

Wernicke Encephalopathy

Always give thiamine BEFORE giving sugar (dextrose or IV fluids with glucose) to any patient at risk—alcoholism, malnutrition, vomiting, or refeeding—because sugar without thiamine can trigger or worsen Wernicke encephalopathy.

THE ONE-LINE TRUTH

Wernicke encephalopathy happens when the body runs out of thiamine (vitamin B1), usually from heavy alcohol use, poor eating, repeated vomiting, stomach surgery, or refeeding after starvation—and giving sugar without thiamine first makes it worse.

The chain — how it unfolds

1. Thiamine stores run out (from alcohol use disorder, malnutrition, hyperemesis, or malabsorption). — why: Chronic alcohol blocks thiamine absorption in the intestine, increases urinary loss, and impairs conversion of thiamine to its active form (thiamine pyrophosphate). Poor diet, repeated vomiting, or gastrointestinal surgery (bariatric surgery, gastrectomy) also prevent thiamine from getting into the body. Stores last only 18 days, so depletion happens fast.
2. Mitochondria cannot make ATP, so brain cells starve for energy. — why: Thiamine is the required co-factor for pyruvate dehydrogenase (converts pyruvate to acetyl-CoA), alpha-ketoglutarate dehydrogenase (runs the Krebs cycle), and transketolase (runs the pentose phosphate pathway). Without thiamine, all three pathways stop, ATP production drops by 60–80%, and neurons cannot power ion pumps, neurotransmitter synthesis, or membrane repair.
3. Giving IV dextrose or starting refeeding WITHOUT giving thiamine first triggers acute Wernicke encephalopathy. — why: A glucose load forces the few remaining thiamine molecules to be consumed instantly trying to process the flood of sugar. This sudden depletion crashes energy production in the most vulnerable brain regions (mammillary bodies, thalamus, periaqueductal gray, cerebellar vermis), and excitotoxicity and free-radical damage kill neurons within hours.

4. The patient develops the classic triad: confusion (encephalopathy), loss of coordination (ataxia) (wide-based, unsteady gait), and ophthalmoplegia or involuntary eye jerking (nystagmus) (eyes not moving together or jerking). — why: The medial thalamus and mammillary bodies (memory and alertness centers) die, causing confusion and disorientation. The cerebellar vermis (balance and coordination center) dies, causing loss of coordination (ataxia). The brainstem nuclei controlling cranial nerves III (oculomotor), IV (trochlear), and VI (abducens) die, causing eye movement problems—involuntary eye jerking (nystagmus) (rhythmic eye jerking, often horizontal) or ophthalmoplegia (paralysis of eye muscles, causing double vision or inability to look in certain directions).
5. Autonomic dysregulation causes hypothermia and low blood pressure (hypotension). — why: The brainstem autonomic centers that regulate body temperature and blood pressure need thiamine to function. When they fail, core temperature drops (often 35–36°C) and blood pressure falls, mimicking sepsis or shock—but fluids and pressors do not fix it; only thiamine does.
6. Peripheral nerves lose their myelin coating, causing neuropathy. — why: Schwann cells need thiamine to maintain the myelin sheath around peripheral nerves. Thiamine deficiency causes distal symmetric polyneuropathy—numbness, tingling, burning pain, and loss of ankle reflexes in a stocking-glove distribution. This is often called dry beriberi.
7. If thiamine is not given in time, confusion becomes permanent (Korsakoff syndrome). — why: Neurons in the mammillary bodies and medial thalamus die permanently, leaving the patient with severe anterograde amnesia (cannot form new memories), confabulation (makes up stories to fill memory gaps), apathy, and lack of insight. About 80% of untreated Wernicke patients progress to Korsakoff, and only 20% of those recover meaningful memory function even with thiamine.
8. The heart can fail (wet beriberi) or the patient can die from cardiovascular collapse. — why: The heart muscle has extremely high energy demand. Thiamine deficiency causes high-output heart failure (the heart pumps fast but weakly, causing warm skin, bounding pulses, wide pulse pressure, pulmonary edema (fluid flooding the lungs), and eventual cardiogenic shock). This is called wet beriberi, and it can be fatal within hours if thiamine is not given.

Spot it — what you see, and why

- Confusion or trouble thinking clearly: The thalamus (the brain's relay station that sorts incoming messages) and the mammillary bodies (memory hubs near the base of the brain) are starving for thiamine (vitamin B1), so they cannot process information or form memories properly.
- Unsteady walk or trouble balancing (ataxia): The cerebellum (the brain's balance and coordination center at the back) is damaged by thiamine shortage, so signals to the legs and trunk get scrambled and muscles lose smooth teamwork.

- Abnormal eye movements — eyes may drift, shake, or not move together (nystagmus or ophthalmoplegia): The brainstem nuclei (tiny control centers in the brainstem that steer the eye muscles) need thiamine to fire properly; without it, the eyes lose their coordinated aim and may jiggle or freeze in certain directions.
- Double vision: Damage to the nerves controlling eye muscles — especially the sixth cranial nerve (the abducens nerve, which pulls the eye outward) — means the two eyes cannot point at the same spot, so the brain sees two images.
- Droopy eyelid (ptosis): The third cranial nerve (the oculomotor nerve, which lifts the upper eyelid and moves the eye in most directions) is weakened by thiamine deficiency, so the lid falls and the eye cannot open fully.
- Low blood pressure when standing (orthostatic low blood pressure (hypotension)) or fast heart rate: The autonomic nervous system (the automatic pilot that adjusts blood vessel tightness and heart rate) is damaged, so blood pools in the legs when you stand and the heart races to try to keep enough blood reaching the brain.

Red flags — act now

- Confusion that gets worse quickly — brain swelling or bleeding may be starting
- Eyes that cannot move in all directions or pupils that are unequal — pressure on brainstem nerves is a medical emergency
- Body temperature below 95 °F (35 °C) or very slow heart rate — the brain's automatic controls are failing and the person may collapse
- Seizures or sudden loss of consciousness — the brain is in crisis and needs thiamine and support immediately
- Signs of bleeding in the brain (severe headache, vomiting, neck stiffness, vision changes) — rare but life-threatening complication of Wernicke encephalopathy

Treat it — in this order

1. IV thiamine 500 mg three times daily for 2-3 days, then 250 mg daily for 3-5 days, then 100 mg by mouth daily → Thiamine is the cofactor that lets the enzyme alpha-ketoglutarate dehydrogenase turn sugar into energy inside the mitochondria (the cell's power plants). Without thiamine, the brain cells in the mammillary bodies, thalamus, and cerebellum starve and misfire, causing confusion, eye jerking, and stumbling. High-dose IV thiamine floods the cells with the missing cofactor so energy production restarts and the misfiring stops.

2. IV magnesium sulfate 2 grams over 15–30 minutes, then 1–2 grams daily while giving thiamine → Magnesium is the mineral cofactor that thiamine-dependent enzymes (transketolase and alpha-ketoglutarate dehydrogenase) need to actually work inside the cell. Without magnesium, thiamine sits unused even if you give plenty of it. Replacing magnesium fixes the broken enzyme system so thiamine can do its job.
3. Do NOT give IV dextrose (sugar water) or oral glucose before thiamine is on board → Sugar floods the brain with demand for energy at the exact moment there is no thiamine to make that energy. The mitochondria try to burn the sugar, fail, and produce toxic lactate instead, which damages neurons faster. Giving thiamine first prevents this metabolic crash.
4. Treat alcohol withdrawal with benzodiazepines (lorazepam 1–2 mg IV every 6 hours or diazepam 5–10 mg IV every 6 hours, adjusted to CIWA score) and address the underlying alcohol use disorder with counseling and medication-assisted treatment → Stopping alcohol removes the root cause that depleted thiamine (alcohol blocks gut absorption and increases urinary loss). Benzodiazepines boost GABA (the brain's main calming signal) to prevent seizures and delirium tremens, which would add more metabolic stress and worsen brain injury.
5. Give balanced nutrition with thiamine 100 mg by mouth daily, plus B-complex vitamins (folate 1 mg, B12 1000 mcg, B6 50 mg), plus regular meals with adequate carbohydrate, protein, and fat → Normal food and other B vitamins support the brain's recovery by keeping all the fuel-making enzymes (not just the thiamine-dependent ones) running smoothly. This prevents relapse and helps repair other malnutrition damage (peripheral neuropathy, anemia).

Your top priorities at the bedside

1. Check if IV dextrose or oral glucose has been given or ordered, and hold it until after the first dose of thiamine 500 mg IV is infused — why: The brain needs thiamine to turn glucose into energy; giving sugar without thiamine forces the brain to use its last thiamine reserves and causes brain cells in the thalamus (memory center) and mammillary bodies (balance center) to swell and die within hours
2. Give thiamine 500 mg IV diluted in 100 mL normal saline over 30 minutes three times daily for at least 2 days before switching to lower doses — why: Oral thiamine absorbs poorly, especially in people who drink alcohol or vomit, and the blood level stays too low to reverse brain damage; IV thiamine at 500 mg reaches the brain quickly and saturates the damaged cells so they can start making energy again and stop dying

3. Give magnesium sulfate 2 grams IV over 15 to 30 minutes, then continue 1 to 2 grams IV daily while the patient receives thiamine — why: Thiamine cannot work inside cells without magnesium because magnesium activates the enzyme thiamine pyrophosphokinase that turns thiamine into its active form; alcohol use and malnutrition deplete magnesium, so replacing both together prevents treatment failure