Bacterial Meningitis

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THE STORY IN ONE SENTENCE

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“Bacterial meningitis means bacteria have infected the cerebrospinal fluid around the brain and spinal cord. Every hour of delay in treatment increases the risk of death or permanent brain damage.”

Normal anatomy — the body's job

The blood-brain barrier is a selective filter in the walls of brain blood vessels that keeps bacteria, toxins, and most immune cells out of the cerebrospinal fluid (the clear liquid that bathes the brain and spinal cord, called CSF). CSF is normally sterile, clear, and low in protein and white blood cells. The meninges (three protective membranes: dura, arachnoid, pia) cover the brain and spinal cord. CSF circulates in the subarachnoid space (the gap between the arachnoid and pia layers).

BODY'S JOB

The blood-brain barrier protects the brain from infection. CSF cushions the brain against trauma, delivers nutrients, and removes waste. The meninges provide a physical barrier and immune surveillance. When these defenses work, the brain stays sterile and the intracranial pressure (pressure inside the skull) stays normal.

1. Blood flows through brain capillaries (tiny blood vessels). The blood-brain barrier (tight junctions between endothelial cells lining these vessels) blocks bacteria and large molecules.
2. CSF is made by the choroid plexus (a spongy tissue inside the brain's ventricles, or fluid-filled chambers). About 500 mL of CSF is produced daily.

3. CSF flows from the ventricles through narrow channels into the subarachnoid space, bathing the brain and spinal cord.
4. CSF is absorbed back into the bloodstream through arachnoid granulations (small projections into the brain's venous sinuses, the large veins that drain blood from the brain).
5. This cycle keeps CSF sterile and the pressure inside the skull balanced. When working normally, bacteria cannot enter, immune cells stay out, and the brain floats safely in a cushion of clean fluid.

What breaks

Bacteria breach the blood-brain barrier and invade the cerebrospinal fluid, turning a normally sterile space into a site of infection and inflammation.

CLINICAL TERM

Bacteria enter the CSF by hematogenous spread (traveling through the bloodstream, most common), direct extension from a nearby infection (sinusitis, otitis media, mastoiditis), penetrating head trauma, neurosurgical procedures, or congenital defects (such as a dermal sinus tract or spina bifida). Common pathogens vary by age: Streptococcus pneumoniae and Neisseria meningitidis in adults and older children, Group B Streptococcus (GBS) and Escherichia coli in neonates, Haemophilus influenzae type b (Hib) now rare due to vaccination, and Listeria monocytogenes in neonates, elderly, and immunocompromised patients. Once in the CSF, bacteria multiply rapidly because the CSF lacks complement (a set of immune proteins that tag bacteria for destruction) and antibodies. Bacterial cell wall components (lipopolysaccharide in gram-negative bacteria, peptidoglycan in gram-positive bacteria) trigger release of cytokines (inflammatory messenger proteins: TNF-α, IL-1, IL-6) from microglia (brain immune cells) and endothelial cells (cells lining blood vessels), causing meningeal inflammation (swelling of the brain's protective membranes), increased blood-brain barrier permeability (more fluid and cells leak into the brain), vasculitis (inflammation of blood vessels), clot formation (thrombosis) (clot formation), cerebral swelling (edema) (brain swelling), and elevated intracranial pressure (ICP).

How the body compensates

The body tries to maintain cerebral blood flow (perfusion) pressure (CPP, the pressure pushing blood into the brain) by raising blood pressure and heart rate, but rising intracranial pressure

(ICP) from brain swelling eventually overwhelms these compensations. Vital signs may appear stable early, masking the severity of injury, until the patient suddenly deteriorates.

1. Early: The body increases mean arterial pressure (MAP, average blood pressure in arteries) and heart rate to keep CPP above 60 mmHg (CPP = MAP − ICP). The patient may be alert or only mildly confused despite having bacteria in the CSF.
2. This is the dangerous compensation phase: vital signs look reassuring, but intracranial pressure is climbing silently. Teams may underestimate severity and delay antibiotics.
3. The crash point: When ICP rises too high, CPP drops, brain tissue becomes blood-starved (ischemic) (starved of oxygen), and the patient herniates (brain tissue shifts and compresses the brainstem, causing sudden coma, abnormal posturing, irregular breathing, and death). In meningococcemia (bloodstream infection with Neisseria meningitidis), Waterhouse-Friderichsen syndrome (adrenal gland bleeding (hemorrhage)) causes sudden shock, pinpoint bleeding spots (petechiae) (small purple skin spots from broken blood vessels), and multiorgan failure.
4. Watch trending vital signs and mental status, not single snapshots. A patient with stable blood pressure but worsening confusion and increasing heart rate is decompensating, not stable.
5. Once herniation or septic shock begins, the window for meaningful recovery narrows rapidly. Early recognition—within the first hours—saves lives and brain function.
6. A 'stable' patient on a downward trend is unstable. Treat aggressively before the crash.

Domino effect

1. Bacteria enter the cerebrospinal fluid (CSF) and multiply. — Bacteria cross the blood-brain barrier via bloodstream spread (most common), direct extension from ear, sinus, or mastoid infection, skull fracture, or neurosurgery. Once in the CSF, bacteria face no immune defenses because CSF normally lacks antibodies and complement, so they replicate freely.
2. Bacterial toxins trigger intense inflammation in the meninges (brain lining). — Bacterial cell wall fragments (lipopolysaccharide in gram-negative bacteria, peptidoglycan in gram-positive bacteria) activate microglia (brain immune cells) and endothelial cells (blood vessel lining) to release cytokines (TNF-α, IL-1, IL-6). These cytokines cause fever (resetting the brain's thermostat), meningeal irritation (inflamed meninges pull on pain-sensitive structures, causing severe headache and a stiff neck (nuchal rigidity), or stiff neck), and increased permeability of the blood-brain barrier (allowing more white blood cells and protein into the CSF).

3. Cerebral swelling (edema) (brain swelling) and elevated intracranial pressure (ICP) develop. — Cytokines cause vasodilation (widening of blood vessels), increased capillary leakage (fluid shifts from blood into brain tissue), and cytotoxic swelling (edema) (neurons swell from injury). Because the skull is rigid, swelling increases ICP, which compresses blood vessels and reduces cerebral blood flow (perfusion) pressure (CPP), starving neurons of oxygen and glucose. This manifests as altered mental status (confusion, lethargy, coma) and, if severe, swelling of the optic nerve (papilledema) (swelling of the optic nerve visible on exam) and herniation (brain tissue shifts, compressing the brainstem).
4. Vasculitis (inflamed blood vessels), clot formation (thrombosis) (clot formation), and blood-starved (ischemic) injury occur. — Inflammatory cytokines damage the endothelium (inner lining of blood vessels in the brain), promoting clot formation and vessel narrowing. This causes focal blood-starved (ischemic) strokes (oxygen-starved brain regions stop working, producing focal neurologic deficits such as weakness, speech problems, or vision loss) and increases seizure risk (ischemic neurons fire abnormally).
5. Systemic complications: septic shock, disseminated intravascular coagulation (DIC), and multiorgan failure. — If bacteria (especially Neisseria meningitidis) spill into the bloodstream (meningococcemia), they trigger a systemic inflammatory response. Cytokines cause widespread vasodilation and capillary leak, dropping blood pressure (septic shock, where organs do not get enough oxygen). DIC (widespread clotting and bleeding from clotting factor depletion) causes pinpoint bleeding spots (petechiae) and purple bleeding blotches (purpura) (purple skin spots from bleeding into the skin). Waterhouse-Friderichsen syndrome (hemorrhage into both adrenal glands) causes sudden adrenal insufficiency (loss of cortisol and aldosterone), worsening shock and electrolyte imbalances.

Symptoms · what the patient feels, and why

- Fever (often above 101°F or 38.5°C): When bacteria invade the meninges (the thin protective layers around the brain and spinal cord), the immune system releases chemicals called pyrogens (like IL-1 and TNF-α). These pyrogens tell the hypothalamus (the brain's temperature control center) to turn up the body's thermostat, making you feel hot and shivery so your immune cells can fight the bacteria better.
- Severe headache: The meninges are swollen and irritated, which stretches the pain-sensing nerve endings in the dura (the tough outer covering of the brain) and nearby blood vessels. When the brain swells, pressure inside the skull goes up, pressing on those nerves even more and causing a pounding, severe headache.
- Stiff neck (nuchal rigidity, hard to bend the neck forward): The inflamed meninges around the spinal cord are so irritated that moving the neck forward would stretch them painfully. To protect you, the muscles in the back of your neck automatically tighten up and resist any forward bending, making your neck feel stiff and locked.

- Confusion, drowsiness, irritability, or coma (altered mental status): When the brain swells, pressure inside the skull rises and squeezes the brain's blood vessels, so brain cells don't get enough oxygen and sugar to work normally. The body's inflammatory chemicals also interfere with how neurons (brain cells) talk to each other, causing confusion, sleepiness, or even loss of consciousness.
- Sensitivity to bright light (photophobia): The inflamed meninges irritate nerves that connect to the eyes, especially the optic nerve (which carries vision signals) and the trigeminal nerve (which senses pain in the face and eyes). When light hits your eyes, these already-irritated nerves send extra-strong pain signals to your brain.
- Nausea and vomiting: Rising pressure inside the skull signals a part of the brainstem called the chemoreceptor trigger zone (the body's nausea alarm). This alarm center also picks up inflammatory chemicals in the bloodstream from the infection and tells your stomach to empty itself by vomiting.
- Seizures (happen in 20 to 30 out of 100 people with bacterial meningitis): Brain cells on the surface of the brain become overexcited and fire out of control because they're not getting enough oxygen (from inflamed, narrowed blood vessels), the fever lowers the brain's seizure threshold, salt levels in the blood drop (from a hormone imbalance called SIADH), and inflammatory chemicals directly irritate the neurons.
- Tiny purple or red spots on the skin that don't fade when pressed (petechiae or purple bleeding blotches (purpura)): When Neisseria meningitidis bacteria get into the bloodstream (called meningococcemia), they damage the lining of blood vessels and trigger uncontrolled clotting all over the body (called DIC). Platelets (tiny clotting cells) and clotting proteins get used up, so small blood vessels leak and bleed under the skin, leaving purple spots. This is a sign of severe, life-threatening infection.
- Weakness on one side, trouble speaking, vision problems, or eye movement trouble (focal neurologic deficits): Inflammation can cause blood vessels in the brain to swell, get irritated (vasculitis), and form clots (thrombosis), blocking blood flow to a specific part of the brain and causing a small stroke. High pressure inside the skull can also pinch the nerves that move the eyes or control facial sensation, so you can't move your eyes normally or feel your face.
- Signs of dangerously high brain pressure: swollen optic nerve in the back of the eye (papilledema), very slow heart rate with high blood pressure and weird breathing (Cushing triad), or abnormal body posturing (stiff arms or legs): When the brain swells and pus blocks the drainage of cerebrospinal fluid (the cushioning liquid around the brain), pressure inside the skull shoots up. The brain gets squished downward and presses on the brainstem (the part that controls heartbeat and breathing). This pressure causes the heart to slow down, blood pressure to rise, and breathing to become irregular (Cushing triad). The body may also stiffen into abnormal postures because the brainstem is being crushed.

Focused exam

- Neck stiffness (nuchal rigidity): When bacteria invade cerebrospinal fluid (the liquid cushioning the brain and spinal cord), the meninges (three protective layers wrapping the brain) swell and become irritated. This swelling triggers muscle spasm in the neck. Ask the patient to tuck chin to chest—if they cannot or if it hurts, the meninges are likely inflamed. This is one of the hallmark signs pointing to infection inside the skull.
- Fever above 100.4°F (38°C): Fever tells you the immune system has spotted bacteria and is fighting back. White blood cells release chemicals called pyrogens (fever-makers) that reset the brain's thermostat higher. In bacterial meningitis, fever climbs quickly and stays high. When fever shows up with neck pain and confusion, think infection in the brain or spinal fluid, not a simple cold.
- Altered mental status (confusion, drowsiness, or trouble responding): Bacteria release toxins that poison nerve cells, and swelling inside the skull squeezes the brain. The patient may not know the date, answer questions oddly, or drift into sleepiness. This means the infection is directly damaging brain tissue and is a medical emergency. The longer the brain is under pressure, the greater the risk of permanent harm.
- Petechial or purpuric rash (tiny red or purple spots that do NOT turn white when you press them): Neisseria meningitidis (meningococcus bacteria) spills poison into the bloodstream that punches holes in the walls of tiny blood vessels called capillaries. Red blood cells leak into the skin, forming spots. Because blood is already in the tissue (not just dilated vessels), pressing the spot does NOT make it blanch (turn pale). This rash means bacteria are in the bloodstream and can trigger septic shock (dangerous drop in blood pressure) within hours.
- Kernig sign (pain and resistance when you straighten the knee while the hip is bent to 90 degrees): Straightening the leg stretches the inflamed meninges and tugs on nerve roots (where spinal nerves exit the spinal cord) bathed in infected fluid. If the patient feels sharp pain or cannot fully extend the knee, the meninges are irritated by bacterial infection. This is another classic meningitis sign.
- Severe headache (often described as the worst headache ever): Inflammation makes the brain and meninges swell inside the hard skull, so pressure builds with nowhere to go. Patients often say this headache is unlike any they have felt—constant, pounding, and getting worse. It is not a tension headache or migraine; it reflects rising pressure on pain-sensing structures inside the skull.

Diagnostics · every test answers a question

- Blood cultures (two sets from separate sites) → Are bacteria traveling in the bloodstream, and which type?

- Lumbar puncture (spinal tap) with cerebrospinal fluid analysis (cell count, protein, glucose, Gram stain, culture) → Is there infection in the spinal fluid, and is it bacterial, viral, or fungal?
- CT scan of the head (only BEFORE lumbar puncture if signs of very high pressure inside the skull) → Is there a mass or severe swelling that makes removing spinal fluid dangerous?
- Complete blood count with differential → Is the bone marrow churning out enough white blood cells to fight the infection?
- Serum glucose (drawn at the same time as lumbar puncture) → What is the blood sugar level so we can compare it to spinal fluid sugar?
- Procalcitonin (optional, not required for diagnosis) → Is this infection bacterial or viral?
- Cerebrospinal fluid polymerase chain reaction (PCR) panel (if available) → Can we detect bacterial DNA even if culture is negative or prior antibiotics were given?

Treatment logic

- Empiric intravenous antibiotics: vancomycin PLUS third-generation cephalosporin (ceftriaxone 2 grams every 12 hours or cefotaxime 2 grams every 4–6 hours) → These antibiotics kill the three most common bacteria causing meningitis in adults: Streptococcus pneumoniae (pneumococcus), Neisseria meningitidis (meningococcus), and—when ampicillin is added—Listeria monocytogenes. Vancomycin covers drug-resistant pneumococcus strains; ceftriaxone covers meningococcus and susceptible pneumococcus. Both cross the blood-brain barrier (the tight seal between blood vessels and brain tissue) and reach high enough levels in spinal fluid to kill bacteria. Start antibiotics within ONE HOUR of suspecting bacterial meningitis—every hour of delay raises the risk of death by eight percent (Infectious Diseases Society of America, IDSA, guidelines). NEVER wait for lumbar puncture if it cannot be done immediately.
- Dexamethasone 10 milligrams intravenous, given 15 to 20 minutes BEFORE or WITH the first antibiotic dose → Dexamethasone is a steroid that quiets the immune system's inflammatory response. It blocks the release of cytokines (chemical alarm signals) in the space around the brain, reducing swelling of the meninges and lowering pressure inside the skull. In adults with pneumococcal meningitis, dexamethasone lowers the risk of death, hearing loss, and long-term brain damage (European Dexamethasone in Adulthood Bacterial Meningitis trial). It works best when started early—ideally before or with the first antibiotic dose—so inflammation is controlled from the start.

- Add ampicillin 2 grams intravenous every four hours if patient is over 50 years old, pregnant, or immunocompromised → Listeria monocytogenes causes meningitis in older adults, pregnant women, newborns, and people with weakened immune systems (chronic steroids, HIV, cancer chemotherapy, organ transplant). Ceftriaxone and cefotaxime do NOT cover Listeria—only ampicillin does. Adding ampicillin ensures all likely bacteria are covered in high-risk groups (IDSA guidelines). Once culture results return and Listeria is ruled out, ampicillin can be stopped.
- Lumbar puncture with cerebrospinal fluid analysis (cell count with differential, protein, glucose, Gram stain, bacterial culture, PCR if available) → Lumbar puncture confirms the diagnosis by showing bacteria or bacterial DNA in spinal fluid and identifies which bacteria is causing the infection. Once culture and sensitivity results return (24 to 72 hours), antibiotics can be narrowed from broad empiric coverage to targeted therapy against the specific germ, reducing side effects and antibiotic resistance. For example, if Neisseria meningitidis is isolated and is penicillin-sensitive, vancomycin can be stopped and the patient switched to penicillin alone.
- Supportive care: elevate head of bed to 30 degrees, maintain normal blood volume (euvolemia) with normal saline, avoid hypotonic fluids, monitor for seizures, ensure oxygen saturation above 94 percent, keep carbon dioxide in normal range → Elevating the head promotes blood drainage out of the brain veins, lowering pressure inside the skull. Normal saline (0.9 percent sodium chloride) keeps blood pressure and brain blood flow (perfusion) (blood flow to brain tissue) stable without worsening brain swelling. Hypotonic fluids such as half-normal saline or dextrose 5 percent in water cause water to shift into brain cells, making swelling worse. Seizures raise brain metabolic demand and increase intracranial pressure; treat with antiepileptic drugs such as levetiracetam or fosphenytoin if they occur. Keep oxygen saturation above 94 percent; low oxygen (hypoxia) injures brain cells. Keep carbon dioxide in the normal range (35 to 45 millimeters of mercury); high carbon dioxide (hypercapnia) dilates blood vessels in the brain and raises pressure.
- Chemoprophylaxis for close contacts: ciprofloxacin 500 milligrams by mouth (single dose), OR ceftriaxone 250 milligrams intramuscular (single dose), OR rifampin 600 milligrams by mouth twice daily for two days—IF Neisseria meningitidis is identified → Meningococcus spreads through respiratory droplets (saliva and mucus from coughing, sneezing, or kissing). Close contacts—household members, roommates, daycare contacts, anyone exposed to the patient's saliva within seven days before illness—may carry the bacteria in the back of the nose and throat without symptoms. Preventive (Prophylactic) antibiotics kill meningococcus in the nasopharynx (back of nose and throat) before it invades the bloodstream, preventing secondary cases. The Centers for Disease Control and Prevention (CDC) and American Academy of Pediatrics (AAP) recommend giving prevention (prophylaxis) within 24 hours of identifying meningococcal disease.

- Anticonvulsant therapy (levetiracetam 1000–1500 milligrams intravenous, or fosphenytoin 15–20 milligrams per kilogram intravenous) if seizures occur → Seizures occur in 20 to 30 percent of bacterial meningitis cases because inflamed brain tissue becomes electrically unstable. Seizures raise brain oxygen demand, increase intracranial pressure, and can cause additional brain injury. Levetiracetam or fosphenytoin stops seizure activity quickly and prevents repeated seizures. Use seizure prevention (prophylaxis) (giving anticonvulsants before seizures happen) ONLY if there is evidence of ongoing seizures on exam or electroencephalogram (EEG), NOT routinely in all patients.

Medications

Ceftriaxone plus vancomycin (started right away, before lab results) · Ceftriaxone is a third-generation cephalosporin (a bacterial cell-wall blocker). Vancomycin is a glycopeptide antibiotic (a different kind of cell-wall blocker).

Ceftriaxone crosses into the fluid around your brain and spinal cord (cerebrospinal fluid, or CSF) and kills bacteria by stopping them from building their protective outer shell (cell wall). It works especially well against the two most common meningitis germs: Streptococcus pneumoniae (pneumococcus) and Neisseria meningitidis (meningococcus). Vancomycin also gets into the inflamed CSF and kills bacteria that are resistant to other drugs, especially drug-resistant S. pneumoniae, by blocking a different part of the same wall-building process. If the patient is over 50 years old, has a weak immune system (from cancer, HIV, transplant medicines, or long-term steroids), is pregnant, or has a chronic illness like diabetes or kidney disease, doctors add ampicillin (a penicillin-type drug) to kill Listeria monocytogenes, a germ that sneaks in through contaminated food and causes meningitis in these groups. If the patient has confusion, seizures, or brain-scan changes that suggest herpes simplex virus (HSV) encephalitis (brain inflammation from the cold-sore virus), doctors add acyclovir (an antiviral drug that stops the virus from copying its DNA).

1. Ceftriaxone latches onto proteins called penicillin-binding proteins on the bacteria's surface. These proteins normally weave together chains of peptidoglycan (the mesh that makes up the cell wall). When ceftriaxone blocks them, the bacteria can't finish building their wall. The bacteria swell and pop (lyse) because they can't hold their insides together.
2. Vancomycin grabs onto short chains of amino acids (called D-Ala-D-Ala) that are sticking out from the growing cell wall of Gram-positive bacteria (like S. pneumoniae). By binding these chains, vancomycin stops the wall from getting longer and thicker. The bacteria die because they can't repair or expand their wall.
3. Ampicillin (if added for Listeria) works the same way as ceftriaxone: it binds penicillin-binding proteins and breaks the Listeria cell wall, causing the bacteria to burst.

4. Acyclovir (if added for suspected HSV) enters infected brain cells. Inside, a viral enzyme called thymidine kinase activates (phosphorylates) acyclovir. Once activated, acyclovir jams the virus's DNA polymerase (the enzyme that copies viral DNA), so the virus can't make new copies of itself and the infection stops spreading.

SAFETY WATCH

Draw two sets of blood cultures (blood samples to grow bacteria in the lab) and perform a lumbar puncture (spinal tap) to get CSF before giving antibiotics—but never delay antibiotics to wait for these tests. If the patient has signs of dangerously high pressure in the skull (such as a stiff neck plus confusion plus swelling of the optic nerve (papilledema)—swelling of the optic nerve seen in the eye exam—or focal neurologic signs like one-sided weakness), or a skin infection over the lumbar puncture site, or a low platelet count (under 50,000) or blood-clotting problem, do a head CT scan first to check for brain swelling or a mass. If any of those are present, skip the lumbar puncture and start antibiotics immediately. · Give dexamethasone (a steroid that dials down inflammation) 0.15 mg per kilogram of body weight by vein (IV) 10 to 20 minutes before—or at the same time as—the first antibiotic dose. This is especially important for pneumococcal meningitis (caused by S. pneumoniae), because dexamethasone lowers the risk of deafness, brain damage, and death. This recommendation comes from the Infectious Diseases Society of America (IDSA) guideline, published in 2004 and reaffirmed in 2016. · Once the CSF Gram stain (a quick dye test that shows the shape and color of bacteria under the microscope), culture (growing the bacteria in the lab), and antibiotic-susceptibility results (which drugs kill that bacteria) come back (usually 24 to 72 hours), narrow or change the antibiotics to match the exact germ and its sensitivities. · Check kidney function (blood creatinine and estimated glomerular filtration rate, or eGFR) before starting treatment and every few days during treatment, because both vancomycin and ceftriaxone are cleared by the kidneys. If kidney function drops, doses may need adjustment. · If the patient has Neisseria meningitidis (meningococcal) meningitis, close contacts—household members, daycare contacts, or anyone who had direct contact with the patient's saliva or respiratory droplets (sharing a drink, kissing, or being within three feet for more than eight hours)—need chemoprophylaxis (preventive antibiotics) to stop the germ from spreading. Give one of these: ciprofloxacin 500 mg by mouth once, rifampin 600 mg by mouth every 12 hours for four doses (two days), or ceftriaxone 250 mg by injection (IM) once. This guidance comes from the Centers for Disease Control and Prevention (CDC). · Do not give dexamethasone if you suspect fungal meningitis (such as Cryptococcus in someone with HIV), tuberculous meningitis (caused by Mycobacterium tuberculosis), or viral meningitis, because steroids suppress the immune system and make it harder for the body to clear those infections. Dexamethasone is safe and helpful only for bacterial meningitis, especially pneumococcal.

Red flags

- Tiny purple or red spots on the skin that don't fade when pressed (petechiae or purple bleeding blotches (purpura)): this means bacteria are in the bloodstream causing clotting problems and severe sepsis—antibiotics must be given immediately.
- Confusion getting worse or falling into a coma: the brain is swelling dangerously and may be starting to shift inside the skull—urgent brain imaging and ICU care are needed.
- Seizures, especially a seizure that lasts more than 5 minutes or keeps repeating (status epilepticus): the brain is severely inflamed or being damaged by lack of blood flow—emergency brain imaging, seizure medicine, and airway protection are needed.
- Very slow heart rate, high blood pressure, and irregular breathing (Cushing triad), or stiff, abnormal body positions (arms bent inward or legs straight and rigid): the brainstem is being crushed by the swelling brain—death is imminent without immediate treatment.
- Low blood pressure, fast heart rate, weak pulses, cold hands and feet, or little to no urine (signs of septic shock): the infection is overwhelming the whole body—IV fluids, blood pressure medicines, and ICU care are critical.

Nursing priorities

1. Draw blood cultures from two different sites, then start empiric intravenous vancomycin and ceftriaxone within 60 minutes of emergency department arrival, ideally within 30 minutes. — Bacterial meningitis kills brain cells every hour antibiotics are delayed; blood cultures taken before antibiotics help identify the exact bacteria later, but treatment cannot wait for culture results because mortality rises 10 to 15 percent for each hour without antibiotics.
2. Give dexamethasone 10 milligrams intravenous 15 to 20 minutes before or with the first antibiotic dose. — Dexamethasone (a steroid) reduces inflammation in the subarachnoid space (the fluid-filled area around the brain) and lowers risk of hearing loss, brain damage, and death, especially in pneumococcal meningitis; it works best when given before antibiotics start killing bacteria and releasing inflammatory toxins.
3. Perform hourly neurologic checks including Glasgow Coma Scale (a 15-point score measuring eye opening, speech, and movement), pupil size and reaction to light, and motor strength in all four limbs. — Rising intracranial pressure from brain swelling causes herniation (brain tissue pushes through skull openings and compresses the brainstem); early signs include decreasing consciousness, unequal pupils, or new arm and leg weakness, and catching these changes allows immediate imaging and life-saving interventions like hyperosmolar therapy or neurosurgery.

4. Place the patient in droplet isolation with a surgical mask on the patient or caregivers within three feet until 24 hours after antibiotics start. — Neisseria meningitidis and Haemophilus influenzae spread through respiratory droplets (coughs and sneezes); droplet precautions protect staff and visitors from inhaling bacteria, and isolation can stop after 24 hours of antibiotics because bacteria die quickly and become non-contagious.
5. Keep the bed flat or head elevated only 0 to 15 degrees, avoid hip flexion above 90 degrees, and keep the neck in neutral alignment without turning the head sharply. — These positions prevent obstruction of jugular veins (the large veins draining blood from the brain); blocked venous drainage raises intracranial pressure and worsens brain swelling, while slight head elevation helps cerebrospinal fluid flow without compromising cerebral blood flow (perfusion) pressure (the blood pressure pushing oxygen into brain tissue).
6. Monitor temperature every hour and give scheduled acetaminophen or cooling blankets to keep temperature below 100.4°F or 38°C. — Fever increases the brain's metabolic rate (how much oxygen and glucose brain cells need) by about 10 percent for every 1°C rise; high fever makes blood-starved (ischemic) injury worse in already swollen, poorly perfused brain tissue, and cooling reduces secondary brain damage.
7. Maintain a quiet, dark room by dimming lights, closing curtains, and limiting unnecessary noise and visitors. — Light sensitivity (Photophobia) (light sensitivity) and sound sensitivity (phonophobia) (sound sensitivity) happen because inflamed meninges make sensory nerves hyperactive; reducing stimulation decreases pain and agitation, lowers sympathetic stress response (which raises intracranial pressure), and improves patient comfort during critical early hours.
8. Keep intravenous access patent with at least one large-bore peripheral IV or central line, and monitor fluid balance to maintain euvolemia (normal fluid status) with isotonic saline. — Meningitis patients need reliable IV access for continuous antibiotics, steroids, and possible vasopressors if septic shock develops; dehydration lowers cerebral blood flow (perfusion) pressure and worsens brain starved blood flow (ischemia), but fluid overload can worsen cerebral swelling (edema) (brain swelling), so careful isotonic fluid replacement maintains blood pressure without adding extra water to swollen brain tissue.

Sources

- IDSA Clinical Practice Guidelines for Bacterial Meningitis, 2017
- CDC Bacterial Meningitis Guidance, 2023
- Cochrane Systematic Reviews on Bacterial Meningitis Treatment
- AAN/IDSA Joint Guideline on Encephalitis and Meningitis, 2023

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