

Diabetes Insipidus

A plain-English guide for you and your family

WHAT IS HAPPENING

Your kidneys are like a smart filter that decides what to throw away and what to keep. Normally, a tiny gland at the base of your brain (the pituitary gland, a pea-sized control center) sends out a hormone called ADH (a chemical messenger that tells the kidneys to hold onto water). When your blood gets even a little bit too salty or thick, this hormone travels through your blood to the kidneys and tells them to pull water back into your body instead of letting it leave as pee. That way your blood stays the right thickness, like a well-mixed soup. In diabetes insipidus, that system breaks. Either the pituitary gland or the brain area just above it (the hypothalamus, the master control room) is hurt and stops making ADH, or the kidneys are damaged and cannot hear the ADH signal anymore. Without that signal, the kidneys act like a broken dam and let water pour out all day and night. You might pee 3 to 20 liters a day (a normal person pees about 1 to 2 liters). Your body senses the blood getting too salty and thick, so it turns on a powerful thirst alarm to make you drink enough to replace what you are losing. As long as you can drink freely and your thirst center works, you stay balanced but exhausted from constant drinking and peeing. But if you cannot drink (maybe you are asleep, sick, or do not have water nearby), your blood salt climbs dangerously high and can cause confusion, seizures, or coma. All the strange symptoms trace back to this one broken piece: no working ADH signal to hold water in.

What you might feel

- Very large amounts of watery pee (polyuria)
- Pee is very pale or clear
- Extreme thirst (polydipsia)
- Getting up many times at night to pee (nocturia)
- Dry mouth and dry skin
- Fatigue and weakness
- High sodium level in the blood (hypernatremia)
- Confusion or irritability

- Weight loss
- Constipation

Call the doctor right away if...

- 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

What the care team will do

- Monitor fluid intake and urine output every hour and compare the volumes to identify severe fluid imbalance
- Check serum sodium level immediately and monitor every 4 to 6 hours, reporting any value above 145 mEq/L to the physician
- Measure urine specific gravity with each void, reporting values below 1.005 that confirm dilute urine
- Ensure the patient has unrestricted access to water at the bedside and can drink freely whenever thirsty
- 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
- Weigh the patient at the same time every day on the same scale, reporting any weight loss greater than 2 pounds in 24 hours
- 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
- Teach the patient to recognize early dehydration signs—dark urine, dry mouth, dizziness, headache—and to drink extra water immediately

Medicines you may be given — and why

Desmopressin (DDAVP)

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.

Hydrochlorothiazide (HCTZ)

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.

Amiloride

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.

Indomethacin

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.

Where this information comes from

- 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