

Adrenal Crisis

Adrenal crisis means cortisol drops so low that blood vessels can no longer tighten properly and blood sugar falls dangerously low, so blood pressure crashes even when doctors give fluids and strong medicines that normally squeeze vessels.

THE ONE-LINE TRUTH

Cortisol helps blood vessels squeeze when you are stressed, tells your liver to make sugar for energy, and keeps your body balanced during illness or injury.

The chain — how it unfolds

1. The adrenal cortex is destroyed by autoimmune attack, tuberculosis, bleeding, or cancer, or the pituitary stops making ACTH because of a tumor, surgery, or chronic steroid use then sudden stop. Either way, cortisol production falls below the level needed to keep blood vessels responsive and blood sugar stable. — why: Primary failure destroys the cells that make cortisol. Secondary failure removes the ACTH signal that tells the adrenals to make cortisol. Both paths lead to the same result: circulating cortisol drops below the critical threshold needed for survival during stress.
2. Blood vessel walls lose their ability to tighten in response to adrenaline and noradrenaline, causing vasopressor-resistant low blood pressure (hypotension) (blood pressure that will not come up even with IV fluids and strong vessel-squeezing drugs). — why: Cortisol is required for the expression and function of alpha-1 adrenergic receptors (the protein locks on blood vessel muscle cells that respond to adrenaline). Without cortisol, adrenaline cannot make blood vessels constrict, so blood pressure falls and stays low no matter how much fluid or vasopressor medicine you give.
3. Blood glucose falls, sometimes to dangerously low levels (below 40–50 mg/dL), causing confusion, seizures, or loss of consciousness. — why: Cortisol tells the liver to make new glucose from amino acids (protein building blocks) and lactate (a byproduct of muscle work) through a process called gluconeogenesis. Cortisol also stops muscle and fat tissue from absorbing too much glucose from the blood. Without cortisol, the liver cannot make new glucose and tissues consume existing glucose faster than it can be replaced, so blood sugar plummets.

4. In primary adrenal insufficiency (when the adrenal glands themselves are destroyed), serum sodium falls (hyponatremia, often below 130 mEq/L) and serum potassium rises (hyperkalemia, often above 5.5 mEq/L). — why: When the adrenal cortex itself is destroyed—not just ACTH deficiency—aldosterone production also stops. Aldosterone normally tells the kidneys to hold onto sodium and let potassium leave in the urine. Without aldosterone, sodium is lost in the urine (causing low sodium in the blood) and potassium piles up in the blood. Secondary adrenal insufficiency (pituitary failure) does NOT cause high potassium (hyperkalemia) or severe low sodium (hyponatremia) because the adrenal glands can still make aldosterone in response to the renin-angiotensin system.
5. The patient develops severe fatigue, nausea, vomiting, abdominal pain (often mistaken for an acute abdomen), and altered mental status ranging from confusion to coma. — why: Low blood pressure reduces blood flow to the brain (cerebral blood flow (perfusion)), causing confusion and lethargy. Low blood sugar further impairs brain function. Electrolyte imbalances disrupt nerve and muscle signaling. Reduced blood flow to the gut causes nausea, vomiting, and severe belly pain that can mimic appendicitis or bowel obstruction.
6. In chronic primary adrenal insufficiency, hyperpigmentation (darkening of skin and the moist linings inside the mouth) appears, especially in sun-exposed areas, skin creases (palms, knuckles, elbows), scars, and the gums and tongue. — why: Loss of cortisol removes negative feedback on the pituitary, so ACTH levels rise very high (often 5-10 times normal). ACTH is made from the same precursor protein (POMC, pro-opiomelanocortin) as melanocyte-stimulating hormone (MSH). Elevated ACTH cross-reacts with melanocortin receptors on melanocytes (pigment-making cells), causing darkening of the skin. This sign does NOT appear in secondary adrenal insufficiency because ACTH is low, not high.
7. Every clinical finding—refractory low blood pressure (hypotension), low blood sugar (hypoglycemia), low sodium (hyponatremia), high potassium (hyperkalemia), hyperpigmentation—points backward to the same upstream break: cortisol deficiency. — why: None of these findings is random. Each one is a direct result of losing cortisol's jobs: keeping blood vessels responsive, keeping blood sugar up, and (in primary failure) keeping salt and potassium balanced. Trace every symptom back to cortisol, and the whole picture clicks.
8. The body's attempts to compensate—faster heart rate, releasing more adrenaline—are trying to maintain blood flow and balance, not the disease itself. — why: The symptoms and signs are the body trying to survive without cortisol. Replacing cortisol (hydrocortisone 100 mg IV immediately, then 50-100 mg IV every 6-8 hours) addresses the root cause and reverses all downstream problems together. Blood pressure rises, blood sugar stabilizes, electrolytes normalize, and the patient wakes up—often within hours.

Spot it — what you see, and why

- Blood pressure that stays dangerously low (systolic below 90 mmHg) even after fluids and medicines to raise blood pressure: Cortisol normally helps blood vessels tighten when the body releases adrenaline. Without cortisol, the vessels stay loose and floppy—like a garden hose with no water pressure—so blood pressure stays low no matter how much fluid or medicine is given.
- Blood sugar dangerously low (often below 50 mg/dL): Cortisol tells the liver to make new sugar from protein and other stored materials. Without cortisol, the liver shuts down this sugar factory, and the body's cells keep using up the little sugar that's left, so blood sugar drops fast.
- Sodium too low in the blood (below 135 mEq/L, often below 130 mEq/L): In primary adrenal failure (the glands themselves are destroyed), aldosterone—the hormone that tells kidneys to hold onto sodium—disappears, so kidneys dump sodium into the urine. In secondary failure (the brain signal is missing), cortisol loss stops the kidneys from getting rid of extra water, so the blood gets watered down and sodium concentration falls.
- Potassium too high in the blood (above 5.0 mEq/L, sometimes above 6.0 mEq/L): Aldosterone normally tells the kidneys to throw out potassium. When the adrenal glands are destroyed, aldosterone is gone, so potassium piles up in the blood like trash building up when the garbage truck stops coming.
- Extreme tiredness, muscle weakness, and confused thinking or reduced alertness: Low blood pressure means less blood reaches the brain. Low blood sugar starves brain cells of their main fuel. Scrambled sodium and potassium levels disrupt the electrical signals brain cells use to talk to each other. All three together make the brain slow down and the muscles feel heavy and weak.
- Nausea, vomiting, and severe belly pain (sometimes mistaken for a surgical emergency like appendicitis): Low blood pressure and messed-up electrolytes reduce blood flow to the intestines and make the gut muscles cramp and spasm, causing nausea, vomiting, and sharp belly pain that can look like a blockage or infection.

Red flags — act now

- Blood pressure below 90 mmHg (top number) that does not come up after 1–2 liters of IV fluid and blood-pressure medicines
- Confusion, extreme sleepiness, or unconsciousness, especially with blood sugar below 50 mg/dL
- Potassium above 6.0 mEq/L with EKG changes (tall pointy T waves, wide QRS), which can trigger a deadly heart rhythm

- Severe belly pain that looks like a surgical emergency (appendicitis, bowel obstruction), delaying the correct diagnosis
- Recent sudden stop of long-term steroid pills (prednisone, dexamethasone) in a patient now in shock

Treat it — in this order

1. Intravenous hydrocortisone 100 mg bolus (large dose all at once) immediately, then 50 mg every 6 hours (or 200 mg per day as a continuous drip) → Hydrocortisone replaces the missing cortisol instantly. It does three life-saving things: (1) it turns on receptors (alpha-1 adrenergic receptors) on blood vessel muscle so that stress hormones (like norepinephrine) can finally make vessels squeeze tight (vasoconstriction), which raises blood pressure; (2) it tells the liver to start making new sugar (gluconeogenesis), which raises blood glucose and feeds the brain and heart; and (3) it calms down the excessive inflammatory signals (cytokines) that can damage organs. At doses of 50 mg or more every 6 hours, hydrocortisone also acts like aldosterone (the salt-retaining hormone), helping the kidneys hold onto sodium and pull fluid back into the blood. This is the definitive, must-do treatment that saves lives in adrenal crisis (Endocrine Society 2016 guidelines).
2. Rapid intravenous infusion of 0.9 percent normal saline (isotonic salt water) 1 to 2 liters over the first hour, then 250 to 500 mL per hour after that. Add 5 percent dextrose (sugar water) if blood glucose is under 70 mg/dL. → Isotonic saline replaces the fluid and salt lost in the urine (from aldosterone deficiency) and from poor drinking. It fills up the blood vessels (increases intravascular volume), which increases the amount of blood returning to the heart (preload) and pumps more blood out to organs (improves cardiac output and tissue blood flow (perfusion)). Dextrose quickly corrects low blood sugar, giving the brain and heart the fuel they need. Volume expansion also dilutes the high potassium in the blood and helps the kidneys flush it out once blood flow is restored.
3. Find and treat the trigger (the precipitating stressor): start broad-spectrum antibiotics (antibiotics that kill many types of bacteria) right away if infection is suspected; clean out infected wounds (debridement); drain pockets of pus (abscesses); manage heart attack or blood clot in the lung; or restart steroid medicine if doses were missed → Removing or controlling the acute stress (infection, surgery, trauma, heart attack) lowers the body's demand for cortisol, reduces the flood of stress hormones, and stops the slide toward multi-organ failure. Treating infection or other acute illness lets the patient stabilize once you have replaced cortisol and fluids.

4. Correct severe high potassium (hyperkalemia) (potassium over 6.5 mEq/L or any level with dangerous EKG changes): give intravenous calcium gluconate 1 gram over 2 to 3 minutes to protect the heart, then insulin (10 units regular insulin IV) with dextrose (25 grams, or one ampule of D50) to shift potassium into cells, and consider sodium bicarbonate (50 mEq IV) if the patient is acidotic → Calcium stabilizes the heart muscle membrane so that high potassium does not trigger a fatal arrhythmia (it buys time but does not lower potassium). Insulin drives potassium from blood into cells (lowers serum potassium by 0.5 to 1.5 mEq/L within 15 to 30 minutes); dextrose is given with insulin to prevent low blood sugar. Bicarbonate also shifts potassium into cells if the blood is acidic. These measures rapidly reduce the immediate risk of cardiac arrest while the underlying cause (aldosterone deficiency) is being corrected with hydrocortisone and fluids.
5. Oral fludrocortisone acetate 0.1 mg once daily—only for primary adrenal insufficiency, started after the acute crisis is over and the patient can swallow pills → Fludrocortisone is a synthetic version of aldosterone. It tells the kidneys to hold onto sodium (reabsorb it from the urine back into the blood) and get rid of potassium (excrete it in urine). This fixes chronic low sodium and high potassium, helps keep blood pressure normal, and prevents future episodes of volume depletion and crisis in patients whose adrenal glands are destroyed.

Your top priorities at the bedside

1. Establish large-bore intravenous access (18-gauge or bigger) and infuse 0.9 percent normal saline 1 to 2 liters wide open over the first hour, then 250 to 500 mL per hour. — why: Severe dehydration and low blood volume cause the dangerously low blood pressure; fast IV fluid refills blood vessels and helps the heart pump blood to the brain and kidneys before organ damage occurs.
2. Administer intravenous hydrocortisone 100 mg bolus immediately, then prepare to give 50 mg every 6 hours or start a continuous drip of 200 mg over 24 hours as ordered. — why: The body has almost no cortisol, so blood vessels cannot squeeze properly and cells cannot make energy; replacing cortisol within minutes reverses shock and prevents death.
3. Check blood glucose at the bedside with a fingerstick meter right away, then every 30 to 60 minutes; if glucose is below 70 mg/dL, add 5 percent dextrose to the IV fluid. — why: Cortisol tells the liver to make glucose (sugar for energy); without cortisol the liver stops and blood sugar drops so low that the brain starves, causing confusion, seizures, or coma.