

Diabetes Insipidus & SIADH

DI and SIADH are opposite switches: DI dumps water out, SIADH traps water in.

THE ONE-LINE TRUTH

DI happens when your body makes too little ADH or your kidneys ignore it, so water floods out in your pee.

The chain — how it unfolds

1. In DI, your brain stops making ADH or your kidneys ignore it, so the water doors in the kidney tubes stay closed. — why: Without ADH's signal, the collecting ducts (final tubes in the kidney) cannot open their water channels (aquaporins), so water that should be pulled back into the blood just pours out into the bladder.
2. You pee huge amounts (often 3 to 20 liters a day) of very thin, pale, dilute urine (specific gravity below 1.005, osmolality below 200 mOsm/kg). — why: All the water your body needs is flooding out because the kidneys have no instructions to hold it — it is like a dam with all the gates stuck open.
3. Your blood becomes too salty (hypernatremia, sodium above 145 mEq/L, often climbing toward 160 or higher). — why: You are losing pure water but keeping salt, so the salt in your blood becomes more and more concentrated — like leaving a pot of soup on the stove until the water boils off and only thick salty liquid is left.
4. You feel horribly, constantly thirsty and crave water. If you can drink, you may gulp gallons a day and barely keep up. — why: Your brain's thirst center senses the high sodium and screams at you to drink — this is the body's only way to try to refill the tank and dilute the blood back to normal.
5. If you cannot drink enough (after surgery, unconscious, a baby, or trapped without water), sodium climbs dangerously high (above 160), your brain cells shrink, blood vessels tear, and you can have seizures, confusion, coma, or bleeding inside the skull. — why: Water moves out of brain cells toward the salty blood to try to balance things, so the brain shrinks away from the skull. This pulls and tears tiny blood vessels, causing bleeding (subdural or subarachnoid bleeding (hemorrhage)) and permanent brain damage or death.

6. In SIADH, your body releases too much ADH, so the water doors in the kidneys stay wide open all the time, pulling too much water back into the blood. — why: The collecting ducts are told to grip water even when the blood is already too watery — the kidneys cannot tell the difference because the ADH signal is lying to them.
7. You pee small amounts of thick, dark, concentrated urine (osmolality above 100 mOsm/kg, often above 300) even though your blood is too watery. — why: Your kidneys are hoarding water and only releasing waste and some sodium, so the urine that does come out is very concentrated — the opposite of DI.
8. Your blood sodium drops (hyponatremia, sodium below 135 mEq/L, often falling toward 120 or lower) because your blood is flooded with too much water, diluting the salt. — why: It is like pouring too much water into your soup — the soup becomes thin and tasteless because the salt is spread out in too much liquid.

Spot it — what you see, and why

- Making huge amounts of very pale, watery pee (polyuria) — often 3 to 20 liters a day: In diabetes insipidus, the body either doesn't make enough of the hormone ADH (also called vasopressin, which tells the kidneys to hold onto water) or the kidneys ignore it. Without that signal, the kidneys let almost all the water pass straight through into the bladder instead of pulling it back into the blood — like a sponge that can't squeeze tight.
- Constant, strong thirst (polydipsia) — needing to drink water all day and night: Because so much water is leaving the body as dilute urine, the blood becomes more concentrated (higher sodium). Special sensors in the brain detect this and turn on the thirst signal to make you drink and replace the lost water.
- Waking up multiple times at night to pee and drink (nocturia): The kidneys keep flushing out water around the clock in diabetes insipidus, so the bladder fills quickly even during sleep. The body then wakes you up with thirst to replace what you lost.
- Dry mouth and skin; feeling dehydrated: If a person cannot drink fast enough to match the water loss in the urine — for example, during sleep or when water is not available — the body starts to dry out. Blood volume drops, and tissues have less water in them.
- Confusion, irritability, or sleepiness (in SIADH): In SIADH, the body makes too much ADH, so the kidneys hold onto water even when they should let it go. This dilutes the sodium in the blood (hyponatremia). Brain cells swell when sodium drops, and that swelling changes how nerve signals work — leading to confusion, trouble thinking, or feeling very tired.
- Nausea and headache (in SIADH): Low blood sodium from too much ADH makes brain cells swell slightly. That swelling increases pressure inside the skull and irritates the area of the brain that controls nausea, causing headache and an upset stomach.

Red flags — act now

- 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

Treat it — in this order

1. For central DI: give desmopressin (DDAVP) as a nasal spray, oral tablet, or injection → Desmopressin is synthetic ADH that replaces the missing hormone. It tells the kidneys to hold water back, so urine volume drops, thirst goes away, and serum sodium falls back to normal (135–145 mEq/L). The effect starts within 1–2 hours and lasts 8–24 hours.
2. For nephrogenic DI: give a thiazide diuretic (hydrochlorothiazide 25–50 mg daily) and tell the person to eat a low-salt diet (< 2 g sodium per day) → Thiazides cause mild dehydration, which makes the body hold onto more water in the early part of the kidney (proximal tubule), so less water reaches the broken collecting duct. A low-salt diet keeps the blood less salty so the kidneys do not need to dump as much water to balance it. Together, urine volume can drop by 30–50%.
3. For nephrogenic DI: add amiloride (5–10 mg daily) if lithium is the cause → Amiloride blocks the channel in the collecting duct that lithium uses to enter kidney cells and cause damage. This protects the cells and can reduce urine output. It is most useful when lithium must be continued (e.g., for bipolar disorder).
4. For SIADH: restrict fluid intake to 800–1000 mL per day (less than urine output) → When you drink less water than your body loses, the blood becomes less diluted and sodium slowly climbs back to normal (135–145 mEq/L). This is the safest first step and often works within 24–48 hours for mild SIADH.
5. For SIADH with severe symptoms (sodium < 120 mEq/L, seizures, confusion, coma): give 3% hypertonic saline IV at 1–2 mL/kg/hour → Concentrated salt water pulls water out of swollen brain cells and stops seizures and coma. Sodium should rise no faster than 4–6 mEq/L in the first 24 hours (maximum 8 mEq/L) to avoid osmotic demyelination syndrome (brain damage from correcting sodium too fast).

Your top priorities at the bedside

1. Check serum sodium level immediately and monitor it every 4 to 6 hours if abnormal. — why: Sodium tells you which condition you are dealing with—high sodium (above 145 mEq/L) points to diabetes insipidus, low sodium (below 135 mEq/L) points to SIADH; sodium below 120 mEq/L can cause brain swelling, seizures, and death.
2. Measure and record every drop of urine output and fluid intake every hour. — why: Strict intake and output tracking shows if the person is losing more water than they are drinking (diabetes insipidus) or retaining too much water (SIADH); it guides how much fluid to give or restrict.
3. Check urine specific gravity (normal is 1.005 to 1.030) with each void or every 2 to 4 hours. — why: Very dilute urine with specific gravity below 1.005 confirms diabetes insipidus because kidneys are not concentrating urine; concentrated urine above 1.020 with low sodium suggests SIADH.