

Diabetes Insipidus & SIADH

A plain-English guide for you and your family

WHAT IS HAPPENING

Your body has a water-control hormone called ADH (anti-diuretic hormone, made deep in your brain by the pituitary gland). ADH tells your kidneys how tightly to hold onto water. When your blood gets too salty or too low on water, your brain releases more ADH, kidneys grip water tighter, and you pee less — keeping your blood balanced. When your blood is too watery, your brain releases less ADH, kidneys let water go, and you pee more. That is the normal job. In DI (diabetes insipidus), this switch breaks: either your brain stops making enough ADH (central DI), or your kidneys stop listening to it (nephrogenic DI). Without that signal, your kidneys let huge amounts of water flood out in your pee — gallons a day, thin and pale like water. Your blood becomes too salty (high sodium), and you feel horribly thirsty all the time because your body is screaming for water to refill the tank. In SIADH (syndrome of inappropriate ADH), the opposite happens: your body releases too much ADH — often triggered by brain injuries, lung infections, some cancers, or certain medicines. Your kidneys grip water too tightly, flooding your blood with water and diluting your sodium (low sodium, called low sodium (hyponatremia)). Your pee becomes thick and concentrated even though your blood is too watery. You do not swell up because the extra water spreads evenly inside cells and blood, but your brain can swell from the low sodium, causing confusion, seizures, or worse. None of this is random. Every weird finding — the gallons of thin pee, the desperate thirst, the high or low sodium — traces back to one broken switch upstream. The body is trying to cope, but it cannot fix the root problem on its own.

What you might feel

- Making huge amounts of very pale, watery pee (polyuria) — often 3 to 20 liters a day
- Constant, strong thirst (polydipsia) — needing to drink water all day and night
- Waking up multiple times at night to pee and drink (nocturia)
- Dry mouth and skin; feeling dehydrated
- Confusion, irritability, or sleepiness (in SIADH)
- Nausea and headache (in SIADH)
- Normal blood pressure and no visible swelling of the legs or belly (in SIADH)

- Seizures or coma (severe SIADH)

Call the doctor right away if...

- Sodium below 120 mEq/L — brain swelling risk is high; person may seize or become unresponsive
- Severe confusion, seizure, or loss of consciousness — emergency; needs immediate hospital care and careful, slow correction of sodium
- Unable to drink or keep up with urine output in diabetes insipidus — leads to dangerous dehydration, low blood pressure, and shock

What the care team will do

- Check serum sodium level immediately and monitor it every 4 to 6 hours if abnormal.
- Measure and record every drop of urine output and fluid intake every hour.
- Check urine specific gravity (normal is 1.005 to 1.030) with each void or every 2 to 4 hours.
- Weigh the patient at the same time every day on the same scale, wearing similar clothes.
- Monitor blood pressure, heart rate, and skin turgor (skin tenting) every 4 hours.
- Assess mental status and neurological signs (confusion, headache, muscle twitching, reflexes) every 2 to 4 hours.
- Enforce strict fluid restriction (usually 800 to 1000 mL per day) for SIADH patients, and explain why to the patient and family.
- Keep cold water or ice chips at the bedside for diabetes insipidus patients, and allow free access to fluids.

Medicines you may be given — and why

Desmopressin (DDAVP)

This is a lab-made copy of the natural 'hold-water' hormone (ADH) that your pituitary normally releases. When your pituitary is damaged and can't make enough of its own, desmopressin steps in to do the same job—it tells your kidneys to hold onto water instead of losing it all as pee.

Hydrochlorothiazide (HCTZ)

This seems backward at first—using a 'water pill' to treat someone losing too much water—but it works by a clever trick. The thiazide makes your body hold a little less salt in a certain part of the kidney, which causes a small drop in blood volume. That drop signals your kidneys to

grab MORE water earlier in the tube (before the broken part), so less water reaches the collecting duct where the problem lives. You end up peeing less overall.

Amiloride

Like the thiazide, amiloride works by blocking a salt channel (ENaC) in the kidney tube. This causes a small upstream increase in water reabsorption, reducing how much water reaches the broken collecting duct. It also does NOT waste potassium, which is helpful if thiazides alone drop your potassium too much. This is especially useful in lithium-caused nephrogenic DI, because amiloride may also block some of the damage lithium does to kidney cells.

Indomethacin

This painkiller has a side effect that actually helps in nephrogenic DI: it lowers the kidney's blood flow slightly and reduces how much water the kidney filters in the first place. It also blocks prostaglandins, chemicals that normally tell the kidney to make more urine. By quieting that signal, you pee less. It's usually added to a thiazide, not used alone.

Fluid restriction

The simplest first step for mild SIADH: if your body is holding onto too much water and diluting your sodium, drinking LESS water lets your kidneys slowly release the excess. Your sodium will drift back up naturally. This only works if the SIADH is mild and your sodium isn't dangerously low to start.

Sodium chloride tablets (salt pills)

You're giving your body extra sodium to raise the blood level directly. In SIADH, your kidneys are dumping sodium into urine while holding onto water—so even though you're diluted, you're also losing salt. Adding salt back helps pull your sodium number up, and the extra salt in your blood can also trigger a small increase in urine output (because the kidney tries to balance the salt).

Urea

Urea is a waste product your body normally makes; here you take extra as medicine. It acts like a gentle water-puller: the urea enters your blood, then your kidneys filter it into urine. Because urea pulls water with it, you pee out more free water (water without extra sodium), which raises your blood sodium. It's especially useful in chronic SIADH when fluid restriction alone isn't enough.

Demeclocycline

This is an old antibiotic that has a useful side effect: it makes your kidneys LESS responsive to ADH. In SIADH, you have too much ADH signal; demeclocycline turns down the kidney's ability

to hear that signal, so you pee out more water. It's used when SIADH is chronic and other treatments haven't worked well enough.

Tolvaptan

This medicine is a blocker that sits on the ADH receptor in your kidney and prevents ADH from docking there. Even though your body is making too much ADH (SIADH), tolvaptan keeps it from working. Your kidneys then release free water as dilute urine, and your blood sodium climbs. It's the most targeted treatment for SIADH, especially when sodium is moderately low and other treatments haven't worked.

Conivaptan

Like tolvaptan, conivaptan blocks the ADH receptor in the kidney so ADH can't make you hold water. It also blocks V1a receptors (on blood vessels), which can slightly relax vessels. It's given through an IV, so it's used in the hospital for patients who can't take pills or need faster action. It works the same way—freeing your kidneys to pee out excess water and raising your sodium.

Hypertonic saline (3% sodium chloride IV)

This is concentrated salt water given directly into your vein. It raises your blood sodium FAST by adding a large dose of sodium all at once. It also pulls water OUT of swollen brain cells (osmotic effect), which is life-saving when low sodium has caused brain swelling, seizures, or coma. It's the emergency treatment for severe, symptomatic SIADH (or any severe low sodium (hyponatremia)).

Where this information comes from

- Endocrine Society Clinical Practice Guideline on Diabetes Insipidus
- American Association of Clinical Endocrinology (AACE) SIADH/Hyponatremia Guidelines
- UpToDate Evidence-Based Clinical Decision Support
- The New England Journal of Medicine (NEJM) Review on Disorders of Water Balance

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