule (the first part of the kidney tube) reabsorb more salt and water.
5. Even though the collecting duct (the ADH-sensitive part) is still broken, less fluid reaches it because more was reabsorbed upstream.
6. The net effect is that total urine output goes down.
7. You still pee dilute urine, but less of it overall.

SAFETY WATCH

Low potassium (hypokalemia): Thiazides make you lose potassium in urine. Watch for muscle cramps, weakness, or irregular heartbeat. You may need potassium supplements or to eat potassium-rich foods like bananas, oranges, and potatoes. · Low sodium (hyponatremia): Adding a diuretic can worsen sodium loss. Your doctor will monitor blood sodium closely. · Dehydration: If you get sick with vomiting, diarrhea, or fever and can't drink, call your doctor. Thiazides can tip you into severe dehydration quickly. · Blood sugar: Thiazides can raise blood glucose slightly. If you have diabetes mellitus, you may need closer sugar monitoring. · Gout: Thiazides can trigger gout attacks in susceptible people by raising uric acid. · Allergy: Rare, but sulfa allergy may cross-react. Tell your doctor if you have sulfa allergies.

Amiloride · Potassium-sparing diuretic

Amiloride is used specifically for lithium-induced nephrogenic diabetes insipidus. Lithium, a mood stabilizer, gets into kidney cells through epithelial sodium channels (ENaC—gates for sodium on the cell surface) in the collecting duct. Once inside, lithium blocks the cells' ability to respond to ADH. Amiloride plugs those sodium channels, keeping lithium out of the cells. This protects the kidney's ADH response and reduces urine output. It is often combined with a thiazide like hydrochlorothiazide for better effect.

1. You take amiloride by mouth, usually once or twice a day.
2. It travels to the collecting duct in the kidney.
3. It blocks ENaC channels on the surface of principal cells.
4. Lithium can no longer enter the cells through these channels.
5. The cells regain some ability to respond to ADH.
6. Aquaporin-2 water channels are inserted into the cell membrane more normally.
7. Water is reabsorbed from urine, reducing urine volume.

SAFETY WATCH

High potassium (hyperkalemia): Amiloride keeps potassium in your body. Watch for muscle weakness, tingling, or irregular heartbeat. Your doctor will check potassium levels regularly. Avoid potassium supplements and salt substitutes (which contain potassium) unless your doctor says otherwise. · Kidney function: If your kidneys are already weak, amiloride can worsen potassium buildup. Dose may need adjustment. · Dehydration: During illness with vomiting, diarrhea, or fever, call your doctor. Fluid balance becomes tricky. · Drug interactions: NSAIDs like ibuprofen and naproxen, ACE inhibitors (blood pressure pills ending in -pril), and ARBs (ending in -sartan) all raise potassium. Combining them with amiloride increases high potassium (hyperkalemia) risk. Always tell your doctor about all medications. · Lithium levels: Amiloride does not lower lithium blood levels. You still need regular lithium monitoring to avoid toxicity.

Indomethacin · Nonsteroidal anti-inflammatory drug (NSAID)

Indomethacin is an NSAID that can reduce urine output in nephrogenic diabetes insipidus by lowering the kidney's filtration rate and blocking prostaglandins. Prostaglandins are chemicals the kidney makes that normally oppose ADH, making the kidney release more water. Blocking prostaglandins helps the kidney hold onto more water. Indomethacin is not a first-choice drug. It's usually reserved for cases that don't respond well to thiazides or amiloride, or in very young infants, because long-term NSAID use has risks.

1. You take indomethacin by mouth, usually two or three times a day.
2. It blocks cyclooxygenase (COX) enzymes, which stops the kidney from making prostaglandins.
3. Without prostaglandins, the kidney's filtration rate (glomerular filtration rate, or GFR) drops slightly, so less water enters the urine in the first place.
4. Prostaglandins normally counteract ADH in the collecting duct. Blocking them lets any remaining ADH effect work better.
5. Water reabsorption increases modestly, and urine output drops.

SAFETY WATCH

Stomach ulcers and bleeding: NSAIDs irritate the stomach lining. Watch for black stools, stomach pain, or vomiting blood. Take with food. Your doctor may prescribe a proton pump inhibitor (PPI) to protect your stomach. · Kidney damage: Long-term NSAID use can harm the kidneys, especially in people with diabetes insipidus who already have kidney stress. Your doctor will monitor kidney function (creatinine and GFR) regularly. · Heart and stroke risk: NSAIDs slightly raise the risk of heart attack and stroke, especially at high doses or long duration. · Bleeding risk: NSAIDs thin the blood slightly. Avoid if you take warfarin or have a bleeding disorder. · Dehydration: If you get sick and can't drink, stop indomethacin and call your doctor. It can worsen dehydration and kidney injury. · Not for routine use: Because of these risks, indomethacin is typically used only when safer options like thiazides and amiloride don't work well enough.

Red flags

- Sudden confusion, severe headache, or seizure—signs that sodium is dangerously high and brain cells are shrinking, which is a medical emergency
- Unable to keep up with thirst or unable to drink enough—risk of severe dehydration, shock, and organ damage
- Fast heart rate, low blood pressure, or dizziness when standing—signals that blood volume is dropping too low and the heart is struggling to pump enough
- New-onset diabetes insipidus after head injury, brain surgery, or brain infection—may mean damage to the hypothalamus or pituitary gland that makes vasopressin
- Infant or young child with fever, vomiting, or refusal to drink—babies dehydrate much faster and cannot tell you they are thirsty, so this is an emergency

Nursing priorities

1. Monitor fluid intake and urine output every hour and compare the volumes to identify severe fluid imbalance — Patients can lose 3 to 20 liters of urine per day, and if intake does not match output, rapid dehydration occurs with hypovolemia (low blood volume), leading to shock, acute kidney injury, and death within hours
2. Check serum sodium level immediately and monitor every 4 to 6 hours, reporting any value above 145 mEq/L to the physician — High sodium (Hypernatremia) (high sodium) develops when water loss exceeds intake, causing brain cells to shrink, which triggers confusion, seizures, coma, and permanent brain damage if sodium rises above 160 mEq/L

3. Measure urine specific gravity with each void, reporting values below 1.005 that confirm dilute urine — Low specific gravity proves the kidneys are not concentrating urine despite dehydration, which is the defining feature of diabetes insipidus and distinguishes it from other causes of frequent urination like diabetes mellitus or diuretic use
4. Ensure the patient has unrestricted access to water at the bedside and can drink freely whenever thirsty — The thirst mechanism is the body's only defense against high sodium (hypernatremia) in diabetes insipidus, so blocking access to water or delaying drinking causes sodium to climb rapidly and brain damage to occur
5. Administer desmopressin (DDAVP) exactly as ordered—typically 0.05 to 0.2 mg by mouth twice daily or 10 to 20 mcg by nasal spray—and monitor for reduced urine output within 1 to 2 hours — Desmopressin is synthetic ADH (antidiuretic hormone) that tells the kidneys to reabsorb water, so it directly treats central diabetes insipidus by replacing the missing hormone and stops life-threatening water loss
6. Weigh the patient at the same time every day on the same scale, reporting any weight loss greater than 2 pounds in 24 hours — Rapid weight loss reflects fluid loss, with each pound equaling about 500 mL of water, so a 4-pound loss means 2 liters of dehydration, which triggers a fast heart rate (tachycardia), low blood pressure (hypotension), and organ poor blood flow (hypoperfusion) (not enough blood flow)
7. Monitor vital signs every 4 hours, watching for heart rate above 100 beats per minute, blood pressure drop of more than 20 mmHg when standing, or decreased level of consciousness — A fast heart rate (Tachycardia) and orthostatic low blood pressure (hypotension) (blood pressure drop with position change) are early signs that blood volume is too low, while altered mental status signals that the brain is not getting enough oxygen or sodium is dangerously high
8. Teach the patient to recognize early dehydration signs—dark urine, dry mouth, dizziness, headache—and to drink extra water immediately — Self-management prevents emergency-room visits and life-threatening high sodium (hypernatremia), because the patient can adjust fluid intake before severe dehydration develops, especially during illness, heat exposure, or exercise when water loss increases

Sources

- Endocrine Society Clinical Practice Guideline on Diabetes Insipidus (2024)
- UpToDate Clinical Reference
- NEJM Review Articles
- American Academy of Pediatrics (for pediatric cases)

Last reviewed: 2026-07-07