

Brain Tumors and Neurologic Oncology Symptoms

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THE STORY IN ONE SENTENCE

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“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.”

Normal anatomy — the body's job

The brain is a soft organ made of billions of neurons (nerve cells that send electrical signals) and glial cells (support cells that hold neurons in place, feed them, and clean up waste). It sits inside a hard, closed skull that cannot expand. The brain has different neighborhoods. The frontal lobes at the front handle thinking, planning, judgment, and personality. The motor cortex (a strip running from ear to ear over the top) controls movement on the opposite side of your body. The sensory cortex (the strip right behind the motor cortex) feels touch, pain, and temperature on the opposite side. The temporal lobes on the sides handle memory, hearing, and language. The occipital lobes at the back process vision. The cerebellum (a wrinkled ball at the lower back) controls balance and smooth movements. The brainstem at the base (made of the midbrain, pons, and medulla) controls automatic functions like breathing, heart rate, and staying awake. Blood vessels run through every part, bringing oxygen and glucose (sugar fuel). A thin skin called the meninges wraps the whole brain in three layers. Cerebrospinal fluid (CSF, a clear salty water) flows around the brain and through four cavities inside it called ventricles, cushioning it like a water balloon. The skull keeps everything safe but also creates a closed box where pressure can build.

BODY'S JOB

The brain's job is to control every part of your body — moving your muscles, feeling touch and pain, seeing and hearing, thinking and remembering, breathing and keeping your heart beating. Each neighborhood does one main job, so damage to one spot causes a specific problem. For example, damage to the left motor cortex causes weakness on the right side of your body, because the left brain controls the right side and vice versa (called contralateral control). The skull and CSF cushion protect the brain from bumps. Blood vessels bring oxygen and glucose so neurons can fire nonstop — the brain uses about 20 percent of your body's oxygen even though it is only 2 percent of your weight. Normally, the amount of brain tissue, blood, and CSF stays balanced, so pressure inside the skull (intracranial pressure, or ICP) stays low and steady — around 5 to 15 millimeters of mercury in a healthy adult lying flat.

1. The skull is a rigid bone box that cannot stretch.
2. Inside, the brain fills almost all the space, with a thin layer of cushioning fluid (CSF) flowing around it and through four cavities (the lateral ventricles, third ventricle, and fourth ventricle) inside it.
3. The choroid plexus (special tissue inside the ventricles) makes about 500 milliliters of fresh CSF every day. The fluid flows from the lateral ventricles through narrow channels into the third ventricle, then through the aqueduct of Sylvius (a tiny tunnel) into the fourth ventricle, and out into the space around the brain and spinal cord, where it gets absorbed back into the bloodstream through one-way valves called arachnoid granulations.
4. Blood vessels (arteries bringing oxygen-rich blood in, veins draining old blood out) run through the brain like roads, delivering oxygen and glucose to every neuron and carrying away waste.
5. Each part of the brain has a specific job: frontal lobes for thinking and personality, motor and sensory cortex for moving and feeling, temporal lobes for memory and language, occipital lobes for vision, cerebellum for balance, brainstem for breathing and heart rate.
6. Normally, pressure inside the skull stays low and steady because the amount of brain, blood, and CSF is balanced — this is called the Monro-Kellie doctrine.
7. If anything extra (like a tumor or swelling) takes up space, pressure rises because the skull cannot expand to make room.

What breaks

A tumor is a lump of cells that grows out of control. It breaks the normal setup in three ways. First, it destroys or pushes aside healthy brain tissue right where it grows, so that part of the brain stops working. You get weakness, speech problems, or vision loss in the exact area that

part controlled. Second, it takes up space inside the closed skull, raising pressure on the whole brain. You get headache, vomiting, and eventually the brain can get pushed downward in a life-threatening shift called herniation. Third, it irritates nearby cortical neurons (the brain cells in the outer layer), making them fire chaotically all at once and trigger seizures. Around the tumor, the blood-brain barrier (the tight seal between blood vessels and brain tissue) breaks down, and fluid leaks out into the white matter (the wiring that connects brain regions) like water seeping out of a cracked pipe. That swelling is called vasogenic swelling (edema), and it takes up even more space and raises pressure further.

CLINICAL TERM

Primary brain tumors originate in the brain itself. Gliomas (including astrocytoma, glioblastoma, and oligodendroglioma) arise from glial cells (the support cells that make up most of the brain's bulk and hold neurons in place). Meningiomas grow from the meninges (the three-layer membrane wrapping the brain). Primary central nervous system lymphomas are cancers of lymphocytes (a type of white blood cell) in the brain. Metastatic brain tumors spread from cancers elsewhere — most commonly lung (especially small cell and non-small cell), breast, melanoma, renal cell carcinoma, and colorectal cancer — through the bloodstream. The tumor mass disrupts function by direct invasion and destruction of neural tissue (neurons and white-matter tracts), causing focal neurologic deficits matched to the anatomic location. It occupies space within the non-compliant skull, raising intracranial pressure and causing headache, nausea, vomiting, swelling of the optic nerve (papilledema) (swelling of the optic nerve head visible on fundoscopy, a sign of high ICP), and risk of herniation. It lowers the seizure threshold through irritation of cortical neurons and disruption of the balance between excitatory neurotransmitters (mainly glutamate) and inhibitory neurotransmitters (mainly GABA, the brain's main calming signal), provoking focal or generalized seizures. Peritumoral vasogenic swelling (edema) (disrupted blood-brain barrier allowing protein-rich fluid to leak into extracellular space in white matter) amplifies mass effect and pressure. Tumors near the ventricular system can obstruct cerebrospinal fluid flow, causing obstructive hydrocephalus and rapid ICP rise.

How the body compensates

At first, the brain tries to keep itself alive by shifting fluid out of the skull, slowing down CSF production, and squeezing blood out of veins to make room for the growing tumor. Blood pressure rises (the Cushing reflex — high blood pressure (hypertension) and a slow heart rate (bradycardia), a classic warning sign of dangerously high intracranial pressure) to force blood through compressed vessels, and the patient may look stable — alert, talking normally, near-normal vital signs — even though pressure is climbing inside. This compensation buys time but eventually fails. Once the brain has used up all its tricks (no more fluid to push out, no more

veins to squeeze), pressure spikes fast, cerebral blood flow drops, the brain swells further from starved blood flow (ischemia) (not enough oxygen), and the patient crashes into herniation, status epilepticus (a seizure that will not stop) (continuous seizure activity), or coma. The danger is that the patient looks okay until suddenly they do not. The team must catch the hidden rising pressure early — before the cliff — by watching for worsening headache, new vomiting, drowsiness, confusion, pupil changes (one pupil larger than the other or sluggish reaction to light), or new focal weakness.

1. {'what': 'Shift CSF and venous blood out of the skull', 'why': 'The brain pushes cerebrospinal fluid down into the spinal canal (which has some stretch) and squeezes blood out of the venous sinuses (the large veins that drain the brain, like the superior sagittal sinus) to free up space for the tumor. This keeps pressure normal for a while but only works until those reservoirs are empty.'}
2. {'what': 'Raise blood pressure to maintain cerebral blood flow (perfusion) (Cushing reflex)', 'why': 'As intracranial pressure climbs, the body raises systemic blood pressure to keep cerebral blood flow (perfusion) pressure high enough (above 60 millimeters of mercury) to push blood through compressed arteries and deliver oxygen to brain cells. The medulla (the lowest part of the brainstem) senses the drop in blood flow and sends signals to the heart and blood vessels. This reflex produces high blood pressure (hypertension) and a slow heart rate (bradycardia) (slow heart rate, often below 60 beats per minute) — a classic warning sign called the Cushing triad when you see it with irregular breathing.'}
3. {'what': 'Reduce CSF production and increase absorption', 'why': 'The choroid plexus (the tissue in the ventricles that makes CSF) slows down production, and the arachnoid granulations (the one-way valves in the superior sagittal sinus that absorb CSF back into the bloodstream) speed up absorption to lower fluid volume inside the skull. This temporary fix delays but does not stop rising pressure.'}
4. {'what': 'Maintain alertness and near-normal exam — until reserves run out', 'why': 'The patient may stay awake, talk normally, and have near-normal vital signs even as intracranial pressure climbs, because compensation is working hard behind the scenes. This stable appearance is deceptive and hides how close the patient is to decompensation. Once compensation fails — no more room to shift fluid, blood pressure cannot climb high enough, cerebral blood flow (perfusion) drops — the crash is sudden and hard to reverse.'}

5. {'what': 'Watch for the crash point: worsening headache, vomiting, drowsiness, confusion, pupil asymmetry, or posturing', 'why': 'Once compensation fails, pressure spikes abruptly, cutting off blood flow (ischemia spreads, causing more swelling in a vicious cycle), shifting the brain downward (herniation), or triggering nonstop seizures (status epilepticus). At that point the window to reverse damage closes fast — minutes to hours. You must catch the trend before the cliff by recognizing early warning signs: headache getting worse over days to weeks, new projectile vomiting (forceful, no nausea warning), increased sleepiness or confusion, one pupil dilating or reacting slowly to light (sign of third cranial nerve compression from uncal herniation), or abnormal posturing (decerebrate posturing with arms and legs extended rigid, or decorticate posturing with arms flexed and legs extended — both signs of brainstem compression).'} }
6. {'what': 'Recognize that stability on a downhill slope is not true stability', 'why': 'A patient who looks stable today but has new or worsening symptoms this week (worse headache, new weakness, first seizure, personality change, increased drowsiness) is decompensating. Trend over time matters more than a single snapshot. Serial neuro checks (level of consciousness using the Glasgow Coma Scale, pupil size and reaction to light, motor strength in all four limbs, speech and language) and close monitoring catch the slow drift before the sudden drop.'} }

Domino effect

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. — 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. — 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. — 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. — 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). — 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-excitabile. 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. — 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. — 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). — 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.

Symptoms · what the patient feels, 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.
- Changes in personality, mood, judgment, or thinking: A tumor in the frontal lobe (the front third of the brain that handles planning, impulse control, and social behavior) damages or compresses those circuits, so personality and thinking shift.
- Vision changes such as blurry vision, double vision, or losing part of your field of view: The tumor presses on the optic nerve (the cable from eye to brain), the optic chiasm (where the optic nerves cross), or the occipital lobe (the back of the brain that decodes images), blocking or scrambling visual signals.
- Trouble speaking or understanding words: A tumor in the language areas (usually in the left side of the brain, near the temple and forehead in spots called Broca's area and Wernicke's area) damages the circuits that turn thoughts into speech or decode incoming words.
- Numbness or tingling on one side of the body: The tumor presses on the sensory cortex (the strip of brain that receives touch, pain, and position signals) on one side, so signals from the opposite side of the body can't get through normally.

Focused exam

- Weakness or numbness in one arm, one leg, or one side of the face — not both sides at once: A tumor grows in one spot and squeezes the slice of brain that controls the opposite side of the body. The motor cortex (the strip of brain that runs movement commands down the body) on the left side controls the right arm and leg, and vice versa. If the tumor presses on the left motor cortex, the right side goes weak. The pattern shows you exactly where the tumor sits.
- Headache that is worse in the morning, worse when you bend over or cough, and eases when you sit up: The skull is a closed box. When a tumor and the fluid swelling around it take up space, pressure inside the head climbs. Lying flat overnight lets pressure build because fluid cannot drain; sitting up lets gravity pull some fluid downward and drops the pressure. This pattern means raised intracranial pressure — the main warning that the brain is running out of room.
- Vomiting without belly pain, especially first thing in the morning: High pressure inside the skull squeezes the area postrema and nucleus tractus solitarius in the medulla (the lower part of the brainstem that triggers vomiting). The vomit does not come from a stomach problem — it is the brain's alarm that pressure is dangerously high. Morning vomiting means pressure climbed overnight while lying flat.
- Seizure — jerking, stiffening, staring spell, or loss of consciousness: The tumor irritates nearby neurons like a splinter irritates skin. Irritated brain cells fire all at once in a chaotic wave, causing a seizure. A first seizure in an adult over age 50 is a brain tumor until proven otherwise — seizures are often the very first sign that brings the person to the hospital.
- Swelling of the optic nerve (Papilledema) — the optic disc (where the nerve leaves the back of the eye) is swollen and blurred when examined with an ophthalmoscope: The optic nerve passes through a bony tunnel from the brain to the eye. High brain pressure squeezes cerebrospinal fluid into the nerve sheath and pushes the disc forward, making it swell. Seeing swelling of the optic nerve (papilledema) proves that pressure has been high for days to weeks — it is visible proof of raised intracranial pressure.
- Memory loss, confusion, or personality change — acting totally unlike themselves: If the tumor sits in the frontal lobe (the front of the brain that handles planning, judgment, and personality) or the temporal lobe (the side of the brain that stores memory and emotion), it damages the wiring that makes you 'you.' The person may not notice the change — family and friends see it first.

Diagnostics · every test answers a question

- MRI brain with and without gadolinium contrast → Is there a mass inside the skull, where exactly is it, and how big is it?

- CT head without contrast (if MRI is not available or you need an answer in minutes) → Is there a mass, bleeding, or dangerous pressure inside the skull right now?
- Chest X-ray or CT chest → Did the brain tumor spread from lung cancer?
- Tumor biopsy or surgical pathology (tissue sample from surgery) → What exact type of tumor is this, and what are its molecular markers?
- Serum tumor markers if cancer origin is unknown (CEA, CA 19-9, PSA, CA-125) → If the brain tumor spread from cancer elsewhere, where did the original cancer start?
- Complete blood count (CBC) with differential → Is the bone marrow making enough blood cells, or is chemotherapy or cancer affecting it?
- Basic metabolic panel (BMP) including sodium → Is brain swelling or tumor pressing on the pituitary gland causing salt and water imbalance?
- Liver function tests (AST, ALT, bilirubin, alkaline phosphatase) → Did cancer spread to the liver, or is the liver handling chemotherapy safely?

Treatment logic

- 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.
- 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.
- 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.
- 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).

- 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.
- Temozolomide 75 mg/m² daily during radiation, then 150 to 200 mg/m² for 5 days every 28 days for 6 to 12 cycles (chemotherapy for glioblastoma) → Kills glioblastoma cells by adding methyl groups to their DNA so they cannot copy themselves and divide. Extends median survival from 12 months (surgery plus radiation alone) to 15 months when combined with radiation (per Stupp protocol, NEJM 2005, and confirmed by multiple trials). Works best if the tumor has MGMT promoter methylation (a marker that means the tumor cannot easily repair this DNA damage), which extends survival to about 21 months.
- Targeted therapy or immunotherapy based on tumor type: bevacizumab for recurrent glioblastoma; pembrolizumab or nivolumab for high-mutation-burden or mismatch-repair-deficient tumors; dabrafenib plus trametinib for BRAF V600E-mutant melanoma metastases → Blocks specific broken genes or proteins that let the tumor grow. Bevacizumab stops VEGF (vascular endothelial growth factor), which tumors use to grow new blood vessels, shrinking the mass temporarily in about 30 to 40 percent of recurrent glioblastomas. Immunotherapy (pembrolizumab, nivolumab — antibodies that block PD-1 on T cells) wakes up the immune system to recognize and attack cancer cells in tumors with high mutation load or mismatch repair defects. Dabrafenib (a BRAF inhibitor) plus trametinib (a MEK inhibitor) shut down the MAPK pathway in BRAF V600E-mutant melanoma cells, shrinking brain metastases in about 50 percent of cases.

Medications

Dexamethasone · Corticosteroid

Dexamethasone turns down swelling around the tumor. When a brain tumor grows, it squeezes and irritates the tissue nearby. That tissue reacts by swelling — fluid leaks out of tiny blood vessels, and immune cells rush in. The extra fluid raises pressure inside the skull, which causes headaches, confusion, weakness, and sometimes dangerous shifts in the brain. Dexamethasone tells blood vessel walls to tighten up and stop leaking, and it tells immune cells — white blood cells like macrophages and lymphocytes — to calm down and stop releasing inflammatory signals, chemicals like cytokines. Within hours to days, swelling shrinks, pressure drops, and symptoms ease. This gives the patient relief while the team plans surgery, radiation, or chemotherapy.

1. Dexamethasone enters cells in the swollen brain tissue.

2. Inside the cell, it binds to glucocorticoid receptors — special docking sites that control inflammation genes.
3. The drug-receptor pair moves into the nucleus and turns off genes that make inflammatory chemicals like cytokines and prostaglandins.
4. Blood vessel walls stop releasing signals that loosen their junctions, so less fluid leaks into brain tissue.
5. Immune cells quiet down and stop swarming the area.
6. Swelling and fluid build-up shrink, lowering pressure inside the skull.
7. Headache, confusion, and weakness improve — usually within hours to a few days.

SAFETY WATCH

High blood sugar — especially risky if you have diabetes or prediabetes; may need insulin adjustments · Stomach irritation or ulcers — take with food; your doctor may add a stomach protector like omeprazole · Mood swings, trouble sleeping, anxiety, or feeling wired · Increased appetite and weight gain — especially around the face, neck, and belly · Weak bones with long-term use — higher risk of fractures (osteoporosis) · Higher infection risk because the drug turns down your immune defenses · Muscle weakness, especially in hips and shoulders, if used for weeks to months · Must taper the dose slowly when stopping — sudden stop can cause dangerous adrenal crisis, when the body cannot make its own stress hormones fast enough

Levetiracetam (Keppra) · Antiepileptic (anti-seizure)

Brain tumors irritate the tissue around them, and that irritation makes neurons — nerve cells — fire in wild, uncontrolled bursts. That is a seizure. Levetiracetam calms those neurons by changing how they release neurotransmitters, the chemical messengers that neurons use to talk to each other. It binds to a protein called SV2A on the storage bubbles inside neurons. When SV2A is bound, neurons release less glutamate — the brain's main excitatory signal — so they are less likely to fire chaotically. Levetiracetam does NOT speed up liver enzymes, which means it does NOT interfere with chemotherapy or other drugs. That is why it is the first-choice anti-seizure drug for brain tumor patients.

1. Levetiracetam enters the brain and finds neurons near the tumor.
2. It binds to SV2A, a protein on synaptic vesicles — the bubbles that store glutamate inside the neuron.
3. That binding slows down the release of glutamate when the neuron fires.
4. With less excitatory glutamate being released, neurons are less likely to fire in the rapid, chaotic bursts that cause seizures.
5. The brain stays calmer and more controlled.

6. Seizure risk drops — usually within a few days of starting the medicine.

SAFETY WATCH

Mood changes — irritability, aggression, anxiety, or depression — common, especially in the first few weeks; tell your doctor if this happens · Dizziness or drowsiness — usually mild · Fatigue or weakness · Do NOT stop suddenly — seizures can come back or become more severe; always taper slowly under a doctor's guidance

Temozolomide (Temodar) · Alkylating chemotherapy agent

Temozolomide is a chemotherapy pill that damages tumor cell DNA so the cells die when they try to divide. Cancer cells divide fast, and when their DNA is broken, they cannot copy it correctly — so they fail and die. Temozolomide works especially well in the brain because it crosses the blood-brain barrier, the tight fence of cells that blocks most drugs from entering the brain. It is the standard first chemotherapy for glioblastoma and anaplastic astrocytoma, usually given after surgery and during and after radiation. Inside the tumor cell, temozolomide breaks down and sticks small chemical tags — methyl groups — onto DNA at specific spots, guanine bases. When the cell tries to divide, its repair machinery sees the damage, tries to fix it, fails, and triggers cell death.

1. Temozolomide is swallowed as a pill and absorbed from the stomach into the bloodstream.
2. It crosses the blood-brain barrier and enters brain tissue.
3. Inside tumor cells, it breaks down into a reactive molecule called MTIC.
4. MTIC attaches methyl groups — CH_3 — to guanine bases on the DNA strand.
5. When the tumor cell tries to divide, DNA copying machinery sees the methylated guanine and stalls.
6. The cell's mismatch repair system tries to fix it but cannot, triggering programmed cell death — apoptosis.
7. Tumor cells die, and the tumor shrinks or stops growing — usually seen over weeks to months.

SAFETY WATCH

Low white blood cell count — increases infection risk; fever during treatment is an emergency — call your doctor immediately · Low platelet count — makes you bruise or bleed easily · Nausea and vomiting — anti-nausea medicine like ondansetron is given with each dose · Fatigue and weakness · Constipation · Risk of *Pneumocystis jirovecii* pneumonia, a lung infection — prevented with an antibiotic like trimethoprim-sulfamethoxazole, also called Bactrim · Rare risk of new cancers years later — secondary malignancies — from DNA damage in healthy cells · Rare liver injury — monitor liver tests

Bevacizumab (Avastin) · Monoclonal antibody (VEGF inhibitor)

Tumors grow by building their own blood supply. They release a chemical signal called VEGF — vascular endothelial growth factor — that tells the body to sprout new blood vessels into the tumor, like roots growing into soil. Bevacizumab is a lab-made antibody that sticks to VEGF and blocks it. Without VEGF signals, new blood vessels cannot form, and some leaky vessels tighten up. The tumor gets less blood, which can slow or shrink it. Swelling — swelling (edema) — around the tumor also drops because vessels stop leaking so much fluid. Bevacizumab is used for recurrent glioblastoma, when the tumor comes back after surgery, radiation, and temozolomide, and it often improves symptoms and MRI appearance even if it does not cure the cancer.

1. Bevacizumab is given by IV infusion into a vein, usually every 2 or 3 weeks.
2. It circulates in the bloodstream and binds tightly to VEGF molecules.
3. With VEGF blocked, signals to grow new blood vessels are turned off.
4. Fewer new vessels sprout into the tumor, so its blood supply shrinks.
5. Some existing leaky vessels normalize — their walls tighten, and they leak less fluid.
6. Less blood feeding the tumor slows its growth.
7. Swelling and fluid around the tumor drop, often improving headache, confusion, and weakness within days to weeks.

SAFETY WATCH

High blood pressure — monitor regularly; you may need blood pressure medicine · Increased bleeding risk — nosebleeds, bleeding gums, gastrointestinal bleeding, or, rarely, bleeding in the brain — intracranial bleeding (hemorrhage) · Slow wound healing — avoid surgery for at least 4 weeks after the last dose; wounds may open or not heal · Protein in the urine — proteinuria — or kidney damage · Blood clots — stroke, heart attack, or a clot in a deep leg vein (deep vein thrombosis) in the legs · Rare but serious: bowel perforation, a hole in the intestine wall that causes severe belly pain, fever, and needs emergency surgery · Fatigue and weakness

Phenytoin (Dilantin) · Antiepileptic (anti-seizure, sodium channel blocker)

Phenytoin stops seizures by blocking sodium channels on neuron membranes. Neurons fire electrical signals by opening sodium channels and letting sodium ions rush in — like opening a gate. When a neuron is irritated — by a tumor pressing on it — it fires too fast and too often, and that chaotic firing spreads to other neurons, causing a seizure. Phenytoin binds to sodium channels when they are open and keeps them closed longer, slowing the neuron's ability to fire repeatedly. Rapid, out-of-control firing is blocked, so seizures are prevented. However, phenytoin has a major downside in brain tumor patients: it speeds up liver enzymes — CYP450

enzymes — that break down chemotherapy and other drugs, lowering their levels in the blood and making treatment less effective. For this reason, levetiracetam — Keppra — or other non-enzyme-inducing drugs are preferred. Phenytoin is used only if other options fail or cause intolerable side effects.

1. Phenytoin enters the brain and finds neurons that are firing rapidly.
2. It binds to sodium channels that are already open — in the active state.
3. It holds the channels closed longer, slowing how fast the neuron can fire again.
4. Rapid, repetitive firing — the hallmark of a seizure — is blocked.
5. Seizures are prevented or stopped.

SAFETY WATCH

Gum overgrowth — gingival hyperplasia — keep up with brushing, flossing, and dental cleanings · Dizziness, unsteady walking — loss of coordination (ataxia) — slurred speech, or double vision — signs the blood level is too high · Rash — can rarely progress to Stevens-Johnson syndrome, a severe, life-threatening skin reaction — stop the drug and call your doctor immediately if rash appears · Low blood counts — anemia, low white cells, low platelets · Speeds up liver enzymes that break down chemotherapy — temozolomide, lomustine — steroids — dexamethasone — birth control pills, warfarin, and many other drugs — dose adjustments often needed · Requires regular blood level checks to stay in the safe and effective range — too low means seizures break through, too high means toxicity · Can cause birth defects — avoid during pregnancy if possible; use effective non-hormonal birth control

Lomustine (CCNU) · Nitrosourea alkylating chemotherapy agent

Lomustine is a chemotherapy pill that crosses into the brain and kills tumor cells by damaging their DNA. Inside the cell, lomustine breaks down into molecules that add chemical groups — alkyl groups — to DNA strands. These groups make the two DNA strands stick together — cross-link — like gluing two ribbons into a tangled knot. When the cell tries to divide, it cannot pull the strands apart to copy them, so it fails and dies. Lomustine is used for recurrent glioblastoma — when the tumor comes back — and certain oligodendrogliomas, often combined with other drugs like procarbazine and vincristine — the PCV regimen. It is given in cycles — one dose every 6 weeks — because it causes severe bone marrow suppression, a drop in blood cell counts that takes weeks to recover.

1. Lomustine is swallowed as capsules on an empty stomach.
2. It is absorbed into the bloodstream and crosses the blood-brain barrier.
3. Inside tumor cells, lomustine breaks down into reactive molecules — isocyanates and alkylating metabolites.

4. Those molecules attach alkyl groups to DNA bases, especially guanine.
5. The DNA strands cross-link — stick together — forming a tangled knot.
6. When the cell tries to divide, it cannot separate the strands to copy them.
7. The cell's repair machinery tries to fix the damage but fails, triggering cell death.
8. Tumor growth slows or stops over weeks to months.

SAFETY WATCH

Severe drop in white blood cells and platelets 4 to 6 weeks after each dose — blood counts must be monitored closely; you may need transfusions or growth factor shots · Nausea and vomiting — often severe on the day of dosing; take anti-nausea drugs like ondansetron before and after the pills · Fatigue and weakness · Liver damage — hepatotoxicity — monitor liver enzyme tests regularly · Lung scarring — pulmonary fibrosis — with long-term use — rare but serious and can be permanent; causes shortness of breath and cough · Kidney damage — nephrotoxicity — with prolonged treatment — monitor kidney function · Increased infection risk during periods of low white cell count · Cumulative bone marrow damage — each cycle makes recovery harder; some patients cannot tolerate more than a few cycles

Red flags

- 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

Nursing priorities

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 — 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 — 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 — 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
4. Keep two suction setups at the bedside, position the patient on their side during meals, and have padded side rails up with the bed in the lowest position if the patient has had a seizure or is at high risk — Patients with brain tumors can seize without warning; turning the head to the side during a seizure lets saliva and vomit drain out instead of being inhaled into the lungs (aspiration), and padding prevents injury from hitting hard bed rails during rhythmic shaking
5. Administer prescribed anti-seizure medication such as levetiracetam or phenytoin at the same times every day and teach the patient never to skip a dose or stop suddenly — Anti-seizure drugs work by keeping brain cells less excitable, but they must stay at steady blood levels to prevent breakthrough seizures; missing doses or stopping suddenly can trigger status epilepticus (a seizure that will not stop) (a seizure lasting longer than 5 minutes), which can cause permanent brain damage
6. Offer small frequent meals, give anti-nausea medicine 30 minutes before breakfast, and keep the room dim and quiet — Increased intracranial pressure stimulates the vomiting center in the brainstem and is worse in the morning after lying flat all night; vomiting raises pressure inside the skull even more and can trigger herniation, so preventing it protects the brain
7. Assess for signs of a clot in a deep leg vein (deep vein thrombosis) (leg swelling, warmth, redness, or pain in the calf) every shift and ensure sequential compression devices (inflatable leg sleeves) are on whenever the patient is in bed — Brain tumors release chemicals that make blood clot too easily, and patients are often less mobile after surgery or during radiation; a clot in the leg vein can break off and travel to the lungs (pulmonary a traveling clot lodging in a vessel (embolism)), causing sudden death

8. Monitor intake and output closely, report urine output greater than 200 mL per hour for 2 hours in a row, and check serum sodium and osmolality (concentration of particles in blood) daily — Brain tumors and surgery near the pituitary gland (a pea-sized gland at the base of the brain) can cause diabetes insipidus (a condition where the kidneys dump huge amounts of dilute urine), leading to severe dehydration and high sodium levels that cause confusion and seizures if not caught early

Sources

- NCCN Clinical Practice Guidelines in Oncology: Central Nervous System Cancers 2024
- American Brain Tumor Association
- UpToDate Clinical Overview: Brain Tumors in Adults
- WHO Classification of Tumours of the Central Nervous System 2021

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