Meningitis

Nurses are the first-line clinicians to observe these patients in the ICU, thus, they're an integral part of preventing major complications.

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When laypersons hear the word "meningitis," they may respond with fear, because the media often equate the condition with death. Meningitis is an inflammation of the meninges of the brain, which is caused by various pathogens. Infections from other areas of the body can migrate into the cerebrospinal fluid (CSF), causing meningeal inflammation and infection. It's important to revisit the anatomy, epidemiology, and types of meningitis, and prevention, nursing care, and treatment for this disease.

The primary entrance of pathogens that cause meningitis is typically the nasopharynx.[1] Because the nasopharynx is in close proximity to the brain, the pathogens can pass through the nasopharyngeal membrane into blood circulation. Once in circulation, local pathogens can invade into the protective layers of the central nervous system, known as the meninges. The meningeal layers are the dura mater, arachnoid, and pia mater. Cerebrospinal fluid originates within the ventricles of the brain, circulates through the subarachnoid space around the brain and spinal cord, and is absorbed by the arachnoid villi.[1] (See "Cranial meninges.") The meninges help provide support against infection. Changes in the blood-brain barrier (BBB) may lead to increased susceptibility for infectious pathogens to pass through the BBB and further infect the meninges.[1] Several factors can increase permeability of the BBB, thereby allowing easier passage of pathogens. These include alterations in the tight junctions of the blood vessels or in glial cells of the membrane that prevents most particles from passing, and variations of hormones that create support for the plasma membranes of the barrier itself.[1]

Organisms

Organisms such as bacteria, viruses, and fungi can cause meningitis. In healthy individuals, these organisms can be a part of the body's normal flora. Pathogens can be endemic to specific populations. These include persons living in close contact, such as in a dormitory or military barracks. Sub-Saharan Africa is a hyperendemic area for meningitis and is known as the "meningitis belt."[2] Individuals with certain risk factors, such as young children, the elderly (age 60 or older), or those with diabetes, malignancy, immunosuppression, or a ventriculoperitoneal shunt (VPS), can become more susceptible to developing meningitis. Neurosurgical procedures and basal skull fractures also predispose a person to contracting meningitis.

Bacterial

Bacterial meningitis may become fatal if left untreated. There are common forms of bacterial meningitis:

1. *Streptococcus pneumoniae* (pneumococcus) is the

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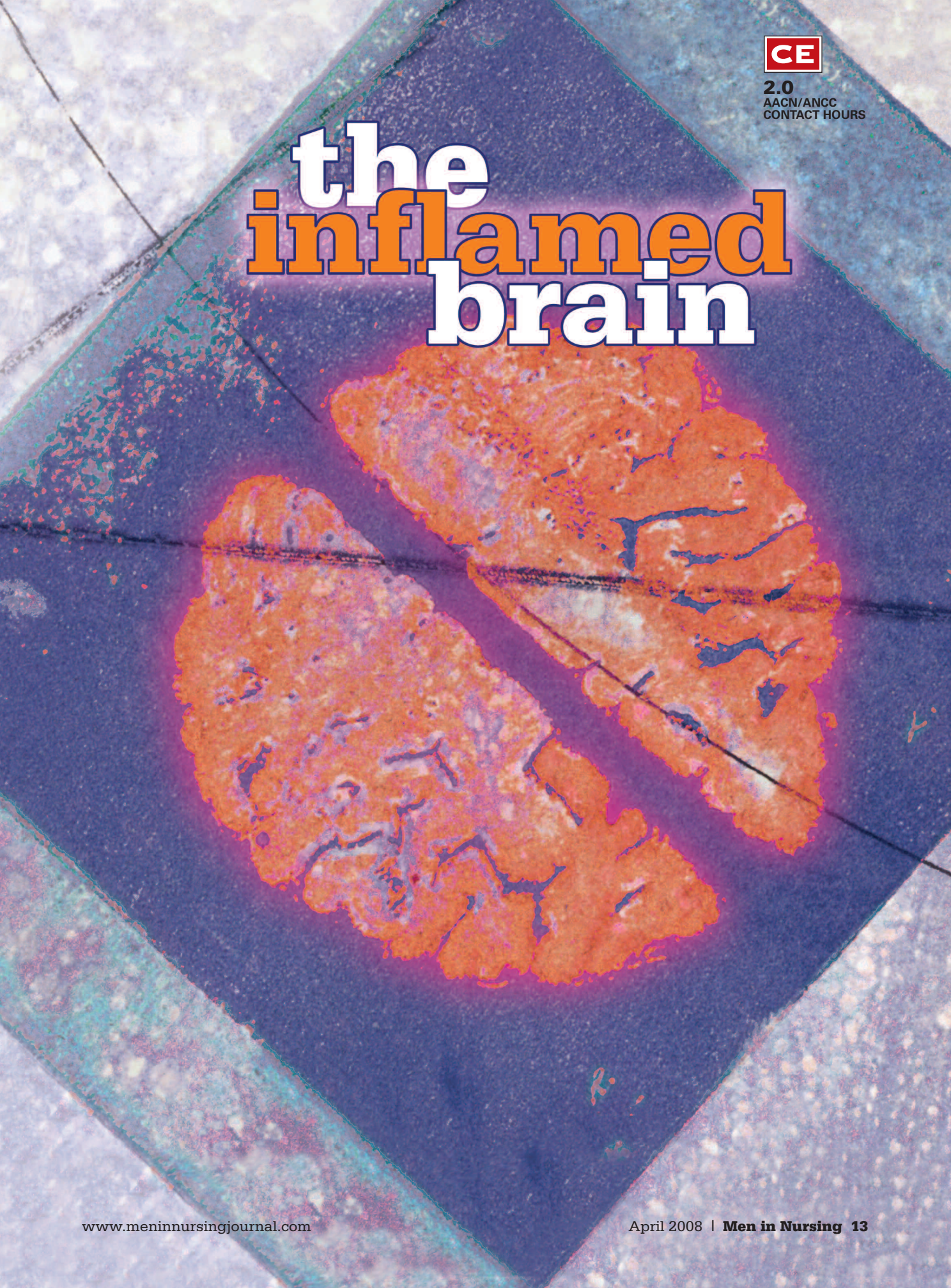
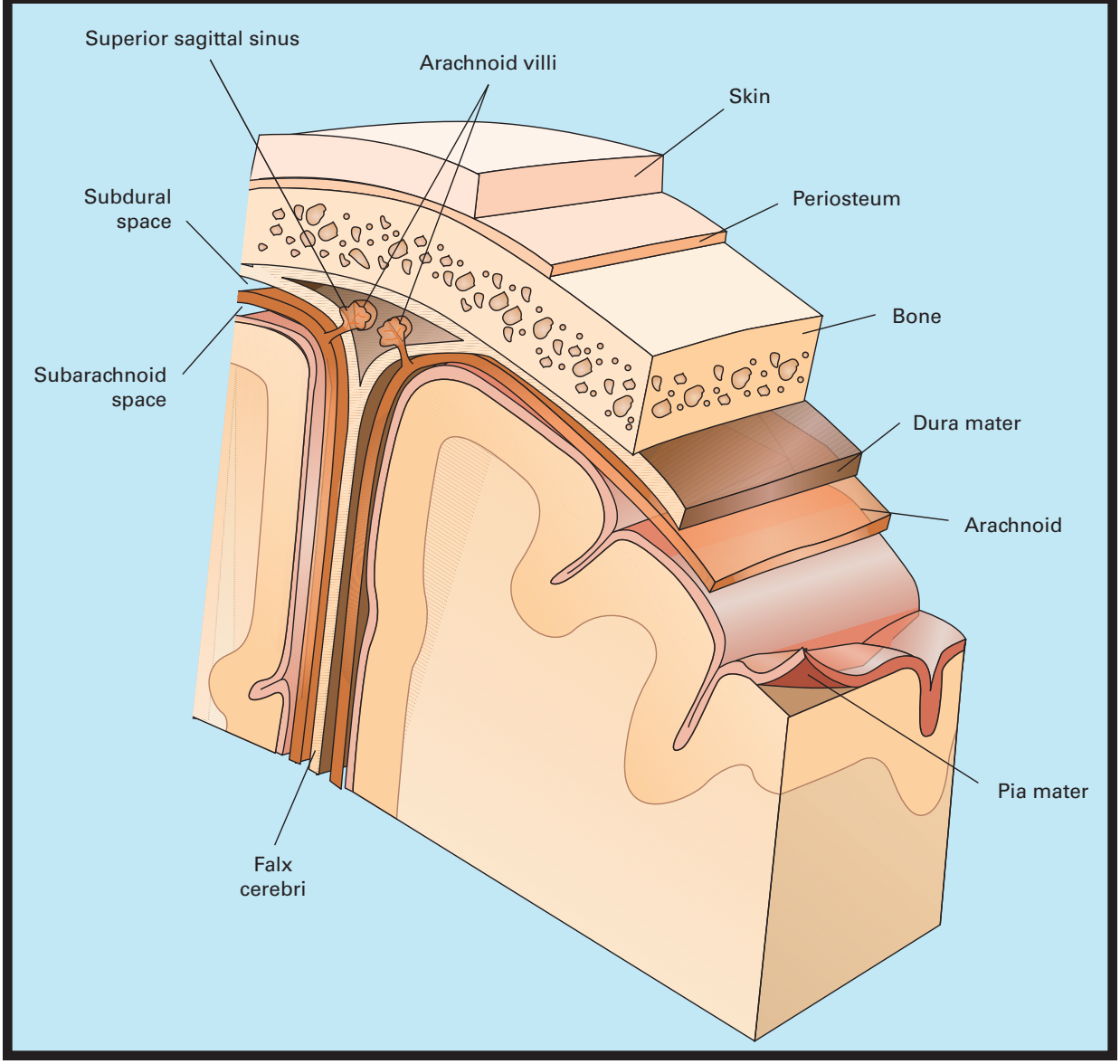
the inflamed brain



Cranial meninges



most common cause of bacterial meningitis, and also causes pneumonia, sinus infection, and otitis media in all age groups except neonates.[3]

2. *Neisseria meningitidis* (meningococcus), which often resides in the nose and throat, causes bacterial meningitis that's associated with areas having increased communal living, such as college dormitories and day care centers.[4] Primary modes of transmission include direct contact or inhalation of respiratory droplets through coughing or sneezing from infected carriers. According to researchers, although 5% to 10% of adults are asymptomatic carriers, the incidence of infection increases 60% to 80% within members of a closed population.[4] Meningococcus is highly contagious and can cause devastating neurologic consequences and death.

3. *Haemophilus influenzae* serotype B (Hib) is transmitted via direct contact with respiratory droplets from the nasopharynx, is formerly the leading cause of bacterial meningitis, and is accountable for upper respiratory infections and otitis media in children. Administration of the Hib vaccine as part of childhood immunizations has dramatically reduced the incidence of *H. influenzae* meningitis.[3]

4. *Listeria monocytogenes* infection, transmitted via ingestion of contaminated foods, is prevalent in pregnant women, the elderly, neonates, and patients with immunosuppression.[5]

5. *Staphylococcus aureus* is associated with head trauma, craniotomies, ventriculoperitoneal shunt, and other neurosurgical procedures.[3] The mode of transmission is through hematogenous spread from an infected site.

Viral

Enteroviruses, which include polioviruses, coxsackieviruses, and echoviruses, are responsible for 85% to 95% of all cases of viral meningitis.[5] Viral meningitis usually clears on its own and is often responsible for gastrointestinal viral infections. Enteroviruses are commonly transmitted via direct contact with contaminated respiratory secretion, including saliva, sputum, and the direct transmission via hands to nose or mouth. They are also transmitted via direct contact with feces. Other common viruses include adenovirus and paramyxovirus (mumps), and the herpes family viruses: HSV-1, HSV-2, varicella-zoster virus, Epstein-Barr virus, and cytomegalovirus.[6]

Fungal

A common nonbacterial, nonviral cause of meningitis is the fungus *Cryptococcus neoformans*, an encapsulated yeast, which is responsible for cryptococcal meningitis. Commonly associated with aged pigeon droppings or bird feces, *C. neoformans* is the most common variety in the United States and is most associated with cryptococcal infections worldwide.[7]

Incidence

According to data, statistics of the incidence of major bacterial pathogens (per 100,000) in the United States in 1995 are as follows:

- *Streptococcus pneumoniae* (13) had a mortality rate of 14% in patients hospitalized[8]
- *Neisseria meningitidis* (0.6) usually increased in late winter and early spring, with a 10% to 14% mortality[3,8]
- *Haemophilus influenzae* serotype b (0.2) in unvaccinated children and adults, with a 3% to 6% mortality.[3,8]

Signs and symptoms

Patients with all forms of meningitis present with signs and symptoms of meningeal inflammation/irritation and systemic infection. Common complaints usually include headache, fever, chills, nuchal rigidity, vomiting, photophobia, and seizures. Depending on age, virulence of the strain, and defense system of the host, patients can become critically ill if not promptly diagnosed and treated.[3]

Infection with bacterial meningitis carries high mortality and morbidity rates with an overall fatality of 25% reported in adults.[4] These patients can have an acute onset of common meningeal symptoms, as listed above, which can progress rapidly to neurological deterioration. As the disease advances, macular skin rash progressing to purpuric and ecchymotic lesions may be present due to petechial hemorrhage, seen specifically in meningococcal disease.[9] As the infection ensues, circulatory shock and death can transpire, even if treated. Therefore, antibiotic therapy should be initiated within 30 minutes of emergency department presentation for those suspected of bacterial meningitis.[3]

Devastating outcomes can occur if patients aren't adequately treated with antifungal medications.

Viral meningitis is more prevalent, but causes less serious complications than bacterial meningitis. Patients with viral meningitis commonly present with similar meningeal symptoms, which may be less severe and appear flu-like in nature. They may complain of a history of an upper respiratory infection that is accompanied by headache, stiff neck, anorexia, or generalized malaise.[9] Depending on the infecting organism, these symptoms may dissipate without treatment.

Clinical manifestation of fungal meningitis may be mild initially. Due to the encapsulated nature of the organism, the body may not exhibit signs of infection until extensive neurological involvement has occurred. Headache, low-grade fever, vomiting, and lethargy are primary symptoms that can occur and may fluctuate throughout the course of illness.[3] Once a patient becomes infected, a characteristic feature of fungal infection is its tendency to recur, especially for patients who are immunosuppressed.[7] Therefore, devastating outcomes can occur if patients aren't adequately treated with antifungal medications.

Testing for meningeal irritation

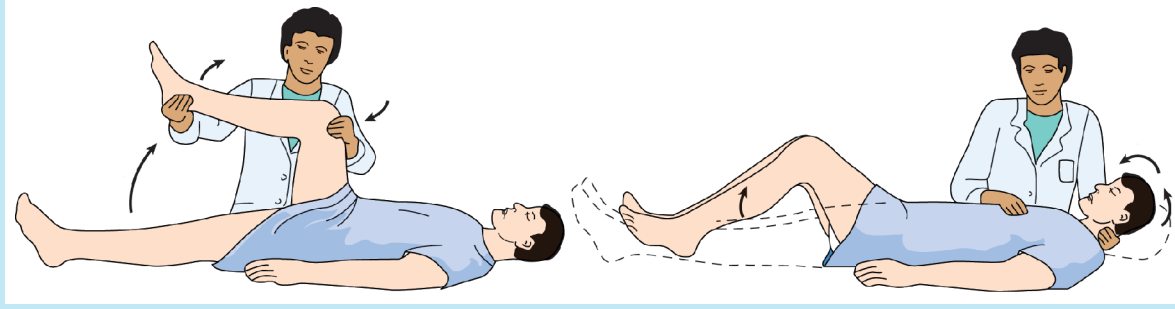
Positive Brudzinski's and Kernig's signs indicate meningeal irritation, a sign of meningitis.

Kernig's sign

Have your patient lie in the supine position. Flex his hip and knee to form a 90-degree angle. Then attempt to extend his leg. If he exhibits pain or resistance to extension and spasm of the hamstring, the test is positive.

Brudzinski's sign

Have your patient lie in the supine position. Then place your hand under his neck and flex it forward, chin to chest. The test is positive if he flexes his knees and hips bilaterally. The patient will typically complain of pain when his neck is flexed.



Nursing assessment

Initial nursing assessment includes a thorough history, neurologic exam, review of systems, and vital signs. The nursing assessment must also focus on history of travel, previous infections, medications, and sources of immunosuppression. The neurologic exam consists of cranial nerve assessment, level of consciousness, motor strength, sensory exam and evaluation of headache. Other signs of meningeal irritation may include positive Kernig's sign, which is pain and hamstring resistance that is elicited upon passive knee extension while patient is supine.[9] Brudzinski's sign is positive if there's an involuntary flexion of hips due to passive neck flexion while patient is supine.[9] (See "Testing for meningeal irritation.") Findings from a neurological exam may include altered mental status, confusion and irritability, vision disturbances such as photophobia, cranial nerve deficits, and changes in level of consciousness.[9]

The gastrointestinal system may be affected if the patient presents with nausea and vomiting that can typically be related to meningeal irritation. All other major body systems may be normal depending on the severity of the initial infection.

Diagnostic tests

Diagnostic tests include computed tomography (CT) scan and magnetic resonance imaging (MRI) to rule out other sources for neurological changes, assess for hydrocephalus, and show meningeal enhancement. A chest X-ray may be done to identify pneumonia or secondary fungal infection. Laboratory tests include complete blood cell count with differential, which may show leukocytosis, serum glucose, which may be drawn as a baseline determination for normal CSF glucose, BUN, creatinine, and liver function studies to assess renal and hepatic function and adjust antibiotic dosages.[3] Serology is needed to identify specific antigens correlated with specific pathogens. Cultures are also required of the following: CSF, blood, urine, nasal passages, and drainage from wounds, for diagnosis of a specific organism and the source of infection.[10] Further investigation of the CSF helps determine the specific type of meningitis and the degree of infiltration. A lumbar puncture is performed to obtain CSF and assess initial opening pressures, which can indicate hydrocephalus. (See "Comparison of CSF findings among organisms.")

Nursing care

Nursing care needs to focus on patient's symptoms as they are presented. The primary treatment is the timely administration of empiric antibiotics, even if an organism hasn't been initially identified.[3] Antibiotics may be started, especially if meningeal symptoms are present, prior to CSF culture results.[6,9] Basic care while in the intensive care unit (ICU) includes reducing the amount of stimulation, maintaining proper body alignment with head straight and elevated to at least 30 degrees, fre-

Comparison of CSF findings among organisms

Normal parameters	Normal CSF	Bacterial meningitis	Viral meningitis	Fungal meningitis
Open pressure (mm H_2O)	100 to 180	200 to 500	≤ 250	> 200
Leukocyte count WBC/mm3	0 to 5	Increased 1,000 to 5,000	Increased 50 to 1,000	Increased > 20
Neutrophils (%)	0	≥ 80	< 40	—
Protein (mg/dL)	18 to 45	Elevated 100 to 500	Elevated < 200	Elevated > 45
Glucose (mg/dL)	45 to 80; 0.6 times blood glucose level	Five to 40; < 0.3 times blood glucose level	> 45	< 40

Source: Bader M, Littlejohns L. *AANN Core Curriculum for Neuroscience Nursing.* Philadelphia, Pa: Saunders; 2004:632.

quent pain and neurological assessment, close monitoring of vital signs, ensuring adequate cerebral perfusion pressure (CPP), assessing and treating fever, maintaining standard precautions, and providing adequate skin care, nutrition, and hydration throughout the hospital stay.[9] Minimal noise levels and dim lights create a more calming environment and can prevent agitation in the patient with meningitis. All these factors can help prevent the devastating outcomes of organism invasion such as shock and increasing intracranial pressure (ICP).

If meningococcal meningitis is suspected, droplet precautions are recommended until 24 hours after initiation of effective therapy.[11] Any close contacts, including family members, healthcare providers who did not wear masks, and anyone in close proximity to the patient, need administration of prophylactic antibiotics within 24 hours after patient diagnosis.[8]

Treatment

Medical interventions depend on whether the meningitis is bacterial, viral, or fungal in nature.

Bacterial treatment: Empiric intravenous antibiotics must be given for the specific organism endemic to the region. When specific organisms are isolated, the appropriate antibiotic may then be started intravenously.[12] Initial empiric antibiotics include third generation cephalosporins such as ceftriaxone (Rocephin), or fluoroquinolones such as ciprofloxacin (Cipro).[8] According to researchers, the use of glucocorticoids, such as dexamethasone (Decadron), with the initiation of the first antibiotic dose has been shown to decrease unfavorable outcomes and mortality from bacterial meningitis with adults.[11] However, there's limited data on dexamethasone use in adults, and if given should be initiated with the first dose of antibiotics for a limited number of days.[3] Steroid use is controversial, and research is ongoing.[13] In a systemic review, experts suggest, "…routine steroid therapy with the first dose of antibiotics is justified in most adult patients in whom acute community acquired bacterial meningitis is suspected."[14] The major effect of dexamethasone is a decrease in the inflammatory response.[14]

Viral treatment: Medical management for viral meningitis is mostly supportive based on symptoms. Bed rest, adequate hydration, antipyretics, antiemetics, and analgesics are recommended. Antiviral medications may be administered and are usually reserved for severe cases of infection caused by herpes viruses.[15]

Fungal treatment: Patients with fungal meningitis should be aggressively treated with antifungal medications such as amphotericin B and fluconazole (Diflucan) given intravenously for several weeks.[7]

Possible complications

Complications of meningitis are exclusive to the presenting symptoms of the individual. The cranium is an isolated vault that cannot sustain much change. Infection within the meninges leads to the inflammatory cascade, producing many dynamic events in the brain from mild headache to herniation and death. Because of the inflammatory response and the result of increased blood flow, several major complications develop from meningitis:

• *Compromised respiratory status* is a possible complication. The respiratory center is located in the brain stem, and additional pressure on this area causes changes in respiratory status. Vigilant assessment of respiratory status should focus on maintaining a patent airway. Changes in status may require noninvasive ventilator support or full ventilator support as indicated by the patient's decline.[11]

Case study

J.T. is a 28-year-old white female who presented to the emergency department with complaints of headache, vomiting, and changes in level of consciousness. A computed tomography scan of the head was done and showed enlarged ventricles, indicating hydrocephalus. She was admitted to the neuroscience intensive care unit. A ventriculostomy was inserted to alleviate ventricular pressure, and samples of CSF were sent for analysis, including cultures. The initial CSF specimen isolated an organism, and indicated protein and glucose levels consistent with meningitis. Collective assessment of laboratory tests, diagnostics, signs and symptoms, and personal history of bird handling has led to her diagnosis. She was diagnosed with infection by *Cryptococcus neoformans*, an infectious diseases physician was consulted, and amphotericin B was initiated intravenously. From admission to day 5 of hospitalization, CSF results showed significant improvements. (See "Results of CSF analysis over 5 days.") Once the infection cleared, J.T. received a ventriculoperitoneal shunt (VPS).

In addition to the multiple shunt revisions during this admission, J.T. dealt with numerous complications in the ICU. Her residual neurological effects included memory deficiencies, slow speech, and flat affect. She developed a deep vein thrombosis, pulmonary embolism, recurrent urinary tract infections, and multiple cardiac events such as hypertension and tachycardia. During her ICU course, a brain biopsy was also done to rule out any malignant diagnosis. She was eventually discharged to the neuroscience floor 4 months after admission, only to return to ICU with respiratory failure requiring ventilator support. This admission was the first of many admissions to the ICU from the neuroscience floor. J.T. spent approximately 6 months in the hospital before she was finally discharged home. In the last month prior to discharge, she received a percutaneous endogastrostomy tube to help maintain caloric intake. Although her recovery was slow, she had ample family support throughout her lengthy hospitalization. Due to her prolonged hospitalization, she also required the assistance of physical therapy, occupational therapy, and speech therapy in her long transition to normal functionality. She eventually went to a rehabilitation facility close to home to finish her recovery.

Although this case may not be a typical patient seen in the neuroscience ICU, it demonstrates the damaging and long-term sequela of fungal meningitis.

Results of CSF analysis over 5 days

	Admission	Day 2	Day 5
Protein (mg/dL)	730	1,111	190
Glucose (mg/dL)	38	45	55

• *Circulatory collapse and shock* can occur during sepsis, which can mount an excessive immune response, specifically the release of cytokines such as tumor necrosis factor alpha. This event then stimulates endothelial damage and a systemic inflammatory response.[13] Nurses should monitor volume status, while paying close attention to vital signs, especially changes in blood pressure. Intravenous crystalloid fluids, volume expanders, and inotropic agents may be necessary to maintain adequate perfusion of organs.[10] A pulmonary artery catheter may become necessary for more detailed monitoring of hemodynamic status.[11]

• *Cerebral edema* is swelling of the cerebral tissue that can also lead to increased ICP, thus affecting neurological status. Methods to control cerebral edema include the administration of hypertonic saline solutions and osmotic diuretics. In extreme cases, craniectomy, or bone flap removal via surgery, can also alleviate pressure.[9]

• *Hydrocephalus* is secondary to decreased CSF absorption, and increases CSF in the central nervous system. This leads to increased ICP and must be relieved to prevent severe neurologic decompensation. A ventriculostomy placed into the ventricles of the brain can help to monitor ICP and drain excess CSF.[11] It also creates a simple avenue to obtain CSF specimens for cultures.

• *Seizure* activity is triggered by alteration in the electrical activity of the brain.[6] There can be several causes of seizure in a meningitis patient including fever, edema, inflammation, and increased pressure. Electroencephalogram (EEG) monitoring is needed to further investigate the location, length, and severity of seizure activity. Cortical inflammation, a definitive cause of seizures in patients with meningitis, may be visible on EEG.[11] Antiepileptic medications should be administered in

meningitis patients who have a clinical suspicion or prior seizure.[11] Witnessed seizures can be treated emergently with benzodiazepines. Protection from self injury, maintaining adequate oxygenation, and airway patency are important during seizures.

• *Diabetes insipidus (DI) and syndrome of inappropriate antidiuretic hormone* (SIADH) are also complications that may occur. Alterations in the pituitary gland lead to electrolyte disturbances and fluid imbalances. The posterior pituitary secretes antidiuretic hormone, and excess amounts will produce SIADH, while decreased amounts will produce DI. Serum sodium and osmolality, body weight, and urine specific gravity must be monitored for trends. SIADH, which result in increased water retention and a low serum sodium, is treated with fluid restriction and in severe cases judicious replacement of sodium using hypertonic saline intravenously.[6] Diuretic therapy with furosemide (Lasix) may be initiated if fluid restriction is not sufficient.[9] Diabetes insipidus can increase water excretion and can be treated with fluid replacement and use of vasopressin if deemed necessary by the medical team, to facilitate water reabsorption.[6]

• *Immobility* is caused by deficiencies in neurological status. Nursing measures include turning the patient every 2 hours, performing range-of-motion exercises, and meticulous skin care. Deep vein thrombosis prophylaxis should also be implemented by use of sequential compression devices and prophylactic anticoagulation if not contraindicated.

• *Gastrointestinal issues* are another complication. Decrease in gastrointestinal motility for meningitis patient can result in complication such as constipation. Stool softeners are given to encourage normal bowel regimen. Increased gastric secretions can cause peptic ulcers, so prophylaxis can be accomplished by administering proton pump inhibitors.[11]

• *Herniation*, one of the most severe complications, will lead to brain death. Herniation is the result of many factors including cerebral edema and increased ICP. Frequent neurological assessments with special attention to pupillary response to light are vital to predicting an impending herniation. A positive assessment of pressure on the third cranial nerve (oculomotor nerve) is a clear indicator of impending downward pressure onto the brainstem. Ipsilateral pupillary dilatation may be an early indicative sign of uncal herniation. Treatment to prevent herniation consists of administration of diuretics, hypertonic saline, osmotic diuretics, and maintaining ICP less than 15 mm Hg and CPP greater than 60 mm Hg.[11] (For more on managing complications, see "Case study.")

Vaccination

An important factor to consider in meningitis is disease prevention strategies. For example, there are several serotypes of infectious meningococci. Certain types are specific to an endemic area. Common serotypes of meningococci include A, B, C, Y, and W-135. The most common groups found in the United States are groups B and C.[8,12] There are two types of vaccines available to prevent meningitis: the conjugate and the polysaccharide. Both of these vaccines prevent infection from serotypes A, C, Y, and W-135, but not from group B. The Centers for Disease Control and Prevention (CDC) recommends meningococcal vaccination for specific groups: persons living in close quarters including military and first-year college students, technicians exposed to the bacteria, and persons traveling to sub-Saharan African countries during the months of December to June.[16] It's also recommended for travel to Mecca during the Hajj (Islamic pilgrimage to Saudi Arabia). The CDC also recommends that those who have had a splenectomy also receive the vaccine.[16] Pneumococcal, Hib, and influenza vaccines have also assisted in the decline of meningitis related to these infections.[8]

Meningococcal infection must be diagnosed and treated promptly with antibiotics to prevent an epidemic.

First line of defense

Meningitis is a universal phenomenon and can be devastating to any patient. Specifically, meningococcal infection must be diagnosed and treated promptly with antibiotics to prevent an epidemic. The medical team must be diligent in its effort to overcome the complex side effects of brain injury secondary to organism invasion. Careful neurological examination by trained caregivers, interpre-

tation of intracranial monitoring must be foremost in all nursing care. Nurses are in an integral part of the prevention of major complications. The nurse must have a thorough understanding of the anatomy of the brain and understanding of the inflammatory cascade and its sequela. Adequate management of respiratory infections and pneumonia can prevent meningitis. Such management includes appropriate vaccinations, especially to high-risk populations, which may lessen the risk of meningitis. Prompt antibiotic administration for basal skull fractures and CSF leaks may also lessen the risk of meningitis.[9]

Community education is also essential to disease prevention. Vaccination as indicated by the CDC is essential in the prevention and spread of bacterial meningitis. Research continues, and with the emergence of genetic factors in healthcare, new therapies and diagnostics may also be emerging. M

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