**Saudi Journal of Medicine and Public Health**

https://saudijmph.com/index.php/pub

https://doi.org/10.64483/202631882

# Nursing Assessment and Management of La Crosse Encephalitis in Pediatric Patients: Clinical Manifestations and Neurological Outcomes.

**Daifallah Dahwi Abdulrhman[(1)] , Nouri Marzouq Mutlaq Alotaibi[(1)] , Rahaf Abddulrahman Almutairi[(2)] , Faten Moeid Almutairi[(3)] , Seham Khalaf Rasheed Almutiri[(4)] , Ayda Saeed Almotiri[(5)] , Anbar Omar Amer Almutiri[(6)] , Maeetm Abdurhman Alhafi[(7)] , Huda Muhammad Metleaa Al Mutairi[(8)] , Nayafah Miedid Mishean Almutayri[(9)]**

*(1)Sajir First Health Center, Ministry of Health, Saudi Arabia*
*(2)Al-Amq Health Center, Madinah Health Cluster, Ministry of Health, Saudi Arabia*
*(3)Al-Mahd Health Center, Madinah Health Cluster, Ministry of Health, Saudi Arabia*
*(4)Al-Mahd General Hospital, Madinah, Madinah Health Cluster, Ministry of Health, Saudi Arabia*
*(5)Mahd Al-Dhahab (Al-Quwaiyah Health Center), Madinah Health Cluster, Ministry of Health, Saudi Arabia*
*(6)Al-Mahd General Hospital, Madinah, Madinah Health Cluster, Ministry of Health, Saudi Arabia*
*(7)Sajir Hospital, Ministry of Health, Saudi Arabia*
*(8)Afif Hospital, Ministry of Health, Saudi Arabia*
*(9)Al-Faisaliyah Health Center, Ministry of Health, Saudi Arabia*

## Abstract

**Background:** La Crosse encephalitis is a mosquito-borne viral infection caused by La Crosse virus, an orthobunyavirus belonging to the California serogroup. The disease primarily affects children and represents an important cause of arboviral encephalitis in the United States. Although most infections remain asymptomatic or produce mild febrile illness, neuroinvasive disease can cause seizures, encephalopathy, cerebral edema, and long-term neurological complications.

**Aim:** This review examines the etiology, epidemiology, pathophysiology, histopathological characteristics, clinical manifestations, diagnostic evaluation, treatment, prognosis, and complications of La Crosse encephalitis, with emphasis on clinical assessment and nursing management.

**Methods:** A narrative review approach was used to examine the available clinical and scientific literature addressing La Crosse virus infection. Relevant evidence concerning viral transmission, disease mechanisms, clinical presentation, laboratory diagnosis, neuroimaging, supportive treatment, and short- and long-term outcomes was synthesized.

**Results:** La Crosse virus is transmitted primarily through infected Aedes mosquitoes, with vertical transmission contributing to viral persistence in mosquito populations. The incubation period ranges from 5 to 15 days. Symptomatic patients commonly develop fever, headache, vomiting, and lethargy, while severe cases may present with seizures, altered consciousness, and cerebral edema. Diagnosis primarily relies on virus-specific IgM antibody detection in serum or cerebrospinal fluid, supported by neutralizing antibody testing. No established specific antiviral treatment is available. Management focuses on supportive care, seizure control, fluid and electrolyte management, and respiratory support when required. Although mortality is relatively low, some survivors experience recurrent seizures, cognitive impairment, behavioral changes, and developmental difficulties.

**Conclusion:** Early recognition, appropriate diagnostic evaluation, and supportive management are essential to reduce complications and improve outcomes. Long-term neurological and developmental follow-up remains important, particularly in affected children.

**Keywords:** La Crosse encephalitis; La Crosse virus; arboviral infection; pediatric encephalitis; neurological complications; cerebrospinal fluid; nursing management.

## Introduction

La Crosse encephalitis is a mosquito-borne viral infection that primarily affects children and may cause inflammation of the brain and surrounding neurological structures. It is caused by La Crosse virus (LACV), an arbovirus transmitted mainly through the bite of infected mosquitoes. The disease is most frequently reported in the central and eastern United States, particularly in regions where environmental conditions support the breeding and survival of its mosquito vectors. Although most infections remain asymptomatic or produce mild systemic manifestations, a small proportion progress to neuroinvasive disease characterized by meningitis, encephalitis, seizures, and altered consciousness. La Crosse encephalitis is recognized as one of the most frequently reported causes of pediatric arboviral encephalitis in North America [2,3]. The disease


*Corresponding author Ddalotaibi@moh.gov.sa (Daifallah Dahwi Abdulrhman).
Receive Date: 5 September 2026, Revise Date: 30 October 2026, Accept Date: 18 November 2026

takes its name from La Crosse County, Wisconsin, where the causative virus was first identified in 1964. This discovery followed an investigation of a 1960 outbreak of California encephalitis among children during the summer. Researchers subsequently isolated the virus from the brain tissue of a four-year-old child who died from meningoencephalitis [1]. This identification established the association between the virus and severe neurological disease in children and contributed to the development of subsequent epidemiological and virological investigations.

La Crosse virus belongs to the California serogroup of orthobunyaviruses, historically classified within the family Bunyaviridae. It possesses a segmented, single-stranded RNA genome and is transmitted primarily by the eastern tree-hole mosquito, Aedes triseriatus, which serves as its principal vector [4]. Other mosquito species, including Aedes albopictus and Aedes japonicus, have also been identified as potential vectors and may contribute to changes in transmission patterns in endemic regions [2]. The clinical significance of La Crosse encephalitis is related to its potential neurological complications, particularly among children younger than 16 years. Patients may initially present with fever, headache, nausea, vomiting, and malaise before developing more severe neurological manifestations. Seizures, confusion, and altered levels of consciousness may require hospitalization and intensive monitoring. Consequently, early recognition, appropriate diagnostic evaluation, and supportive management are important components of patient care. Nursing professionals contribute through neurological assessment, monitoring of vital signs, seizure precautions, fluid management, and family education. Understanding the epidemiology and transmission mechanisms of the disease also supports community prevention strategies aimed at reducing mosquito exposure.

**Etiology**

La Crosse encephalitis results from infection with La Crosse virus, an arbovirus belonging to the California serogroup of orthobunyaviruses. The virus is transmitted primarily through the bite of infected Aedes triseriatus mosquitoes. Its transmission cycle involves mosquito vectors and several small vertebrate mammals that act as natural reservoirs. Chipmunks, squirrels, and woodchucks are among the animals involved in maintaining viral circulation within endemic environments. The interaction between these hosts and mosquito populations allows the virus to persist in nature and periodically infect humans [5].




**Figure-1:** Etiology and epidemiology of La Crosse Encephalitis.

Two major transmission mechanisms contribute to the maintenance of La Crosse virus in mosquito populations. Horizontal transmission occurs when adult female mosquitoes acquire the virus while feeding on the blood of infected vertebrate hosts. Following infection, the virus replicates within the mosquito and can subsequently be transmitted to susceptible animals or humans during later blood meals. Although humans can develop severe neurological disease, they generally function as dead-end hosts because their circulating viral concentrations are insufficient to infect mosquitoes during subsequent feeding. This characteristic limits the contribution of human infections to the natural transmission cycle. In addition to transmission through vertebrate reservoirs, horizontal transmission between mosquitoes during mating has also been described as a possible mechanism of viral maintenance [6].

Vertical transmission is another important feature of La Crosse virus epidemiology. Infected female mosquitoes can transmit the virus to their offspring through the ovaries and developing eggs. This process, known as transovarial transmission, allows viral genetic material to pass from one mosquito generation to another. Infected eggs can survive unfavorable environmental conditions during winter and remain dormant until suitable temperatures and moisture conditions support hatching. When larvae emerge during the following spring, they may already carry the virus and subsequently develop into infected adult mosquitoes [7]. Vertical transmission is particularly important because it enables the virus to persist between mosquito seasons without continuous transmission through mammalian reservoirs. This mechanism

contributes to the long-term maintenance of La Crosse virus in endemic environments and explains why infections can recur annually in areas with established mosquito populations [8]. Several environmental and ecological factors influence the likelihood of human infection. Aedes triseriatus commonly breeds in natural and artificial tree holes, particularly in wooded residential environments. Containers, discarded tires, and other objects capable of retaining standing water may also provide suitable mosquito breeding sites. Human exposure increases during outdoor activities in wooded areas, particularly during warmer months when mosquito activity is high. Children may experience greater exposure because of outdoor recreational activities and their frequent presence in residential areas near mosquito habitats. Therefore, understanding the viral transmission cycle is essential for developing mosquito-control programs, reducing exposure, and educating families about preventive measures.

**Epidemiology**

La Crosse encephalitis is an uncommon but important cause of arboviral neurological disease in children in North America. The infection is endemic primarily in the central and eastern United States, with a concentration of reported cases in the upper Midwestern, mid-Atlantic, and Appalachian regions. West Virginia, North Carolina, Tennessee, and Ohio have historically accounted for a substantial proportion of reported infections. The geographical distribution reflects the presence of suitable mosquito habitats, vertebrate reservoir populations, and environmental conditions that support viral transmission. Although infections can occur throughout the mosquito season, most recognized cases develop between July and September, corresponding to periods of increased mosquito activity. Surveillance data demonstrate the predominantly pediatric distribution of clinically recognized La Crosse virus infections. An analysis of reported cases between 2003 and 2019 identified 1,281 human infections, approximately 92% of which involved neuroinvasive disease. The median patient age was eight years, and 88% of reported cases occurred in individuals younger than 18 years. Males represented approximately 59% of affected individuals. During the same period, the average annual national incidence of neuroinvasive La Crosse virus disease was estimated at 0.02 cases per 100,000 population. West Virginia, North Carolina, Tennessee, and Ohio together accounted for approximately 80% of reported cases [9]. These findings demonstrate the concentration of clinically recognized infections in specific geographical regions and younger age groups. The reported incidence substantially underestimates the total number of infections because most individuals infected with La Crosse virus do not develop severe neurological manifestations. Estimates suggest that approximately 96% to 99% of infections are asymptomatic or produce mild, nonspecific symptoms resembling influenza. These manifestations may include fever, headache, fatigue, and muscle discomfort. Consequently, many infections are never investigated or reported through routine surveillance systems [11].

Serological studies provide additional information about viral circulation in endemic areas. Investigations of wild mammals in northwestern Virginia identified an overall seroprevalence of nearly 2% among surveyed animals, including eastern gray squirrels, eastern cottontails, and red foxes. Human seroprevalence has reached approximately 35% in some highly endemic communities, indicating that exposure may be considerably more common than clinically diagnosed disease [10]. These findings emphasize the importance of distinguishing infection prevalence from reported encephalitis cases. Age is an important epidemiological factor because children experience a greater proportion of clinically recognized neuroinvasive infections. Their outdoor activities and exposure to mosquito habitats may increase opportunities for infection. Seasonal variation also influences disease occurrence, with the majority of cases developing during warmer months. From a public health and nursing perspective, these patterns support targeted mosquito-control interventions, community education, and early recognition of unexplained febrile neurological illness in children living in endemic regions.

**Pathophysiology**

La Crosse encephalitis develops following the transmission of La Crosse virus into human tissues through the bite of an infected mosquito. During feeding, the mosquito introduces virus-containing saliva into the subcutaneous tissue. The virus possesses an enveloped, segmented, single-stranded RNA genome and surface glycoproteins that participate in viral attachment and cellular entry. The G1 and G2 glycoproteins have been associated with interactions involving mammalian and mosquito cells, respectively, although the molecular mechanisms governing cell-specific infection are complex [12]. Following inoculation, viral replication may initially occur in local tissues, including muscle cells near the site of the mosquito bite. The virus subsequently enters the circulation and produces systemic infection involving components of the reticuloendothelial system and other susceptible tissues. Viremia facilitates viral dissemination and provides an opportunity for the virus to reach the central nervous system. The precise mechanisms responsible for neuroinvasion remain incompletely understood, but infection or disruption of vascular endothelial cells has been proposed as an important pathway. After entering the central nervous system, La Crosse virus can infect neuronal and glial cells. Neuronal infection interferes with normal cellular

functions and may produce neuronal injury, degeneration, and inflammatory responses. The resulting neurological inflammation contributes to the clinical manifestations of encephalitis, including headache, altered consciousness, seizures, and behavioral changes. The severity of neurological dysfunction depends on the extent of viral involvement, inflammatory responses, and associated cerebral tissue injury.

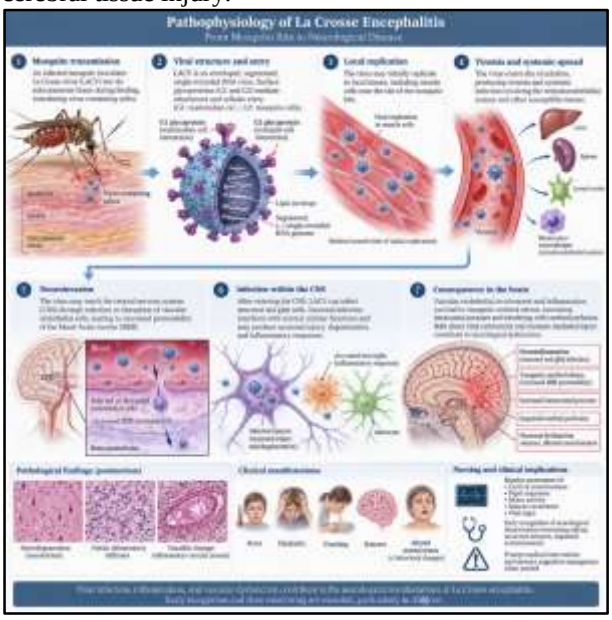



**Figure-2:** Pathophysiology of la crosse encephalitis.

Vascular endothelial involvement may also contribute to increased permeability of the blood-brain barrier. Disruption of normal endothelial function can facilitate the movement of fluid and inflammatory mediators into brain tissue, producing vasogenic cerebral edema. This process may increase intracranial pressure and interfere with cerebral perfusion. Reversible cerebral edema is considered an important proposed mechanism in the development of neurological manifestations associated with La Crosse encephalitis [14]. However, the relative contribution of direct viral cytotoxicity, immune-mediated injury, and vascular dysfunction continues to require investigation. Postmortem investigations have identified neurodegeneration, patchy inflammatory infiltrates, and vasculitic changes in affected brain tissue [13]. These findings support the involvement of both neuronal injury and inflammatory vascular processes. The inflammatory response may contribute to neuronal dysfunction even when extensive tissue destruction is absent. From a clinical perspective, these pathological processes explain the development of fever, headache, vomiting, seizures, and altered mental status. Cerebral edema and neurological inflammation may also produce focal neurological abnormalities in severe cases. Children with suspected encephalitis require regular assessment of consciousness, pupil responses, motor activity, seizure occurrence, and vital signs. Early recognition of neurological deterioration is important because increasing cerebral edema, recurrent seizures, and impaired consciousness may require intensive supportive management. Nursing surveillance therefore plays an important role in identifying complications and supporting timely medical intervention.

**Histopathology**

Histopathological changes in La Crosse encephalitis primarily involve the central nervous system and reflect the combined effects of viral infection, neuronal injury, endothelial involvement, and inflammatory responses. Tissue examination is generally performed in severe or fatal cases because brain biopsy is not routinely required for clinical diagnosis. Most histopathological information has therefore been obtained from postmortem investigations, which provide insight into the cellular and vascular changes associated with advanced neuroinvasive disease. Immunohistochemical and indirect immunofluorescence investigations have demonstrated viral antigen distribution within neuronal cells and capillary endothelial cells. Prominent immunofluorescence staining in these structures supports the involvement of both neuronal and vascular components in the disease process [13,14]. Neuronal infection may interfere with normal cellular metabolism and function, producing cellular degeneration and necrotic changes. The extent of neuronal injury varies according to disease severity and the distribution of viral involvement within the brain. Light microscopic examination has demonstrated perivascular inflammatory infiltrates, predominantly involving mononuclear cells. These inflammatory cells accumulate around small blood vessels and reflect the immune response to viral infection. Perivascular inflammation may be accompanied by focal tissue injury and necrosis. Such changes indicate that the pathological process is not restricted to direct viral damage to neurons but also involves inflammatory responses affecting the surrounding neural tissue [13].

Vasculitic changes represent another important histopathological feature. Inflammation involving cerebral blood vessels may disrupt endothelial integrity and increase vascular permeability. These changes provide a pathological basis for the proposed development of vasogenic cerebral edema. Endothelial cell infection and inflammatory injury may impair the blood-brain barrier, allowing plasma components and fluid to enter the surrounding brain tissue. The resulting edema may contribute to increased intracranial pressure and neurological dysfunction [14]. The distribution of inflammatory lesions and neuronal degeneration may be patchy rather than uniformly affecting the entire brain. This pattern is consistent with the variable neurological presentations observed among affected patients. Some individuals develop relatively limited neurological symptoms, whereas

others experience seizures, altered consciousness, and severe encephalopathy. Histopathological findings are important for understanding the biological mechanisms of La Crosse encephalitis, although they have limited direct application in routine patient assessment. Diagnosis is generally based on clinical findings, cerebrospinal fluid analysis, neuroimaging when indicated, and virus-specific laboratory testing. From a nursing perspective, understanding the underlying tissue changes helps explain the importance of monitoring neurological status, seizure activity, consciousness, and signs of increased intracranial pressure. Early identification of deterioration remains essential for preventing secondary neurological injury and supporting favorable clinical outcomes [14].

**History and Physical**

Most La Crosse virus infections are asymptomatic and remain undiagnosed. Approximately 1% to 4% of infected individuals develop clinical symptoms. Symptomatic disease ranges from a mild febrile illness lasting several days to severe encephalitis associated with neurological impairment and, occasionally, death. The overall case-fatality rate among patients diagnosed with La Crosse encephalitis is approximately 1%. However, severe neurological disease may result in substantial morbidity, particularly among children who experience prolonged seizures, cerebral edema, or persistent cognitive difficulties.[9,13] The incubation period generally ranges from 5 to 15 days following an infected mosquito bite. Among symptomatic patients, approximately 80% to 90% experience a relatively mild clinical course. Initial manifestations commonly include fever, headache, vomiting, and general malaise lasting 1 to 3 days, followed by gradual improvement during the subsequent week. Nevertheless, some patients develop neurological manifestations, including lethargy, behavioral changes, confusion, altered consciousness, and seizures. Seizures represent an important clinical feature of hospitalized patients. Approximately 50% of individuals admitted with La Crosse encephalitis experience seizures. These may be focal or generalized, occurring in approximately 40% to 60% of hospitalized patients, while status epilepticus has been reported in approximately 10% to 15%.[14]

Clinical presentation varies with age and disease severity. Children are more likely to develop meningoencephalitis, encephalopathy, and seizures. Adults more commonly present with fever, headache, hyponatremia, and altered mental status. Although seizures occur less frequently in adults, severe cases may progress to cerebral edema, impaired consciousness, and respiratory compromise. Mechanical ventilation may be required in up to 25% of patients with complicated disease.[4] Common clinical manifestations include fever, lethargy, headache, confusion, vomiting, and seizures. Less frequent findings include neck stiffness, photophobia, hyponatremia, respiratory distress, and coma. Physical examination should assess temperature, hydration status, neurological function, and level of consciousness. Clinicians should evaluate orientation, speech, motor responses, pupillary reactions, and seizure activity. Assessment for meningeal irritation is also important. In severe cases, repeated neurological examinations help identify deterioration that may indicate increased intracranial pressure or cerebral edema. From a nursing perspective, systematic assessment is essential. Nurses should monitor vital signs, neurological responses, fluid balance, and changes in behavior. Seizure precautions should be implemented when indicated. Any reduction in consciousness, abnormal respiratory pattern, or new focal neurological finding requires prompt clinical evaluation.

**Evaluation**

The diagnosis of La Crosse encephalitis relies on clinical findings, epidemiological exposure, cerebrospinal fluid analysis, and laboratory confirmation of infection. Enzyme-linked immunosorbent assay (ELISA) for immunoglobulin M (IgM) antibodies in serum or cerebrospinal fluid (CSF) represents the most commonly used diagnostic approach. Reported performance from one study demonstrated a sensitivity of 75% and specificity of 98%.[15] Detection of La Crosse virus-specific IgM supports recent infection. A fourfold increase in antibody titers between acute and convalescent serum samples collected approximately 2 weeks apart also provides evidence of infection.[13] Testing is available through selected commercial laboratories, state public health laboratories, and the Centers for Disease Control and Prevention (CDC). Serological results require careful interpretation because IgM assays may cross-react with antibodies against related arboviruses. Therefore, a positive result should be confirmed using a neutralizing antibody test performed by an appropriate public health laboratory or the CDC. Plaque reduction neutralization testing can help distinguish La Crosse virus infection from infections caused by related viruses. Molecular testing using polymerase chain reaction (PCR) may provide additional diagnostic evidence. PCR is particularly useful during the early stages of infection, when viral RNA may still be detectable, and in severely immunocompromised patients who may have an inadequate antibody response. Its sensitivity depends on specimen type and collection timing. Detection is more likely during active viremia and in central nervous system tissue. However, sensitivity is generally lower in CSF and decreases after the acute phase. PCR may also be used in postmortem investigations when viral concentrations in brain tissue remain detectable.

CSF examination commonly demonstrates lymphocytic pleocytosis, increased protein

concentration, and normal glucose levels. Viral culture has limited diagnostic value because it frequently produces negative results. Additional laboratory investigations include a complete blood count and basic metabolic panel. These tests may identify leukocytosis and electrolyte disturbances, particularly hyponatremia, which can accompany La Crosse encephalitis. Neuroimaging helps evaluate complications and exclude alternative diagnoses. Head computed tomography (CT) and magnetic resonance imaging (MRI) may initially appear normal because the disease does not have a consistent characteristic imaging pattern. Severe cases may demonstrate cerebral edema, increased intracranial pressure, or cerebral herniation. Basal ganglia hemorrhage has also been reported.[13] MRI may reveal cortical gadolinium enhancement in affected children.[16] Electroencephalography (EEG) is useful in patients with altered consciousness or suspected seizures. Findings may include unilateral slowing, periodic lateralized epileptiform discharges, and electrographic seizures. EEG abnormalities have been reported in up to 90% of individuals with severe disease.[4] Nursing responsibilities include monitoring neurological status, preparing patients for diagnostic procedures, documenting clinical changes, and supporting timely specimen collection.

**Treatment / Management**

No specific antiviral treatment has been established for La Crosse encephalitis. Management therefore focuses on supportive care, symptom control, and prevention of neurological complications. Treatment intensity depends on disease severity, clinical manifestations, and the presence of complications such as seizures, cerebral edema, respiratory compromise, and electrolyte disturbances. Patients with mild illness characterized by fever and headache may receive conservative treatment involving rest, adequate hydration, and analgesics. Clinical reassessment remains important because neurological deterioration may occur after the initial presentation. Patients presenting with acute encephalopathy require prompt investigation for potentially treatable causes of encephalitis. Intravenous acyclovir may be administered when herpes simplex virus encephalitis remains a diagnostic possibility. Treatment can continue until appropriate investigations exclude herpes simplex virus infection.[13] This approach is important because delayed treatment of herpes simplex encephalitis may result in serious neurological consequences. Ribavirin has been investigated as a potential antiviral treatment for La Crosse encephalitis. Intravenous administration has been used in some cases, although clinical effectiveness remains unproven.[14] Ribavirin is proposed to inhibit viral replication by interfering with viral RNA-dependent RNA polymerase activity. Earlier investigations in children suggested that intravenous ribavirin could penetrate the CSF, with concentrations reaching approximately 70% of corresponding plasma levels.[17,18] However, subsequent clinical investigations did not establish a clear therapeutic benefit. Oral ribavirin is not considered a suitable alternative because its CSF concentrations are lower than those achieved through intravenous administration.[19] An in vitro investigation demonstrated inhibition of La Crosse virus replication at a ribavirin concentration of approximately 0.3 μmol/L. Nevertheless, laboratory findings do not establish clinical effectiveness.[20]

Seizure management represents a major component of treatment in patients with neurological involvement. Patients who develop focal or generalized seizures may require antiseizure medications. Continuous or repeated EEG monitoring may be appropriate when consciousness remains impaired or nonconvulsive seizures are suspected. Long-term antiseizure treatment may be considered in individuals who develop recurrent seizures or post-encephalitic epilepsy. Severe encephalitis may produce cerebral edema and increased intracranial pressure. Management requires close neurological observation and appropriate critical care interventions. Patients with coma or respiratory compromise may require endotracheal intubation and mechanical ventilation. Fluid and electrolyte management is also essential, particularly when hyponatremia develops. Central venous pressure monitoring may assist in assessing intravascular volume in selected critically ill patients, although fluid management should be guided by the overall clinical condition.[13] Nursing care includes monitoring vital signs, neurological responses, oxygen saturation, fluid intake and output, and serum electrolyte levels. Nurses should maintain seizure precautions, administer prescribed medications, assess airway protection, and report changes in consciousness or respiratory function. Family education should address the expected clinical course, potential neurological complications, and the need for follow-up after discharge.

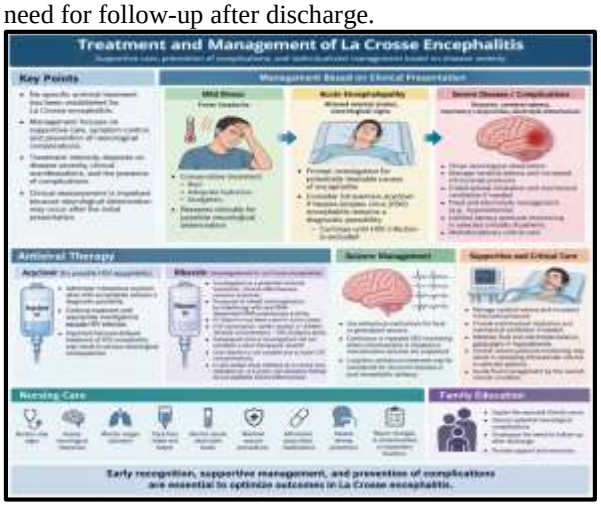



**Figure-3:** Treatment and Management of La crosse Encephalitis.

**Differential Diagnosis**

The differential diagnosis of La Crosse encephalitis includes several infectious and noninfectious disorders that produce fever, headache, meningeal irritation, seizures, altered consciousness, or focal neurological abnormalities. Clinical findings alone may not reliably distinguish La Crosse encephalitis from other causes of central nervous system inflammation. Diagnostic assessment should therefore consider the patient's age, geographical location, season of presentation, mosquito exposure, immune status, clinical progression, and laboratory findings.

**Infectious causes**

Other viral infections represent important differential diagnoses. Herpes simplex virus is particularly important because it can cause severe encephalitis and requires prompt antiviral treatment. Other California serogroup viruses, including Jamestown Canyon virus and Snowshoe hare virus, may produce overlapping neurological manifestations. Enteroviruses, varicella-zoster virus, rabies, and mumps should also be considered. Other arboviruses, including West Nile virus, St. Louis encephalitis virus, Powassan virus, and Eastern equine encephalitis virus, may cause similar clinical presentations. Epidemiological exposure and virus-specific laboratory testing help distinguish these infections. Bacterial meningitis and encephalitis must also be excluded, particularly infections caused by *Streptococcus pneumoniae*, *Neisseria meningitidis*, and *Haemophilus influenzae*. Rocky Mountain spotted fever may produce fever and neurological manifestations, especially when accompanied by compatible exposure history and systemic findings. Tuberculosis and toxoplasmosis may cause central nervous system disease, particularly in patients with relevant risk factors. Fungal infections, including cryptococcosis, coccidioidomycosis, and histoplasmosis, should be considered in appropriate geographical and immunological contexts. Amebic infections represent less common but potentially severe causes of neurological disease.

**Vascular causes**

Cerebral vasculitis may produce headache, seizures, altered mental status, and focal neurological deficits. Subarachnoid hemorrhage should be considered in patients presenting with sudden severe headache, vomiting, neck stiffness, or reduced consciousness. Dural venous sinus thrombosis may cause headache, seizures, and signs of increased intracranial pressure. Neuroimaging and vascular investigations help distinguish these conditions from infectious encephalitis.

**Autoimmune causes**

Autoimmune disorders may produce neurological inflammation without an active infection. Autoimmune vasculitis can cause cerebral ischemic or inflammatory manifestations. Neurosarcoidosis may involve the meninges and brain tissue, resulting in headache, cranial nerve abnormalities, and altered neurological function. Behçet disease may also involve the central nervous system and produce inflammatory neurological complications.

**Drug-induced meningitis**

Drug-induced aseptic meningitis should be considered when symptoms develop after exposure to specific medications. Immune checkpoint inhibitors, including nivolumab and ipilimumab, may produce immune-mediated neurological inflammation. Antimicrobial agents such as amoxicillin and sulfonamides have also been associated with drug-induced meningitis. Nonsteroidal anti-inflammatory drugs represent another potential cause. Distinguishing these conditions requires integration of clinical history, medication exposure, CSF analysis, microbiological testing, neuroimaging, and selected autoimmune investigations. Nursing assessment contributes by documenting symptom onset, medication history, neurological changes, and associated systemic manifestations.

**Prognosis**

The prognosis of La Crosse encephalitis is generally favorable, particularly among patients who develop mild disease without significant neurological complications. Most infections remain asymptomatic, while symptomatic cases frequently resolve with supportive management. The reported case-fatality rate among diagnosed patients is approximately 1%, although mortality may increase in individuals who develop severe neurological complications. The outcome depends on the extent of central nervous system involvement, the occurrence of prolonged seizures, the development of cerebral edema, and the need for respiratory support. Many patients experience improvement after the acute febrile and neurological phases. Headache, fever, vomiting, and lethargy generally resolve over days to weeks. However, recovery from severe encephalitis may take longer, particularly in children who experience recurrent seizures, prolonged altered consciousness, or other complications. Hospitalization may be necessary for neurological observation, seizure management, and supportive treatment. Patients with severe disease may require intensive care and mechanical ventilation. Although mortality is relatively low, neurological morbidity remains an important concern. Some individuals develop recurrent seizures following recovery from the acute infection. Approximately 5% to 15% of recovered patients may experience recurrent seizures.[13] This finding highlights the importance of neurological follow-up, particularly among children who experienced seizures during hospitalization. In selected patients, recurrent seizures may indicate the development of post-encephalitic epilepsy and require long-term antiseizure treatment.

Long-term outcomes may also involve cognitive, behavioral, and academic difficulties. Children who experience severe neurological involvement may demonstrate problems with attention, memory, learning, or behavioral regulation. These difficulties may become more apparent when they return to school and resume age-appropriate activities. Consequently, recovery should not be evaluated solely through the resolution of fever or normalization of neurological observations during hospitalization. Follow-up assessment may include neurological examination, seizure evaluation, cognitive screening, and review of educational performance. Children with persistent difficulties may benefit from neuropsychological assessment, rehabilitation, and educational support. Nurses contribute to recovery by educating families about possible delayed complications, encouraging adherence to follow-up appointments, and identifying changes in behavior or functional ability. Overall, early recognition of neurological deterioration and appropriate supportive management contribute to favorable outcomes. However, patients with severe encephalitis require continued assessment because neurological sequelae may persist after the acute infection has resolved.

**Complications**

La Crosse encephalitis may produce complications during the acute infection and after clinical recovery. Their severity varies according to the extent of neurological involvement, the occurrence of seizures, and the development of cerebral edema or other intracranial abnormalities. Mild cases may resolve without lasting consequences. However, severe disease can cause critical illness, permanent neurological impairment, and death. Acute complications include hyponatremia, seizures, cerebral edema, brain herniation, basal ganglia hemorrhage, coma, and death.[13] Hyponatremia is an important metabolic complication that may contribute to neurological symptoms and complicate the management of patients with encephalitis. Electrolyte disturbances require laboratory monitoring and appropriate correction based on the underlying clinical condition. Severe hyponatremia may increase the risk of seizures and altered consciousness. Seizures are among the most frequently recognized neurological complications, particularly in pediatric patients. They may occur as focal or generalized events and can progress to status epilepticus. Prolonged or recurrent seizures may require multiple antiseizure medications, continuous EEG monitoring, and intensive care management. Some patients continue to experience seizures after the acute infection has resolved. This may indicate persistent neurological dysfunction or the development of post-encephalitic epilepsy.

Cerebral edema represents another potentially life-threatening complication. Increased intracranial pressure may impair cerebral perfusion and cause progressive neurological deterioration. Severe cases may develop brain herniation, coma, and respiratory compromise. These complications may require airway protection, mechanical ventilation, and intensive neurological monitoring. Basal ganglia hemorrhage has also been documented in patients with severe La Crosse encephalitis.[13] Long-term complications are particularly important in children because neurological injury may affect development, learning, and daily functioning. Reported sequelae include recurrent seizures, cognitive impairment, poor academic performance, behavioral changes, attention deficit hyperactivity disorder, cognitive delays, personality disorders, and neuromotor delay.[2,19,21,22] Cognitive difficulties may involve memory, concentration, information processing, and learning. Behavioral changes may affect social interaction, emotional regulation, and school participation. Neuromotor complications may interfere with coordination, balance, and the development of age-appropriate motor skills. The presence of long-term sequelae emphasizes the importance of follow-up after hospital discharge. Children who experience severe encephalitis should undergo assessment of neurological function, cognitive development, behavior, and school performance. Rehabilitation services, psychological support, and educational interventions may be necessary when persistent impairments are identified. Nursing care plays an important role in preventing and recognizing complications. Nurses should monitor consciousness, seizure activity, respiratory status, fluid balance, and electrolyte levels. Changes in neurological function should be reported promptly. After discharge, nurses can support families by explaining the importance of follow-up assessments and recognizing possible changes in cognition, behavior, mobility, or seizure frequency.

**Conclusion:**

La Crosse encephalitis is an important mosquito-borne neurological infection that primarily affects children and can produce outcomes ranging from mild febrile illness to severe encephalitis. Its clinical significance arises from the potential development of seizures, cerebral edema, altered consciousness, and persistent neurological complications. Diagnosis requires integration of clinical findings, epidemiological history, cerebrospinal fluid analysis, and virus-specific antibody testing. Because no established specific antiviral treatment is available, management relies on supportive care, seizure control, fluid and electrolyte regulation, and respiratory support in severe cases. Nursing assessment is essential for identifying neurological deterioration, monitoring treatment responses, and preventing complications. Although most patients recover, recurrent seizures and long-term cognitive, behavioral, and developmental difficulties may occur. Follow-up should therefore

extend beyond the acute illness, particularly for children with severe neurological involvement. Public health education, mosquito control, and preventive measures remain important strategies for reducing transmission and disease burden.

**References:**


1. Thompson Wh, Kalfayan B, Anslow Ro. Isolation Of California Encephalitis Group Virus From A Fatal Human Illness. Am J Epidemiol. 1965 Mar;81:245-53.
2. Byrd BD. La Crosse Encephalitis: A Persistent Arboviral Threat in North Carolina. N C Med J. 2016 Sep-Oct;77(5):330-3.
3. Goldman T, Hamer DH. Current Status of La Crosse Virus in North America and Potential for Future Spread. Am J Trop Med Hyg. 2024 May 01;110(5):850-855.
4. Teleron AL, Rose BK, Williams DM, Kemper SE, McJunkin JE. La Crosse Encephalitis: An Adult Case Series. Am J Med. 2016 Aug;129(8):881-4.
5. Woodring J, Chandler LJ, Oray CT, McGaw MM, Blair CD, Beaty BJ. Short report: Diapause, transovarial transmission, and filial infection rates in geographic strains of La Crosse virus-infected Aedes triseriatus. Am J Trop Med Hyg. 1998 May;58(5):587-8.
6. Thompson WH, Beaty BJ. Venereal transmission of La Crosse virus from male to female Aedes triseriatus. Am J Trop Med Hyg. 1978 Jan;27(1 Pt 1):187-96.
7. Darby CS, Featherston KM, Lin J, Franz AWE. Detection of La Crosse Virus In Situ and in Individual Progeny to Assess the Vertical Transmission Potential in *Aedes albopictus* and *Aedes aegypti*. Insects. 2023 Jul 03;14(7)
8. Day CA, Byrd BD, Trout Fryxell RT. La Crosse virus neuroinvasive disease: the kids are not alright. J Med Entomol. 2023 Nov 14;60(6):1165-1182.
9. Vahey GM, Lindsey NP, Staples JE, Hills SL. La Crosse Virus Disease in the United States, 2003-2019. Am J Trop Med Hyg. 2021 Jul 19;105(3):807-812.
10. Jones TF, Erwin PC, Craig AS, Baker P, Touhey KE, Patterson LE, Schaffner W. Serological survey and active surveillance for La Crosse virus infections among children in Tennessee. Clin Infect Dis. 2000 Nov;31(5):1284-7.
11. Faw LR, Riley J, Eastwood G. La Crosse Virus Circulation in Virginia, Assessed via Serosurveillance in Wildlife Species. Infect Dis Rep. 2023 Jun 30;15(4):360-369.
12. Hacker JK, Volkman LE, Hardy JL. Requirement for the G1 protein of California encephalitis virus in infection in vitro and in vivo. Virology. 1995 Feb 01;206(2):945-53.
13. McJunkin JE, Khan RR, Tsai TF. California-La Crosse encephalitis. Infect Dis Clin North Am. 1998 Mar;12(1):83-93.
14. McJunkin JE, Khan R, de los Reyes EC, Parsons DL, Minnich LL, Ashley RG, Tsai TF. Treatment of severe La Crosse encephalitis with intravenous ribavirin following diagnosis by brain biopsy. Pediatrics. 1997 Feb;99(2):261-7.
15. Murphree R, Dunn JR, Schaffner W, Jones TF. La Crosse encephalitis surveillance using single versus paired serologic testing. Zoonoses Public Health. 2012 May;59(3):181-3.
16. McJunkin JE, de los Reyes EC, Irazuzta JE, Caceres MJ, Khan RR, Minnich LL, Fu KD, Lovett GD, Tsai T, Thompson A. La Crosse encephalitis in children. N Engl J Med. 2001 Mar 15;344(11):801-7.
17. Connor E, Morrison S, Lane J, Oleske J, Sonke RL, Connor J. Safety, tolerance, and pharmacokinetics of systemic ribavirin in children with human immunodeficiency virus infection. Antimicrob Agents Chemother. 1993 Mar;37(3):532-9.
18. Hosoya M, Shigeta S, Mori S, Tomoda A, Shiraishi S, Miike T, Suzuki H. High-dose intravenous ribavirin therapy for subacute sclerosing panencephalitis. Antimicrob Agents Chemother. 2001 Mar;45(3):943-5.
19. McJunkin JE, Nahata MC, De Los Reyes EC, Hunt WG, Caceres M, Khan RR, Chebib MG, Taravath S, Minnich LL, Carr R, Welch CA, Whitley RJ. Safety and pharmacokinetics of ribavirin for the treatment of la crosse encephalitis. Pediatr Infect Dis J. 2011 Oct;30(10):860-5.
20. Cassidy LF, Patterson JL. Mechanism of La Crosse virus inhibition by ribavirin. Antimicrob Agents Chemother. 1989 Nov;33(11):2009-11.
21. Haddow AD, Haddow AD. The use of oral ribavirin in the management of La Crosse viral infections. Med Hypotheses. 2009 Feb;72(2):190-2.

Balkhy HH, Schreiber JR. Severe La Crosse encephalitis with significant neurologic sequelae. Pediatr Infect Dis J. 2000 Jan;19(1):77-80