

Diabetes Insipidus & SIADH

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THE STORY IN ONE SENTENCE

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

Normal anatomy — the body's job

Your brain watches your blood's saltiness (sodium level) every second. When blood gets too salty or volume drops, a tiny gland deep in your brain (the pituitary) releases ADH into your blood. ADH travels to your kidneys and tells the collecting ducts (the final tubes before urine leaves) to open water doors and pull water back into your blood, so you pee less and keep water. When blood is too watery, your brain releases less ADH, kidneys let water go, and you pee more. This loop keeps your blood sodium and water balanced every minute of every day.

BODY'S JOB

ADH is your body's water-saving remote control. It keeps your blood from getting too salty or too dilute by adjusting how much water your kidneys hold or release. Without it, your kidneys have no instructions and just let all water pour out.

1. Blood gets too salty or too low on water → brain senses the change
2. Brain releases more ADH from the pituitary gland into the bloodstream
3. ADH travels through blood to the kidneys
4. ADH tells the collecting ducts (final tubes in the kidney) to open water channels (aquaporins) and pull water back into blood
5. You pee less, keep water, blood becomes less salty and more full
6. When blood is too watery → brain releases less ADH → kidneys let water go → you pee more and blood stays balanced

What breaks

The ADH system breaks in two main ways. In central DI, your brain stops making enough ADH — usually because of brain surgery, a head injury, a tumor pressing on the pituitary, or (rarely) a genetic problem. In nephrogenic DI, your brain makes plenty of ADH, but your kidneys ignore it — often caused by long-term lithium (a mood medicine), low potassium, high calcium, kidney diseases, or (rarely) a genetic defect in the water channels. In SIADH, the opposite happens: your body releases too much ADH when it should not — triggered by brain injuries (bleeds, infections, tumors), lung problems (pneumonia, small-cell lung cancer), some cancers that make their own ADH, or medicines like SSRIs, carbamazepine, and certain chemo drugs.

CLINICAL TERM

Central diabetes insipidus results from inadequate ADH secretion by the posterior pituitary (causes: neurosurgery, traumatic brain injury, pituitary tumors, craniopharyngioma, hypophysitis, genetic mutations in the ADH gene). Nephrogenic diabetes insipidus results from renal resistance to ADH (causes: chronic lithium therapy, high calcium (hypercalcemia), low potassium (hypokalemia), chronic kidney disease, genetic mutations in the AVPR2 or AQP2 genes). SIADH results from excess ADH secretion or action (causes: CNS disorders such as SAH, meningitis, traumatic brain injury; pulmonary disorders such as pneumonia, small-cell lung cancer; ectopic ADH production by malignancies; medications including SSRIs, carbamazepine, cyclophosphamide, vincristine, and NSAIDs).

How the body compensates

Your body tries to cope by making you desperately thirsty in DI (to replace lost water) or by dumping some sodium in the urine in SIADH (to try to raise blood sodium). These tricks buy time but cannot fix the root problem. The danger is that the body can look stable for a while — normal blood pressure, normal heart rate — while sodium is climbing or crashing silently. Once sodium swings too far too fast, the brain swells or shrinks, and seizures or coma can happen suddenly. The window to reverse brain injury closes fast.

1. In DI, you feel horribly thirsty and drink huge amounts of water. This is the body screaming for water to replace what is flooding out in the pee. If you can drink enough, your sodium may stay almost normal — but if you cannot drink (after surgery, unconscious, or a baby), sodium climbs dangerously high and the brain shrinks and bleeds.
2. In SIADH, your body tries to dump some sodium into the urine to fight the low blood sodium. This buys a little time, but it cannot keep up if ADH stays too high, so sodium keeps dropping.

3. Vital signs (blood pressure, heart rate) can look normal in both DI and SIADH while sodium is swinging dangerously — this is the dangerous middle, because the team can miss how sick the patient really is.
4. The crash point in DI is severe high sodium (hypernatremia) (sodium above 160 mEq/L): the brain shrinks, blood vessels inside the skull tear, causing bleeding, seizures, coma, and death.
5. The crash point in SIADH is severe low sodium (hyponatremia) (sodium below 120 mEq/L, especially if it drops fast): the brain swells inside the rigid skull, causing herniation (brain tissue gets squeezed down), seizures, coma, and death.
6. Even if you fix the sodium, fixing it too fast causes its own disaster: correcting sodium faster than 8–10 mEq/L in 24 hours can cause osmotic demyelination syndrome (the brain's insulation strips off), leading to permanent paralysis, speech loss, or death.
7. Watch trends, not just one number. A sodium of 145 that was 130 yesterday is falling fast — that patient is not stable. A sodium of 130 that has been stable for days is much safer.
8. A calm-looking patient on a downhill sodium slope is a ticking time bomb — catch it early, before the brain swells or shrinks past the point of no return.

Domino effect

1. In DI, your brain stops making ADH or your kidneys ignore it, so the water doors in the kidney tubes stay closed. — 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). — 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). — 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. — 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. — 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. — 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. — 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. — 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.
9. You do not swell up or gain visible water weight, because the extra water spreads evenly inside your blood and cells, not into the tissues under your skin. — Swelling (Edema) (swelling) happens when water leaks out of blood vessels into tissue spaces — but in SIADH, water stays inside the vessels and cells, so you look normal on the outside even though your blood is too dilute.
10. Your brain cells swell as water moves into them to try to balance the low sodium outside. This causes confusion, headache, nausea, seizures, and (if severe) coma or brain herniation (brain tissue gets squeezed down through the skull opening and dies). — Water always moves toward salt to try to even things out. When blood sodium is low, water rushes into brain cells, but the skull is a rigid box, so swollen brain tissue has nowhere to go — it gets crushed against the skull and can herniate downward, cutting off blood flow and killing brain tissue.

Symptoms · what the patient feels, 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.
- Normal blood pressure and no visible swelling of the legs or belly (in SIADH): Even though the kidneys are holding extra water, the body balances it by shifting fluid into cells and slowing down how much sodium the kidneys let go. This keeps total blood volume from rising too high, so you don't see puffy ankles or weight gain like you do with heart or kidney failure.
- Seizures or coma (severe SIADH): When blood sodium falls very low — usually below 120 milliequivalents per liter — brain cells swell so much that the electrical activity in the brain misfires. This can cause a seizure (uncontrolled shaking) or, if sodium drops even further, the brain can shut down into a coma.

Focused exam

- Excessive urination (Polyuria) (very large amounts of pale, watery urine—often 3–20 liters per day): The kidneys cannot hold water back because ADH (the hormone that tells them to save water) is either missing or the kidneys ignore it. This massive water loss is the signature sign of diabetes insipidus and tells you the body's water-saving system is broken.
- Excessive thirst (Polydipsia) (extreme thirst, drinking constantly): The brain senses water pouring out and turns on the thirst center to force the person to drink and replace the lost water. If the person cannot drink (sleeping, confused, unable to access water), sodium climbs dangerously high and brain cells shrink, which can cause seizures or coma.

- Serum sodium above 145 mEq/L (often 145–160 mEq/L): Water leaves the body but salt stays behind, so the blood becomes saltier and saltier. A sodium above 145 mEq/L is the strongest clue that DI is present—it shows the kidneys are dumping water instead of holding it.
- Urine osmolality below 200 mOsm/kg (often as low as 50–100 mOsm/kg): Normal kidneys concentrate urine to save water; DI kidneys make urine so dilute it is almost like drinking water. A urine osmolality below 200 when the blood is thick (high plasma osmolality) proves the kidneys are not responding to ADH.
- Plasma osmolality above 295 mOsm/kg (often 295–320 mOsm/kg): The blood becomes thick and concentrated because water has been lost but particles (sodium, glucose) remain. The mismatch—thick blood and thin urine at the same time—is DI's fingerprint and tells you ADH is failing to do its job.
- Signs of dehydration: dry mouth and lips, poor skin turgor (skin tents when pinched), dizziness or drop in blood pressure when standing (orthostatic low blood pressure (hypotension)): DI drains so much water from the body that tissues shrink and blood volume drops. These physical signs show how severe the water loss is and warn you the person may collapse if fluids are not replaced soon.

Diagnostics · every test answers a question

- Serum sodium → Is the blood getting too salty because water is being lost faster than salt?
- Plasma osmolality → Is the blood thick and concentrated, which should trigger ADH release?
- Urine osmolality → Are the kidneys concentrating urine to save water, or are they dumping water out?
- 24-hour urine volume → How much urine is the person making in a full day?
- Water deprivation test (also called desmopressin challenge) → Can the kidneys concentrate urine when we withhold water, and do they respond when we give synthetic ADH?
- Serum ADH (vasopressin) level → Is the pituitary making ADH, or is the problem in the kidneys?
- MRI of the brain (pituitary and hypothalamus) → Is there damage, a tumor, or bleeding in the pituitary or hypothalamus that would stop ADH production?
- Medication review and serum lithium or calcium level → Is a drug or metabolic problem blocking the kidneys from responding to ADH?

Treatment logic

- 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.
- 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%.
- 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).
- 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.
- 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).
- For SIADH: give oral salt tablets (1–3 g sodium three times daily), urea powder (15–30 g twice daily), or a vasopressin receptor antagonist (tolvaptan 15–60 mg daily) → Salt tablets and urea make the blood saltier so the kidneys must hold more water to dilute it, which raises serum sodium. Tolvaptan blocks ADH receptors in the kidney, so the kidneys ignore ADH and pee out pure water (aquaresis), concentrating the blood sodium.
- Find and treat the root cause: pituitary surgery or tumor for central DI; stop lithium or fix high calcium for nephrogenic DI; treat cancer, pneumonia, or stop the offending drug (SSRIs, carbamazepine) for SIADH → Fixing the cause can restore normal ADH production or kidney response, so the body regulates water on its own again. For example, removing a pituitary tumor may allow ADH cells to recover, and stopping lithium can let kidneys heal over weeks to months.

Medications

Desmopressin (DDAVP) · Synthetic vasopressin analog

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.

1. You take the medicine (nasal spray, pill, or injection).
2. It travels through your blood to your kidneys.
3. It locks onto V2 receptors on the surface of kidney cells.
4. That signal tells the cell to put tiny water-pores (called aquaporins) into the wall of the tube.
5. Water from the tube now flows back into your body through those pores, so less water leaves as urine.
6. Your urine becomes darker and smaller in volume; you stop being so thirsty.

SAFETY WATCH

Watch for water overload: if you drink too much while taking this, your sodium can drop dangerously low (hyponatremia), which can cause confusion, seizures, or even brain swelling. · Limit how much you drink—only when truly thirsty—while on this medicine. · Nasal spray may not work well if you have a cold or stuffy nose. · Let your doctor know if you get a headache, feel very tired, or feel nauseous—these can be early signs of low sodium.

Hydrochlorothiazide (HCTZ) · Thiazide diuretic ('water pill')

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.

1. You take the pill by mouth.
2. It blocks a salt-moving pump (NCC) in the distal tubule.
3. Less salt is pulled back into your body at that spot, so a bit more salt leaves in urine.
4. Your blood volume drops slightly.
5. The kidney senses that drop and turns on systems (like renin-angiotensin) to save water upstream.

6. More water gets reabsorbed early, so less water arrives at the broken collecting duct.
7. Your total urine volume goes down by about 30–50 percent.

SAFETY WATCH

You **MUST** eat a low-salt diet for this to work—if you eat a lot of salt, the medicine can't do its job. · Can cause low potassium, so your doctor may check blood tests and tell you to eat bananas or take a potassium pill. · Can raise blood sugar and uric acid slightly, so monitor if you have diabetes or gout. · Drink enough to satisfy thirst, but not more—dehydration is still a risk if you restrict fluids too much.

Amiloride · Potassium-sparing diuretic

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.

1. You take the pill.
2. It blocks ENaC sodium channels in the collecting duct.
3. Less sodium is pulled back, so a bit more stays in the urine.
4. The kidney compensates by reabsorbing more water earlier.
5. Urine volume drops.
6. Your potassium stays stable (unlike with thiazides).

SAFETY WATCH

Can cause **HIGH** potassium (hyperkalemia), especially if you also take ACE inhibitors, ARBs, or potassium supplements—your doctor will check blood tests. · Still requires a low-salt diet to be effective. · Tell your doctor if you feel weak, have an irregular heartbeat, or feel numbness—these can be signs of potassium trouble.

Indomethacin · Nonsteroidal anti-inflammatory drug (NSAID)

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.

1. You take the pill.
2. It blocks an enzyme (COX) that makes prostaglandins.

3. With fewer prostaglandins, the kidney vessels tighten a little, so less blood is filtered.
4. Less filtration means less urine is made overall.
5. The tubules also become slightly more responsive to whatever ADH signal is present.

SAFETY WATCH

Can hurt your stomach—take with food to lower the risk of ulcers or bleeding. · Can raise blood pressure or cause fluid retention in some people. · Can harm kidney function if used long-term or in high doses—your doctor will monitor creatinine. · Avoid if you have a history of stomach ulcers, kidney disease, or heart failure. · Not safe during pregnancy (third trimester).

Fluid restriction · Non-drug intervention

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.

1. You limit how much you drink each day (usually 800–1,000 mL, about 4 cups).
2. Your kidneys keep holding water (because of the SIADH), but you're not adding NEW water faster than they release the old.
3. Over days, the balance tips: more water leaves than comes in.
4. Your blood sodium climbs slowly back toward normal.
5. Symptoms (confusion, nausea, headache) improve as sodium rises.

SAFETY WATCH

Hard to stick with—people get very thirsty, and it's tough to limit fluids for weeks. · Does NOT work if you keep eating a lot of salt or drinking sports drinks, because those add fluid back in. · Your doctor will check your sodium every few days to make sure it's rising at a safe, slow pace.

Sodium chloride tablets (salt pills) · Electrolyte supplement

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).

1. You swallow the salt tablet (usually 1–2 grams, one to three times a day).

2. The sodium dissolves and enters your blood.
3. Your blood sodium level climbs a bit.
4. The higher sodium may trigger your kidneys to release a little more water as urine.
5. Over days, sodium normalizes if you also restrict fluids.

SAFETY WATCH

Can cause nausea or stomach upset—take with food. · NOT safe if you have heart failure or severe kidney disease, because extra salt can cause dangerous fluid overload and swelling. · Your doctor will monitor sodium levels to make sure you're not raising it too fast (no more than 8 mEq/L in 24 hours) to avoid osmotic demyelination syndrome (brain damage from too-rapid correction).

Urea · Osmotic diuretic (by mouth)

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.

1. You take urea powder mixed in juice or water (tastes salty-bitter).
2. It's absorbed into your blood.
3. Your kidneys filter it into the urine.
4. Urea pulls water into the urine with it (osmotic effect).
5. You pee out extra free water, so your blood sodium goes up.
6. You can drink more normally because the urea helps you get rid of the excess water.

SAFETY WATCH

Tastes unpleasant—mix with juice to hide the flavor. · Can cause stomach upset, nausea, or diarrhea. · Requires regular sodium checks to avoid raising sodium too fast. · Not widely available in all countries; some patients have trouble finding or affording it.

Demeclocycline · Tetracycline antibiotic (used off-label)

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.

1. You take the pill twice a day.

2. It enters kidney cells.
3. It interferes with the ADH signaling pathway (exact mechanism still not fully clear, but it blocks water-pore insertion).
4. The kidney becomes partially resistant to ADH.
5. You pee out more dilute urine, losing free water.
6. Your blood sodium rises over several days.

SAFETY WATCH

Can cause kidney damage itself (nephrogenic diabetes insipidus as a side effect)—your doctor will monitor creatinine. · Makes your skin very sensitive to sunlight—wear sunscreen and avoid tanning beds. · Can cause nausea, diarrhea, and tooth discoloration (in children under 8). · Avoid in pregnancy and breastfeeding. · Do NOT take with dairy, antacids, or iron—these block absorption; take on an empty stomach with water.

Tolvaptan · Vasopressin V2-receptor antagonist (vaptan)

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.

1. You take the pill once a day.
2. Tolvaptan travels to your kidneys and locks onto V2 receptors.
3. ADH cannot attach to those receptors anymore.
4. The kidney stops inserting water-pores (aquaporins) into the duct.
5. Water stays in the urine tube and leaves your body.
6. You pee large amounts of dilute urine (aquaresis—water without sodium).
7. Your blood sodium rises, often within hours to days.

SAFETY WATCH

MUST start in the hospital with frequent sodium checks (every 4–6 hours at first) because it can raise sodium dangerously fast, risking osmotic demyelination syndrome (permanent brain damage). · Causes intense thirst and large urine output—patients need access to water and a bathroom. · Can cause liver injury—requires liver function tests before and during treatment; do NOT use if you have liver disease. · Very expensive and not available everywhere. · Do NOT use for more than 30 days in a row (FDA restriction due to liver risk). · Interactions: do NOT take with grapefruit juice or strong CYP3A4 inhibitors (like ketoconazole)—they make tolvaptan levels dangerously high.

Conivaptan · Vasopressin V1a/V2-receptor antagonist (vaptan)

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.

1. The nurse gives it through an IV (loading dose, then continuous drip).
2. It blocks V2 receptors so ADH can't signal the kidney.
3. Water-pores are not inserted; urine stays dilute.
4. You pee out large volumes of free water.
5. Blood sodium rises over 1–2 days.
6. Treatment lasts up to 4 days, then you switch to pills or other therapy.

SAFETY WATCH

MUST be used in the ICU or monitored bed with sodium checks every 4–6 hours—can raise sodium too fast. · Watch the IV site closely—can cause irritation or phlebitis; needs a large vein or central line. · Can drop blood pressure (from V1a blockage), especially if you're also on blood pressure medicines. · Do NOT use if you have liver disease (can worsen liver damage). · Many drug interactions—tell the team every medicine you take. · Very expensive; insurance may require pre-approval.

Hypertonic saline (3% sodium chloride IV) · Intravenous electrolyte solution

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)).

1. The nurse gives it through an IV, either as a slow drip or as small, repeated 100 mL boluses.
2. Sodium enters your blood immediately.
3. Your blood becomes more concentrated (hypertonic).
4. Water shifts out of brain cells into the blood to balance the concentration.
5. Brain swelling goes down; seizures or confusion improve within hours.
6. Sodium level rises quickly (goal: 4–6 mEq/L in the first few hours, then STOP or slow down).

SAFETY WATCH

Only used in emergencies with severe symptoms (seizure, coma, altered mental status)—NOT for mild or chronic low sodium. · MUST be given in the ICU with sodium checks every 2–4 hours. · If sodium rises too fast, the team may give IV desmopressin (DDAVP) and free water to bring it back down—this is called 'overcorrection rescue.' · Can cause fluid overload in people with heart failure or kidney disease—watch for shortness of breath and swelling. · Burns the vein—needs a large IV or central line; can cause phlebitis. · Maximum safe rise: 8 mEq/L in 24 hours, 18 mEq/L in 48 hours.

Red flags

- 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

Nursing priorities

1. Check serum sodium level immediately and monitor it every 4 to 6 hours if abnormal. — 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. — 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. — 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.
4. Weigh the patient at the same time every day on the same scale, wearing similar clothes. — Daily weight is the most accurate way to track total body water—rapid weight loss means dehydration in diabetes insipidus; weight gain means water overload in SIADH; one liter of fluid equals one kilogram (2.2 pounds).
5. Monitor blood pressure, heart rate, and skin turgor (skin tenting) every 4 hours. — Diabetes insipidus causes dehydration, leading to low blood pressure, fast heart rate, and dry, tented skin; catching dehydration early prevents shock and allows time to replace fluids or start desmopressin.
6. Assess mental status and neurological signs (confusion, headache, muscle twitching, reflexes) every 2 to 4 hours. — Low sodium in SIADH causes brain cells to swell with water, leading to confusion, seizures, coma, and brain herniation (brain squeezing down); early detection allows careful, slow sodium correction to prevent permanent brain damage.
7. Enforce strict fluid restriction (usually 800 to 1000 mL per day) for SIADH patients, and explain why to the patient and family. — Limiting fluid intake is the main treatment for SIADH because the body is holding onto too much water and diluting sodium; restricting fluids allows sodium to rise slowly and safely back to normal.
8. Keep cold water or ice chips at the bedside for diabetes insipidus patients, and allow free access to fluids. — People with diabetes insipidus lose massive amounts of water and must drink constantly to avoid life-threatening dehydration; restricting fluids in these patients will cause shock and organ failure.

Sources

- 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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