

Brain Tumors and Neurologic Oncology Symptoms

Brain tumors — new weakness on one side, morning headache with vomiting, first-time seizure in an adult, or personality change that gets worse over weeks.

THE ONE-LINE TRUTH

A growing lump inside the skull causes trouble in three ways: it crushes or invades the brain tissue where it sits (causing weakness or speech problems in that exact spot), it takes up space and raises pressure inside the rigid skull (causing morning headaches and vomiting), and it irritates nearby brain cells (triggering seizures).

The chain — how it unfolds

1. A tumor starts growing in the brain — either from brain cells themselves (glioma from glial support cells, meningioma from the meninges, primary CNS lymphoma from lymphocytes in the brain) or from cancer cells that traveled through the bloodstream from another organ (most often lung, breast, melanoma, kidney, or colon) and lodged in the brain's blood vessels. — why: The tumor is a clump of cells dividing out of control. It takes up space, pushes into or invades healthy brain tissue, and releases factors that break down the blood-brain barrier (the tight junctions between endothelial cells lining brain capillaries that normally keep the brain's environment stable). Fluid leaks out of the damaged vessels into the extracellular space in the white matter, forming vasogenic swelling (edema) (swelling from leaked fluid) like a bruise around the tumor. The total mass (tumor plus swelling) keeps getting bigger inside a skull that cannot expand.

2. The growing mass destroys or compresses the exact part of the brain where it sits, causing a focal neurologic deficit — weakness or numbness on one side of the body (hemiparesis or hemisensory loss), loss of language ability (aphasia) (trouble speaking or understanding language), hemianopsia (vision loss in half the visual field in both eyes), loss of coordination (ataxia) (loss of smooth coordination), or personality change — depending on which brain region is damaged. — why: Each part of the brain has one main job, so damage to that spot causes a specific, predictable problem. Left motor cortex damage causes right arm and leg weakness (contralateral weakness on one side (hemiparesis), because the motor fibers cross in the medulla at the pyramidal decussation). Left frontal or temporal lobe damage (in the dominant hemisphere for language, which is the left side in about 95 percent of right-handed people and 70 percent of left-handed people) causes Broca loss of language ability (aphasia) (trouble speaking but understanding is okay) or Wernicke loss of language ability (fluent speech that makes no sense, and poor understanding). Occipital lobe damage causes homonymous hemianopsia (vision loss in the right or left half of both eyes' visual fields, because visual fibers from each side cross at the optic chiasm). Frontal lobe damage — especially prefrontal cortex — causes personality and judgment changes, disinhibition (saying or doing socially inappropriate things), apathy (not caring about things that used to matter), or poor executive function (trouble planning, organizing, or shifting tasks). Cerebellar damage causes loss of coordination (ataxia) (wide-based gait, trouble with finger-to-nose or heel-to-shin movements) and dysmetria (overshooting or undershooting when reaching for something).
3. The tumor and swelling (edema) take up space inside the closed skull, raising intracranial pressure and compressing blood vessels, which cuts down cerebral blood flow and oxygen delivery to the whole brain. — why: The skull is rigid and cannot stretch, so any extra volume (tumor, swelling (edema), or trapped CSF) squeezes everything else — brain tissue, arteries, veins — and pressure climbs. The Monroe-Kellie doctrine says the skull holds three things in a fixed total volume: brain tissue (about 80 percent), blood (about 10 percent), and CSF (about 10 percent). If one increases and the others cannot shrink enough to compensate, intracranial pressure goes up. When ICP gets high enough, it crushes small arteries and capillaries, starving brain cells of oxygen (ischemia). The cells swell from lack of energy (cytotoxic swelling, because the sodium-potassium pumps in cell membranes fail without ATP), which makes pressure rise even more in a vicious cycle. Cerebral blood flow (perfusion) pressure (mean arterial pressure minus intracranial pressure) drops, and once it falls below about 60 millimeters of mercury, brain cells start dying.

4. Rising intracranial pressure causes headache (worse in the morning, with coughing, bending, or lying flat), nausea, and projectile vomiting without feeling sick to your stomach first. The patient may also become drowsy, confused, or irritable as pressure climbs. — why: High intracranial pressure stretches pain-sensitive structures — the meninges (the brain's three-layer wrapping: dura, arachnoid, and pia) and the walls of large blood vessels like the middle meningeal artery — causing headache. It also compresses the area postrema and vomiting center (clusters of neurons in the medulla at the base of the fourth ventricle) that trigger nausea and vomiting. Morning headache happens because lying flat all night lets CSF and venous blood pool in the skull (gravity is not pulling them down anymore), raising pressure; the headache gets better after you stand up because gravity pulls fluid down into the spine and neck veins. Coughing, bending, or straining (Valsalva maneuver, like bearing down or lifting something heavy) spikes intracranial and venous pressure briefly, worsening the headache. As ICP rises further, it compresses the reticular activating system (a network of neurons running through the brainstem and thalamus that keeps you awake and alert), causing drowsiness, confusion, and eventually decreased level of consciousness.
5. The tumor irritates nearby cortical neurons, lowering the seizure threshold and causing the brain cells to fire all at once in a chaotic electrical storm — a seizure — which may start in one body part (focal seizure, like jerking of the right hand or face, tingling, or a strange smell or taste) or spread to the whole body (focal to bilateral tonic-clonic seizure, with loss of consciousness, stiffening, then rhythmic shaking of all limbs). — why: Tumor cells, vasogenic swelling (edema), disrupted blood-brain barrier, local starved blood flow (ischemia) (low oxygen), low glucose, and abnormal ion concentrations create an imbalance between excitation (driven by glutamate, the brain's main excitatory neurotransmitter, which opens channels that let sodium and calcium rush into neurons and make them fire) and inhibition (driven by GABA, gamma-aminobutyric acid, the brain's main calming signal, which opens chloride channels and makes neurons harder to fire). Cortical neurons become hyper-excitable. Once a few fire, the electrical wave spreads like a chain reaction through connected circuits via excitatory synapses, causing jerking movements (if the wave hits the motor cortex), sensory hallucinations (if it hits the sensory or visual cortex), or loss of awareness (if it spreads to both hemispheres and shuts down the thalamus and cortex). Seizures are especially common with slow-growing tumors (like low-grade gliomas, oligodendrogliomas, or meningiomas) that have time to irritate the cortex, and with tumors near the motor or sensory cortex.

6. If the tumor blocks the flow of cerebrospinal fluid through the brain's fluid channels (the lateral ventricles, third ventricle, aqueduct of Sylvius, or fourth ventricle), fluid backs up and enlarges the ventricles upstream (obstructive hydrocephalus), raising pressure even faster and causing rapid decline in alertness, worse headache, vomiting, and sometimes a triad of gait trouble, urinary incontinence, and dementia. — why: CSF is made continuously by the choroid plexus (a spongy tissue inside the ventricles) at about 20 milliliters per hour, or about 500 milliliters per day. It normally flows from the lateral ventricles (one in each hemisphere) → through the foramen of Monro (a narrow opening) → into the third ventricle → through the aqueduct of Sylvius (a tiny tunnel only 3 millimeters wide in the midbrain) → into the fourth ventricle → out through the foramina of Luschka (two side holes) and Magendie (one middle hole) → into the subarachnoid space around the brain and spinal cord, where it gets absorbed into the bloodstream through arachnoid granulations in the superior sagittal sinus. A tumor near any of these pathways acts like a dam, so fluid accumulates upstream. The ventricles swell. Intracranial pressure spikes quickly because the total volume inside the skull is climbing fast and there is nowhere for the fluid to go. Obstructive hydrocephalus is a neurosurgical emergency. The classic triad of normal-pressure hydrocephalus (gait apraxia, urinary incontinence, dementia — sometimes called wet, wobbly, and wacky) can happen with slower obstruction, but acute obstruction causes rapid stupor and herniation.

7. As intracranial pressure keeps rising, the brain starts to shift and herniate — soft brain tissue gets pushed down through the foramen magnum (the hole at the base of the skull where the spinal cord exits) or sideways under the falx cerebri (the membrane between the two hemispheres) or the tentorium cerebelli (the shelf between the cerebrum above and cerebellum below) — compressing the brainstem and causing life-threatening changes: irregular breathing (Cheyne-Stokes breathing with cycles of fast breaths then pauses, or uncoordinated (ataxic) breathing with no pattern), wide or unequal pupils, abnormal posturing (decerebrate or decorticate rigidity), and eventually coma or death. — why: Herniation syndromes occur when pressure gradients push brain tissue into spaces it does not belong. Uncal herniation (the medial edge of the temporal lobe, called the uncus, pushed down and sideways through the tentorial notch) compresses the third cranial nerve (the oculomotor nerve, which controls pupil constriction and most eye movements), causing a dilated, unreactive pupil on the same side (ipsilateral), and compresses the cerebral peduncle in the midbrain (which carries motor fibers down from the cortex), causing weakness on the opposite side (contralateral weakness on one side (hemiparesis)) and decreased consciousness as the reticular activating system shuts down. Tonsillar herniation (the cerebellar tonsils, the lowest part of the cerebellum, pushed down through the foramen magnum) compresses the medulla, which controls breathing (via the respiratory centers in the reticular formation) and heart rate (via the cardiac center and vagus nerve), causing irregular respirations (Cheyne-Stokes, then uncoordinated (ataxic), then pauses in breathing (apnea)), a slow heart rate (bradycardia), then respiratory and cardiac arrest. Central herniation (downward shift of both cerebral hemispheres and the diencephalon — the thalamus and hypothalamus — through the tentorial notch) compresses the brainstem in stages from top to bottom, causing progressive loss of consciousness, small reactive pupils (diencephalic stage) → midposition fixed pupils (midbrain stage), decorticate posturing (arms flexed at elbows, legs extended — diencephalic and upper midbrain injury) → decerebrate posturing (arms and legs extended and rotated inward — midbrain and pons injury), and then pauses in breathing and death (medulla injury). Without emergency treatment — surgery to remove the mass or debulk it, or medical intracranial pressure reduction with hyperosmolar therapy (mannitol 0.25–1 gram per kilogram IV or hypertonic saline 3 percent or 23.4 percent IV, which pulls water out of brain tissue into the bloodstream via osmosis), brief hyperventilation to a PaCO₂ of 30–35 millimeters of mercury (which constricts cerebral arteries and lowers blood volume in the skull), head-of-bed elevation to 30 degrees (to help venous drainage), and sometimes a ventriculostomy (a catheter placed into a lateral ventricle to drain CSF) — herniation is fatal within hours.

8. The only way to stop the dominoes is to find the tumor early (with brain MRI with gadolinium contrast, the gold standard for detecting and characterizing brain tumors — it shows the mass, the surrounding swelling (edema) as bright signal on T2 and FLAIR sequences, and abnormal enhancement where the blood-brain barrier is broken), lower the pressure (with dexamethasone, a corticosteroid that reduces vasogenic swelling by stabilizing the blood-brain barrier and decreasing vascular permeability, typically 10 milligrams IV load then 4 milligrams every 6 hours, or emergent surgery to remove the mass or place a ventriculostomy to drain CSF), treat seizures (with antiepileptic drugs, most commonly levetiracetam 500–1000 milligrams twice daily, which is preferred because it does not interact with chemotherapy and has fewer side effects than older agents like phenytoin), and attack the tumor itself with definitive therapy tailored to tumor type: maximal safe surgical resection (removing as much tumor as possible without damaging eloquent cortex), radiation therapy (external beam or stereotactic radiosurgery), chemotherapy (temozolomide for glioblastoma, high-dose methotrexate for primary CNS lymphoma), or targeted therapy (BRAF inhibitors for BRAF-mutant tumors, EGFR inhibitors) and immunotherapy (checkpoint inhibitors) depending on tumor histology and molecular markers (IDH mutation, MGMT promoter methylation, 1p/19q codeletion, BRAF mutation, EGFR amplification). — why: Once you remove or shrink the tumor, you eliminate the upstream cause — mass effect, vasogenic swelling (edema), and cortical irritation — so intracranial pressure drops, cerebral blood flow recovers, vasogenic swelling resolves as the blood-brain barrier heals, and seizure threshold normalizes as excitation-inhibition balance returns. Treating symptoms alone (analgesics like acetaminophen or opioids for headache, antiemetics like ondansetron for nausea) does not fix the root problem and lets the tumor keep growing toward herniation and death. Early diagnosis (prompted by new focal neurologic deficit like weakness on one side (hemiparesis) or loss of language ability (aphasia), new-onset seizure in an adult — especially over age 40 with no prior seizure history, or progressive headache with red flags: worse in the morning, worse with Valsalva, associated with vomiting, swelling of the optic nerve (papilledema) on fundoscopy, or focal deficit) and prompt neurosurgical consultation save lives and preserve brain function. Prognosis depends on tumor type (meningiomas and low-grade gliomas like pilocytic astrocytoma or oligodendroglioma have better outcomes with five-year survival often over 70 percent; glioblastoma has median survival around 15 months even with aggressive treatment; some metastases like breast and renal can respond well to therapy, while melanoma and lung often have poorer prognosis), location (tumors in eloquent cortex near motor strip, Broca or Wernicke area, or visual cortex are harder to resect completely without causing permanent deficit; tumors in the brainstem are very high risk and often inoperable), performance status (Karnofsky score above 70 predicts better tolerance of treatment), age (younger patients tolerate treatment better and have longer survival), and molecular features (in gliomas: IDH mutation, MGMT promoter methylation, and 1p/19q codeletion all predict better response to chemotherapy and longer survival, while EGFR

amplification and PTEN loss predict worse outcomes). Guidelines are from NCCN (National Comprehensive Cancer Network) for brain tumors, AAN (American Academy of Neurology) for seizure management, and Neurocritical Care Society for intracranial pressure management.

Spot it — what you see, and why

- Slow or long-lasting weakness on one side of the body: The tumor presses on or damages the motor cortex (the strip of brain that sends movement commands to muscles) on one side, so signals can't reach the opposite side of the body and muscles grow weak.
- Swelling of the optic nerve disk seen on eye exam (papilledema): The tumor or extra fluid around it raises pressure inside the rigid skull, pushing backward on the optic nerve where it enters each eye and making the nerve head swell like a balloon.
- Seizure (sudden shaking, staring, or loss of awareness): The tumor irritates brain cells around it, causing them to fire electrical signals in a wild, uncontrolled storm instead of the normal orderly pattern, and that storm spreads through nearby brain tissue.
- Headache that is worse in the morning or wakes you up: The tumor and swelling take up extra space inside the rigid skull, raising pressure; lying flat overnight lets fluid pool and pushes pressure even higher until you sit up.
- Nausea and vomiting, especially in the morning: High pressure inside the skull squeezes the brainstem (the stalk connecting brain to spinal cord), triggering the vomiting center there even when your stomach is perfectly fine.
- Trouble with balance, walking, or coordination: A tumor in or pressing on the cerebellum (the wrinkled ball at the back of the brain that fine-tunes balance and movement) scrambles the signals that keep you steady and smooth.

Red flags — act now

- Sudden severe headache or headache that is getting worse quickly
- New seizure in an adult with no history of seizures
- Sudden weakness, numbness, or loss of vision
- Confusion, drowsiness, or loss of consciousness
- Persistent vomiting with severe headache and no other cause

Treat it — in this order

1. Dexamethasone 4 to 10 mg by mouth or IV every 6 hours (corticosteroid) → Shrinks the fluid swelling (vasogenic swelling (edema)) around the tumor within hours to days by tightening leaky blood vessels in the brain, which lowers pressure inside the skull and eases headache, vomiting, and confusion immediately. Buys time for surgery or radiation to work.
2. Levetiracetam 500 to 1,000 mg twice daily or phenytoin 300 mg daily (anti-seizure medicine) → Stops the brain from misfiring and causing seizures when the tumor irritates nearby neurons. Levetiracetam calms overexcited neurons by binding to the SV2A protein on brain cells, blocking chaotic signaling. Prevents injury from falling, tongue biting, or loss of consciousness during a seizure.
3. Surgical resection (craniotomy to remove as much tumor as safely possible) → Takes the mass out of the skull, which drops pressure inside the head immediately and stops the tumor from squeezing brain tissue and blood vessels. Gives tissue for exact diagnosis and molecular markers. Extends survival in glioblastoma (median survival goes from 3 to 4 months without surgery to 12 to 15 months with surgery plus radiation and temozolomide, per Stupp protocol) and often cures meningiomas and some low-grade gliomas.
4. External beam radiation therapy (whole-brain or focal, 30 to 60 Gy total dose over 2 to 6 weeks) → Zaps tumor cells with high-energy X-rays, damaging their DNA so they cannot divide. Shrinks the mass over weeks to months, which lowers skull pressure over time. Standard after surgery for glioblastoma (60 Gy in 30 fractions, per Stupp protocol) and for multiple brain metastases (> 4 lesions, 30 Gy in 10 fractions for whole-brain radiation).
5. Stereotactic radiosurgery (gamma knife or cyberknife, single high-dose session of 15 to 24 Gy) → Delivers a single focused blast of radiation to small tumors (usually < 3 cm), sparing nearby brain. Shrinks or stops growth in 1 to 4 brain metastases with local control rates over 85 percent at one year. Done in one day without opening the skull, so recovery is faster than surgery.

Your top priorities at the bedside

1. Position the head of the bed at 30 degrees and keep the patient's head in midline neutral position, not turned to either side — why: Elevating the head lets gravity help drain cerebrospinal fluid (the clear fluid around the brain) and venous blood away from the brain, lowering intracranial pressure; turning the head to the side kinks the jugular veins in the neck and traps blood in the skull, raising pressure

2. Perform and document a full neurologic assessment every 2 to 4 hours using the Glasgow Coma Scale (a 15-point scoring system for eye opening, verbal response, and motor response) and checking pupil size, symmetry, and reaction to light — why: A falling Glasgow Coma Scale score or new pupil changes (one pupil larger than the other or sluggish to react) are early warning signs that intracranial pressure is rising and the brainstem is being compressed, requiring immediate physician notification and possible emergency surgery
3. Administer dexamethasone (a corticosteroid that reduces brain swelling) exactly on time every 6 hours and monitor blood glucose levels before meals and at bedtime — why: Dexamethasone shrinks vasogenic swelling (edema) (fluid leaking from abnormal blood vessels around the tumor) within hours and lowers intracranial pressure, but it also raises blood sugar by making cells resistant to insulin, so glucose must be checked and treated if above 180 mg/dL