

HHS • Hyperosmolar Hyperglycemic State

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THE STORY IN ONE SENTENCE

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“Hyperosmolar hyperglycemic state (HHS) — extremely high blood sugar with severe dehydration — happens mostly in type 2 diabetes when insulin runs low enough that sugar piles up but just enough remains to stop the body from burning fat for fuel (so no ketones). Always give fluids first to thin the blood, then start insulin slowly.”

Normal anatomy — the body's job

The pancreas releases insulin, a hormone that unlocks cells so sugar (glucose) can move from the bloodstream into muscle, fat, and liver cells for energy or storage. At the same time, the kidneys filter blood and normally reabsorb every bit of glucose back into the body — none is wasted in urine. This keeps blood sugar steady between 70 and 100 mg/dL, even after meals.

BODY'S JOB

Insulin's job is to keep blood sugar from climbing too high by letting cells pull sugar out of the blood. The kidneys' job is to reclaim sugar during filtering so the body does not lose fuel. Together, they keep blood sugar in a narrow, safe range so every organ — especially the brain, which runs only on glucose — gets a steady supply of energy.

1. You eat food → sugar enters the bloodstream.
2. The pancreas senses rising sugar → releases insulin into the blood.
3. Insulin binds to receptors on muscle, fat, and liver cells → unlocks the door so sugar flows inside.
4. Blood sugar drops back to normal.

5. The kidneys filter blood continuously → capture and return every molecule of glucose (up to about 180 mg/dL) so none is lost in urine.
6. Result: blood sugar stays steady, cells get fuel, brain stays clear and awake.

What breaks

In type 2 diabetes, cells become resistant to insulin — the key still turns, but the lock is sticky, so less sugar gets inside. The pancreas tries to compensate by making more insulin, but over time it wears out and insulin levels drop. When a stressor hits (infection, heart attack, stroke, or stopping diabetes medicine), the body releases stress hormones like cortisol and epinephrine that push blood sugar even higher and block insulin further. Sugar climbs above 600 mg/dL. The kidneys try to dump the excess into urine, but sugar drags water with it by osmosis (water follows sugar), so the person urinates gallons and becomes severely dehydrated. Blood volume shrinks, blood thickens, and the kidneys — now low on blood flow — can no longer filter effectively, so sugar stays trapped in the blood, climbing higher still.

CLINICAL TERM

Severe insulin resistance plus relative insulin deficiency, triggered by physiologic stress (infection, heart attack (myocardial infarction), stroke (cerebrovascular accident), nonadherence to medication, or corticosteroid use). Plasma glucose exceeds 600 mg/dL (often >1000 mg/dL). Glucosuria causes osmotic diuresis with losses of 8–12 liters of free water. Serum osmolality rises above 320 mOsm/kg. Dehydration reduces glomerular filtration rate, trapping glucose in the circulation and perpetuating high blood sugar (hyperglycemia). Enough residual insulin remains to suppress lipolysis and ketogenesis, so serum ketones remain low or absent and pH stays above 7.30 (distinguishing HHS from DKA).

How the body compensates

The body tries to protect blood pressure and organ blood flow (perfusion) even as it loses massive fluid volume. The sympathetic nervous system (the body's fight-or-flight system) releases epinephrine and norepinephrine to constrict blood vessels and raise heart rate, keeping blood pressure from collapsing. The renin-angiotensin-aldosterone system (RAAS) in the kidneys holds onto sodium and water to defend blood volume. But these responses backfire: epinephrine and cortisol push blood sugar even higher, and holding onto sodium

without enough free water makes the blood even thicker. Vital signs may look deceptively stable until the person suddenly crashes into shock, coma, or multi-organ failure.

1. {'step': 'The sympathetic nervous system fires — heart rate climbs, blood vessels squeeze tight.', 'why': 'Baroreceptors (pressure sensors in the carotid arteries and aorta) detect falling blood volume and signal the brainstem to release epinephrine and norepinephrine, raising heart rate and constricting vessels to keep blood pressure up and blood flowing to the brain and heart.'}
2. {'step': 'The adrenal glands (small glands on top of the kidneys) pour out cortisol, the main stress hormone.', 'why': 'Cortisol tells the liver to make more glucose and blocks insulin's action even further, worsening high blood sugar (hyperglycemia) — the body thinks it is starving and tries to raise fuel, not knowing that the real problem is too much sugar already.'}
3. {'step': 'The kidneys activate the renin-angiotensin-aldosterone system (RAAS) to hold onto sodium and water.', 'why': 'Low blood volume triggers the juxtaglomerular cells in the kidneys to release renin, starting a hormone cascade that ends with aldosterone telling the kidneys to reabsorb sodium and water, trying to restore volume — but without free water intake, this just concentrates the blood further.'}
4. {'step': 'Blood pressure and heart rate may stay near-normal for hours or days — the dangerous "stable" phase.', 'why': 'Compensatory mechanisms are working overtime, masking how sick the person really is; the team may not realize the patient is on the edge of collapse until compensation suddenly fails.'}
5. {'step': 'The crash point: brain swelling from overly fast fluid replacement.', 'why': 'If IV fluids flood in too quickly, water rushes into brain cells faster than osmolality can equalize, causing cerebral swelling (edema) (brain swelling), seizures, coma, and death — the number-one caused by medical care (iatrogenic) (doctor-caused) complication of HHS treatment.'}
6. {'step': 'Acute kidney injury (AKI) or complete kidney shutdown.', 'why': 'Prolonged low blood flow starves the kidney tubules of oxygen (acute tubular tissue death (necrosis)), and they stop filtering — waste and potassium build up, threatening life.'}
7. {'step': 'Clot formation (Thrombosis) — blood clots form in legs, lungs, or brain.', 'why': 'Thick, concentrated blood flows slowly and clots easily; dehydration raises hematocrit and platelet stickiness, triggering a clot in a deep leg vein (deep vein thrombosis) (DVT), pulmonary embolism (a clot lodging in a lung artery) (PE), or stroke.'}
8. {'step': 'Severe low potassium (hypokalemia) (dangerously low potassium).', 'why': 'High glucose and osmotic diuresis pull potassium out of cells and dump it in urine; then, when insulin is started, it drives potassium back into cells, dropping blood potassium even lower — this can stop the heart.'}

9. {'step': 'Watch trends — glucose dropping faster than 100 mg/dL per hour, sodium rising as glucose falls, worsening confusion — not just a single snapshot.', 'why': 'HHS evolves over hours to days; improvement or deterioration shows up in the direction and speed of lab changes, not in any one number — tracking the trajectory tells you if your treatment is working or causing harm.'}

Domino effect

1. Insulin resistance plus a trigger (infection, heart attack, stroke, or missed medicine) causes blood sugar to skyrocket above 600 mg/dL. — The pancreas cannot make enough insulin to overcome the resistance, and stress hormones — cortisol and epinephrine — tell the liver to dump even more glucose into the blood while blocking insulin's action, so sugar piles up with nowhere to go.
2. The kidneys try to dump the excess sugar into urine, and sugar drags water with it by osmosis. — When blood sugar exceeds the kidney's reabsorption limit (about 180 mg/dL), glucose spills into the urine; because sugar is osmotically active, it pulls water molecules along, causing massive urine output (polyuria) — often 8 to 12 liters of fluid lost over days.
3. Severe dehydration sets in — the person loses so much water that blood volume shrinks and blood becomes thick, concentrated, and syrupy. — Water leaves the bloodstream faster than it can be replaced by drinking (especially in elderly, confused, or bed-bound patients who cannot access fluids), so the remaining blood has the same amount of salt, sugar, and protein in far less liquid — like reducing a sauce on the stove.
4. Serum osmolality climbs above 320 mOsm/kg, and brain cells shrink. — Osmolality measures the concentration of particles in blood; when it rises, water is pulled out of brain cells by osmosis to try to balance the concentration, shrinking the cells and disrupting electrical signaling — this causes confusion, lethargy, seizures, or coma.
5. The kidneys, now starved of blood flow, can no longer filter effectively, so they trap glucose in the circulation instead of clearing it. — Dehydration drops blood pressure and flow to the kidneys (prerenal waste buildup in the blood (azotemia)); the glomerular filtration rate falls, so the kidneys cannot dump glucose into urine anymore — sugar stays in the blood, climbing even higher in a vicious cycle.
6. Despite extreme high blood sugar (hyperglycemia), there is no major acid buildup (pH stays above 7.30) and only trace or absent ketones. — Enough insulin is still present to suppress hormone-sensitive lipase in fat cells, so the body does not break down fat into free fatty acids and convert them into ketones (acetoacetate, beta-hydroxybutyrate) — this distinguishes HHS from diabetic ketoacidosis (the acid crisis of missing insulin) (DKA), where insulin is nearly zero and ketoacid production is massive.

7. Thick blood clots easily — risk of a clot in a deep leg vein (deep vein thrombosis) (DVT), pulmonary embolism (a clot lodging in a lung artery) (PE), and stroke spikes. — Dehydration raises hematocrit (the fraction of blood that is red cells), increases blood viscosity (thickness), and slows flow, especially in the legs; slow, thick blood plus sticky platelets and clotting factors create the perfect setup for clots to form and break off, traveling to the lungs or brain.
8. Treatment starts with IV normal saline to restore volume and thin the blood, then switches to half-normal saline once blood pressure stabilizes. — Fluids must go in gently — 1 to 1.5 liters in the first hour, then 250–500 mL/hr — to avoid cerebral swelling (edema) (brain swelling from water rushing into cells too fast); normal saline (0.9% NaCl) replaces volume without dropping osmolality too quickly, then half-normal saline (0.45% NaCl) replaces free water as osmolality falls.
9. Low-dose IV insulin is started only after fluids begin and potassium is confirmed above 3.3 mEq/L, with a goal of lowering glucose by 50–70 mg/dL per hour. — Insulin without fluids first can drop blood pressure (because insulin dilates vessels) and worsen shock; insulin also drives potassium from blood into cells, so if potassium is already low, insulin can trigger fatal arrhythmias — always replace potassium before starting insulin, and never drop glucose faster than 100 mg/dL/hr or osmolality faster than 3 mOsm/kg/hr to prevent cerebral swelling (edema).

Symptoms · what the patient feels, and why

- Extreme thirst and dry mouth: When blood sugar climbs very high, the kidneys try to flush out the extra sugar by pulling water from the body into the urine. This drains water from tissues and blood, so the body sends a powerful thirst signal to get you to drink and replace what's being lost.
- Peeing much more than usual: The kidneys spill the sky-high sugar into urine, and sugar drags water with it (like salt pulling water through a sponge). This makes huge amounts of urine, sometimes several liters a day, which washes water and salts out of the body.
- Severe dehydration—dry skin, sunken eyes, fast heart rate: Days of heavy urination drain so much water that blood volume drops. The skin loses its bounce, the eyes sink back, and the heart speeds up to try to pump the smaller, thicker blood volume around the body. Blood pressure may fall because there isn't enough fluid in the vessels.
- Confusion, drowsiness, or trouble waking up: The combination of very high blood sugar (often over 600 mg/dL) and severe dehydration makes the blood thick and sludgy. Brain cells shrink when water is pulled out by the high sugar, and the sluggish blood can't deliver enough oxygen and nutrients. This disrupts the brain's electrical signals, causing confusion, sleepiness, or even coma.

- **Weakness and fatigue:** Without enough insulin, sugar piles up in the blood but can't get into muscle cells to power them. At the same time, dehydration lowers blood pressure and oxygen delivery. Muscles feel heavy and weak, and the whole body runs low on energy—like a car with a full gas tank but a broken fuel pump.
- **Warm, flushed skin with no sweating:** Severe dehydration shuts down sweat production because the body can't spare any more water. The skin feels warm and dry to the touch, and the person may run a mild fever simply from the stress and dehydration, not from infection.
- **Nausea or belly pain:** High blood sugar and dehydration slow down the stomach and gut. Food sits longer, and the gut's normal rhythm is thrown off, causing nausea. Severe dehydration can also trigger cramping pain as the intestines struggle to move contents along with less fluid.
- **Vision changes or blurry sight:** Very high blood sugar pulls water out of the lens of the eye, changing its shape and bending light differently. This makes vision blurry or doubled. The problem usually reverses once blood sugar and hydration are brought back to normal.

Focused exam

- **Blood glucose ≥ 600 mg/dL:** In HHS, the kidneys cannot dump glucose fast enough, so it piles up in the blood. This is the hallmark number that defines HHS (≥ 600 mg/dL), not DKA (usually 250–350 mg/dL).
- **Severe dehydration:** dry mouth, cracked lips, very dry skin, sunken eyes: The body has lost a massive amount of water—often 8 to 12 liters. High blood glucose pulls water out of cells and into the blood, then the kidneys flush it out in the urine. What you see on exam is the result: the person is profoundly dehydrated.
- **Altered mental status:** confusion, lethargy, or even coma: Thick, concentrated blood (high osmolality) causes brain cells to shrink because water shifts out of them. The brain cannot work right when its cells are dehydrated. This is the key danger sign in HHS.
- **A fast heart rate (Tachycardia)** (fast heart rate, often > 100 bpm) and weak pulse: The heart is trying to pump thick, low-volume blood around the body. It beats faster to keep oxygen moving, but each beat is weak because there is not enough fluid in the circulation (low stroke volume).
- **Serum osmolality ≥ 320 mOsm/kg:** Osmolality measures how concentrated the blood is. Water left the blood to follow glucose into the urine, but sodium and other particles stayed behind, making the blood thick like syrup. This number (≥ 320) is one of the diagnostic criteria for HHS.

- Minimal or absent ketones and $\text{pH} > 7.30$ (no severe too much acid in the blood (acidosis)): In HHS, there is still enough insulin circulating to stop the liver from making large amounts of ketones (acidic fat breakdown products). This is the key difference from DKA, where pH is usually < 7.30 and ketones are high. HHS is a water crisis, not an acid crisis.

Diagnostics · every test answers a question

- Serum glucose → Is the blood sugar high enough to define HHS?
- Serum osmolality (calculated or measured) → How thick and concentrated is the blood?
- Serum sodium (Na^+) → How concentrated is sodium in the blood, and how much water has been lost?
- Serum potassium (K^+) → Is there enough potassium in the blood to safely give insulin, or is it dangerously low?
- Arterial or venous blood gas (pH and bicarbonate) → Is there significant too much acid in the blood (acidosis), or is this HHS and not DKA?
- Serum or urine ketones → Are ketones present, suggesting DKA instead of pure HHS?
- Blood urea nitrogen (BUN) and creatinine → Are the kidneys still working, or has dehydration damaged them?
- Workup for the trigger: urinalysis and culture, chest X-ray, ECG, cardiac enzymes → What event triggered the HHS—infection, heart attack, stroke, or missed medications?

Treatment logic

- IV fluids: Start with 0.9% normal saline 1–1.5 L in the first hour, then 250–500 mL/h; switch to 0.45% half-normal saline once serum sodium is normal or high and blood pressure is stable → Replaces the 8–12 liters of water the body lost, thins out the thick blood, restores kidney blood flow so kidneys can filter glucose, and lowers blood sugar by dilution. Corrects dehydration and hyperosmolality—the main broken step in HHS.
- Check potassium before starting insulin; if $\text{K}^+ < 3.3$ mEq/L, hold insulin and give IV potassium (20–30 mEq/h) until K^+ is ≥ 3.3 ; maintain K^+ at 4–5 mEq/L during treatment → Prevents fatal cardiac arrhythmias (heart stopping). Insulin drives potassium from the blood into cells, so if you start insulin when potassium is already low, serum K^+ can drop to dangerous levels (< 2.5 mEq/L) and cause ventricular fibrillation or asystole.
- IV insulin: Start regular insulin infusion at 0.1 units/kg/h (or 0.14 units/kg/h without a bolus) AFTER fluids have started and potassium is ≥ 3.3 ; aim to lower glucose by 50–70 mg/dL per hour → Tells muscle and fat cells to take glucose out of the blood and into cells (via GLUT4 transporters) and stops the liver from making new glucose (blocks gluconeogenesis). Lowers blood sugar slowly and safely—rapid correction can cause cerebral swelling (edema) (brain swelling).

- Add 5% dextrose to IV fluids once glucose reaches 250–300 mg/dL, and continue insulin to normalize osmolality and mental status → Prevents blood sugar from dropping too low (hypoglycemia) while you continue insulin to fix the hyperosmolality and wake up the brain. The goal is not just lower glucose—it is normal osmolality (< 315 mOsm/kg) and clear thinking.
- Find and treat the trigger: antibiotics for infection (UTI, pneumonia, sepsis), aspirin and cardiac catheterization for MI, adjust or restart home insulin if doses were missed → Removes the stress that overwhelmed the body's insulin reserve. Infection, heart attack, and stroke all raise cortisol (stress hormone) and epinephrine, which block insulin and raise blood sugar. Treating the trigger stops the vicious cycle.
- Monitor glucose, sodium, potassium, and osmolality every 1–2 hours initially, then every 2–4 hours; track mental status and urine output closely → Tells you if fluids are working (osmolality dropping, mental status improving), if potassium is staying safe (4–5 mEq/L), and if glucose is falling at the right speed (50–70 mg/dL/h). Confusion clearing is the best sign that the brain is getting enough water and oxygen.
- Transition to subcutaneous insulin once glucose is < 250 mg/dL, mental status is normal, the person can eat and drink, and the trigger is controlled; overlap SC and IV insulin by 1–2 hours to avoid rebound high blood sugar (hyperglycemia) → Maintains stable blood sugar outside the ICU and prevents recurrence. Long-acting basal insulin (glargine, detemir, degludec) controls baseline glucose, and rapid-acting insulin (lispro, aspart) covers meals.

Medications

Intravenous fluids (normal saline 0.9% initially, then 0.45% saline) ·

Crystalloid fluid replacement

HHS causes severe dehydration—the body loses huge amounts of water because sky-high blood sugar acts like a sponge in the bloodstream, pulling water out of cells and flushing it into the urine. IV fluids refill the pipes (blood vessels), restore blood volume, and bring down the dangerously thick, syrupy blood. Normal saline (salt water that matches the body's salt level) goes in first to rescue blood pressure and kidney flow. Once sodium starts dropping toward normal, we switch to half-normal saline (gentler, less salty) to keep refilling without overshooting sodium levels.

1. Start with 1–1.5 liters per hour of 0.9% normal saline in the first hour to rapidly expand blood volume and stabilize blood pressure.
2. Continue normal saline at 250–500 mL per hour if sodium is normal or low; switch to 0.45% saline if sodium is high (above 150) to avoid worsening salt imbalance.
3. Fluids dilute the glucose concentration in blood, helping lower blood sugar without insulin at first.

4. As blood volume rises, kidney blood flow improves, allowing the kidneys to filter and dump excess sugar into urine.
5. Total fluid replacement in the first 24 hours is often 6–9 liters, guided by urine output, blood pressure, and heart/kidney function.

SAFETY WATCH

Watch for fluid overload, especially in older adults or people with heart failure—listen for lung crackles (fluid backing up into the lungs) and check oxygen levels. · Track sodium every 2–4 hours; if it rises despite fluids, switch to half-normal saline to avoid high sodium (hypernatremia) (dangerously high sodium). · Monitor urine output; if kidneys aren't making urine despite fluids, suspect kidney injury or heart failure and slow the infusion rate. · Avoid giving too much fluid too fast in the first few hours, which can rarely trigger cerebral swelling (edema) (brain swelling), though this is much less common in HHS than in DKA.

Potassium chloride (IV or oral potassium replacement) · Electrolyte replacement

Even though initial blood tests often show normal or even high potassium, the body's total potassium stores are severely depleted because high blood sugar and dehydration have flushed potassium out through the urine. Once insulin and fluids start working, potassium rushes from blood into cells, and blood potassium can drop dangerously low (hypokalemia), risking fatal heart rhythm problems. Replacing potassium before or alongside insulin keeps blood levels safe while cells refill their stores.

1. Check potassium before starting insulin; do NOT give insulin if potassium is below 3.3 mEq/L (risk of life-threatening arrhythmia).
2. If potassium is low (below 3.3), give 20–30 mEq per hour of IV potassium chloride until it rises above 3.3, then start insulin.
3. If potassium is normal (3.3–5.2), add 20–40 mEq of potassium chloride to each liter of IV fluids to prevent a drop when insulin begins.
4. If potassium is high (above 5.2) initially, hold potassium replacement but recheck every 2 hours—it will drop once insulin and fluids start, and replacement will be needed.
5. Continue replacing potassium to keep blood levels between 4 and 5 mEq/L throughout treatment.

SAFETY WATCH

Never give potassium faster than 10–20 mEq per hour through a peripheral IV (can burn veins and cause severe pain); use a central line if faster replacement is needed. · Recheck potassium every 2–4 hours during treatment; both too low and too high can cause deadly heart arrhythmias. · Watch the heart monitor (EKG) for peaked T-waves (potassium too high) or flattened T-waves and U-waves (potassium too low). · Use extreme caution if the patient has kidney failure—potassium can build up quickly and stop the heart.

Regular insulin (IV continuous infusion) · Rapid-acting insulin hormone replacement

In HHS, the body either makes too little insulin (in type 2 diabetes stressed by infection, heart attack, or other triggers) or cells resist insulin's signal, so glucose piles up in the blood instead of entering cells for energy. The liver also keeps dumping more glucose into the bloodstream because it doesn't sense insulin's 'stop' signal. IV regular insulin delivers the missing hormone directly into the bloodstream, unlocking cell doors to let glucose in, shutting off the liver's sugar factory, and gradually lowering blood glucose back toward normal.

1. Wait until IV fluids have started and potassium is safely above 3.3 mEq/L before giving insulin—fluids alone drop glucose initially, and insulin without potassium replacement can stop the heart.
2. Start continuous IV regular insulin at 0.1 units per kilogram per hour (typical adult dose 5–10 units/hour).
3. Expect blood glucose to fall 50–70 mg/dL per hour; if it drops faster, lower the insulin rate to avoid overshooting.
4. Once glucose reaches 250–300 mg/dL, cut the insulin rate in half and add dextrose (sugar water) to the IV fluids to keep glucose steady while continuing to clear the hyperosmolar state.
5. Keep insulin running until osmolality normalizes and the patient is mentally clear, then transition to subcutaneous insulin injections when the patient is stable and able to eat.

SAFETY WATCH

Check blood glucose every hour during the infusion; dropping too fast (more than 100 mg/dL per hour) can cause brain swelling (cerebral swelling (edema)), though this is rare in adults with HHS. · Never stop IV insulin abruptly—glucose will rebound and the metabolic crisis can restart; always overlap with subcutaneous insulin for 1–2 hours before stopping the drip. · Watch for signs of low blood sugar (hypoglycemia) if insulin rate isn't adjusted when glucose nears 250 mg/dL: shakiness, sweating, confusion, fast heartbeat. · Monitor potassium closely—insulin drives potassium into cells and can cause dangerous drops in blood potassium if not replaced.

Dextrose 5% in IV fluids (added once glucose approaches 250–300 mg/dL) · Intravenous glucose supplementation

Once insulin brings blood sugar down near 250–300 mg/dL, the immediate danger is over, but the body is still severely dehydrated and osmolality (blood thickness) is still high. Stopping insulin too early lets glucose rebound. Adding dextrose (sugar water) to the IV allows insulin to keep running at a lower rate, clearing the thick blood and restoring normal brain and kidney function, without pushing glucose dangerously low.

1. When blood glucose drops to 250–300 mg/dL, switch IV fluids to 0.45% saline with 5% dextrose.
2. Cut the insulin infusion rate by half (e.g., from 6 units/hour to 3 units/hour) but keep it running.
3. Adjust dextrose concentration (5% vs. 10%) and insulin rate to hold glucose stable at 200–250 mg/dL for 12–24 hours while osmolality normalizes.
4. Continue this 'glucose clamp' until mental status clears and lab values (osmolality, sodium) return to near-normal.

SAFETY WATCH

Recheck blood glucose every 1–2 hours to make sure it stays in the safe zone of 200–250 mg/dL; adjust dextrose or insulin rate as needed. · Do NOT use dextrose if glucose is still above 300 mg/dL—it will worsen high blood sugar (hyperglycemia). · Watch for fluid overload, as adding dextrose continues high-volume IV hydration.

Red flags

- Confusion, extreme drowsiness, or can't be woken up—the brain is shutting down from dehydration and sky-high sugar; this can lead to coma and death without emergency treatment.

- Blood pressure dropping or heart racing over 120 beats per minute at rest—the blood volume is dangerously low and the heart is struggling to keep organs alive.
- Seizure or one-sided weakness—severe dehydration and thick blood can trigger a stroke or seizure, especially in older adults.

Nursing priorities

1. Start two large-bore IV lines and hang normal saline at 1 to 1.5 liters per hour immediately — Severe dehydration from osmotic diuresis (sugar pulling water into urine) causes shock and organ damage; rapid fluid replacement expands blood volume, improves blood pressure, and actually lowers glucose by dilution before insulin even starts
2. Check fingerstick glucose and send lab work for basic metabolic panel with sodium and potassium before giving any insulin — We must know the potassium level first because insulin pushes potassium into cells; giving insulin when potassium is already low (below 3.3 mEq/L) can cause life-threatening heart rhythms (arrhythmias) and cardiac arrest
3. Monitor mental status and neurologic checks every hour, documenting any confusion, sleepiness, or seizure activity — High serum osmolality (concentration of blood) shrinks brain cells and causes altered mental status; tracking changes tells us if treatment is working, and sudden worsening may signal cerebral swelling (edema) (brain swelling) from too-fast correction
4. Replace potassium through IV fluids if level is between 3.3 and 5.3 mEq/L, and hold insulin if potassium drops below 3.3 — Insulin therapy drives potassium from blood into cells, dropping serum potassium dangerously; maintaining normal potassium prevents fatal heart rhythm problems while we treat the high blood sugar
5. Start regular insulin infusion at 0.1 units per kilogram per hour only after fluids are running and potassium is safe — Fluids alone drop glucose 50 to 100 mg/dL in the first hour and prevent shock; starting insulin too early or too fast risks severe low potassium (hypokalemia) (low potassium) and can cause blood sugar to crash, triggering cerebral swelling (edema)
6. Measure intake and output hourly, watching for urine output at least 0.5 mL per kilogram per hour — Adequate urine output confirms kidneys are getting enough blood flow (perfusion) and shows rehydration is working; poor urine output despite fluids signals kidney injury or inadequate resuscitation requiring treatment adjustment
7. Switch IV fluid from normal saline to half-normal saline when serum sodium normalizes or reaches 145 to 150 mEq/L — Normal saline can cause high sodium (hypernatremia) (high sodium) as glucose falls and water moves back into cells; half-normal saline prevents dangerous sodium spikes while continuing rehydration and lowering osmolality safely

8. Investigate and treat the underlying trigger with cultures, chest X-ray, EKG, urinalysis, and appropriate antibiotics or cardiac care — HHS does not happen by itself; infection, heart attack, or medication problems caused it, and the patient will not recover or will have another crisis unless we find and fix the root cause

Sources

- American Diabetes Association Standards of Care in Diabetes 2024
- Endocrine Society Clinical Practice Guideline on Hyperglycemic Crises
- ADA/EASD Consensus Report on Hyperglycemic Emergencies
- UpToDate Clinical Review: Hyperosmolar Hyperglycemic State

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