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Herpes infection encephalitis: An overview of diagnosis, treatment, management, and nursing interventions

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Abstract
Background: Herpes simplex encephalitis (HSE) is a severe central nervous system disorder caused by herpes simplex viruses (HSV-1 and HSV-2). Despite advances in antiviral treatments, the condition is associated with high morbidity and mortality. Prompt diagnosis and management are critical to reducing adverse outcomes.
Aim: This study provides an overview of HSE, including its epidemiology, etiology, pathophysiology, clinical manifestations, diagnostic methods, treatment strategies, and nursing interventions.
Methods: A comprehensive literature review was conducted to analyze HSE from historical, clinical, and therapeutic perspectives. Data were collected on the pathogenesis, diagnostic tools such as cerebrospinal fluid polymerase chain reaction (CSF-PCR), and the efficacy of antiviral therapy. Specific attention was given to nursing care in managing HSE patients.
Results: HSE is predominantly caused by HSV-1, with a global incidence of 2–4 cases per million annually. Typical symptoms include fever, altered mental status, seizures, and focal neurological deficits. Diagnosis relies on CSF-PCR, neuroimaging, and clinical evaluation. Treatment with acyclovir significantly reduces mortality but does not eliminate the risk of long-term neurological deficits. Nursing interventions focus on symptom management, patient education, and psychosocial support.
Conclusion: Despite significant advancements in diagnostic and therapeutic strategies, HSE remains in a life-threatening condition. Early identification and comprehensive management, including tailored nursing care, are essential to improve patient outcomes. Multidisciplinary approaches are recommended for optimal care.

Keywords: Herpes simplex encephalitis, HSV-1, antiviral therapy, cerebrospinal fluid analysis, nursing care, neurological deficits

Introduction

Herpetic infections have been extensively recorded, with references dating back to ancient Greek literature. Herpesviruses are characterized as large, double-stranded DNA viruses, with eight distinct types known to affect humans, collectively referred to as human herpes viruses (HHV). Central nervous system (CNS) manifestations of herpesvirus infections typically include symptoms such as fever, headache, seizures, focal neurological deficits, and altered levels of consciousness [1]. Herpes simplex encephalitis represents an acute or subacute neurological disorder linked to focal or diffuse cerebral impairment caused by herpes simplex viruses. These viruses are classified into two types: herpes simplex virus type 1 (HSV-1) and type 2 (HSV-2). Among these, HSV-1 accounts for the vast majority of cases, while HSV-2 contributes to less than 10% of infections [2]. Post-neonatal herpes simplex encephalitis is almost exclusively attributable to HSV-1, which is the most frequent cause of fatal encephalitis worldwide. HSV-1-related encephalitis occurs sporadically and is not influenced by seasonal trends. Conversely, neonatal herpes simplex encephalitis can be caused by both HSV-1 and HSV-2, although the latter is more prevalent in neonates. Additionally, HSV-2 is a notable cause of encephalitis in immunocompromised individuals. HSV-1-related encephalitis in adults carries significant risks of morbidity and mortality, even with the administration of antiviral therapy. In pediatric patients, herpes simplex encephalitis typically has a nonspecific and gradual onset and can lead to severe outcomes regardless of medical intervention [3]. Although herpes simplex encephalitis is an infrequent condition, its untreated form is associated with a mortality rate reaching 70%, and only a small percentage of patients achieve full recovery without residual neurological deficits [2]. Neonatal encephalitis resulting from HSV-2 tends to involve the brain in a more widespread manner, leading to a higher likelihood of long-term neurological complications.

Etiology

Herpes simplex viruses (HSV-1 and HSV-2) are part of the human herpesvirus family, which also includes varicella-zoster virus (HHV-3), Epstein-Barr virus (HHV-4), cytomegalovirus (HHV-5), HHV-6, HHV-7, and

HHV-8 (Kaposi sarcoma-associated herpesvirus). While most human herpesviruses, except HHV-8, are implicated in encephalitis, HSV-1 accounts for approximately 90% of cases, whereas HSV-2 causes the remaining 10%. HSV-1 and HSV-2 are large double-stranded DNA viruses encapsulated in an icosahedral capsid and enveloped by a glycoprotein-rich membrane. Host cell infection is mediated through herpesvirus entry mediator (HVEM) and nectin proteins, with nectin-1 playing a critical role in facilitating viral entry into the nervous system [12]. In adults, the severity of infection is influenced by viral factors and host immune responses. The initial infection involves the herpesvirus infiltrating epithelial cells of the mucosa or skin. The virus is transported retrogradely via peripheral neuron axons to the cell bodies, where it establishes latency. The maintenance of this latent state involves mechanisms such as suppression of viral lytic-phase genes via histone deposition by pattern recognition proteins and cytokine-induced activation of the intrinsic immune system. In immunocompetent hosts, systemic infections are typically avoided due to the activation of both intrinsic and innate immune systems [4][5].

Epidemiology

HSV-1 is the predominant cause of sporadic, life-threatening encephalitis worldwide and exhibits no seasonal variation. Between 60% and 90% of adults globally are seropositive for HSV-1, while HSV-2 prevalence is higher in populations with elevated rates of sexual transmission, particularly among women. Age is another determining factor, with HSV-2 seropositivity increasing with age [6]. A US survey conducted between 2005 and 2010 reported seroprevalence rates of approximately 54% for HSV-1 and 16% for HSV-2 in individuals aged 14 to 49 years. The annual global incidence of herpes simplex encephalitis ranges from 2 to 4 cases per million individuals. Mortality rates without treatment can reach 70%, and even with optimal treatment, mortality remains between 20% and 30% [7]. Viral infections account for 20% to 50% of encephalitis cases, with HSVs responsible for 50% to 75% of these viral cases [8]. In the UK, a multicenter population-based study identified HSV as the leading cause of infectious encephalitis [9], a finding consistent with data from Australia [10]. Similar incidence rates of HSV-1 encephalitis have been reported in Sweden and the United States [7][11]. Although all age groups are affected, children, adolescents, and individuals over 50 years are most frequently impacted. Nearly one-third of cases occur in younger populations, while approximately half affect older adults. Both sexes are equally susceptible [9, 12].

Pathophysiology

Transmission of HSV-1 and HSV-2 requires close interpersonal contact. Initial primary infection involves viral replication within epithelial cells, leading to inflammation, tissue damage, and the formation of characteristic herpes blisters. HSV-1 typically manifests as oral lesions (fever blisters) with recurrent episodes of decreasing severity over time, while HSV-2 predominantly causes genital lesions approximately one to two weeks post-infection. Relapse severity diminishes as host immunity improves [29]. Primary infection accounts for about 30% of herpes simplex encephalitis cases, with the remainder attributed to reactivation or reinfection. HSV-1 is hypothesized to invade the brain via three mechanisms: through trigeminal or olfactory nerves following primary oropharyngeal infection, via reactivation along these neuronal pathways, or through in-situ reactivation of latent HSV-1 within the brain [13]. Neuronal destruction arises from direct viral injury and immune-mediated cell damage. Herpes simplex encephalitis predominantly affects the temporal lobes and limbic system, with diffuse brain involvement in neonates. Immunocompromised patients often exhibit atypical tissue involvement, including lesions in the brainstem, cerebellum, and cerebral cortex [29]. Immunosuppressive therapies, such as natalizumab and TNF-α inhibitors, increase susceptibility to HSV-1 encephalitis. Cases have also been observed following whole-brain radiation therapy [14].

Histopathology

Gross pathological examination reveals lytic and hemorrhagic changes in the medial temporal and inferior frontal lobes. Hemorrhagic encephalitis in orbitofrontal or limbic specimens is considered pathognomonic for herpes simplex encephalitis [15]. In the early stages, neuronal cells exhibit decreased cytoplasm due to ischemia, capillary dilation, and hemorrhage. Cowdry type A inclusions, consisting of eosinophilic intranuclear inclusions, are characteristic of HSV and varicella-zoster virus infections. Viral antigens are predominantly detected in regions such as the medial temporal lobes, hippocampus, and cingulate gyrus. Initial neutrophil infiltration is followed by macrophages and lymphocytes after 10 to 15 days. By three weeks, cellular necrosis is extensive, with accompanying inflammation and gliosis, while viral antigen detection diminishes [8]. In immunocompromised patients, lesions often lack inflammation and hemorrhage, with viral antigens persisting for three weeks. These atypical presentations emphasize the necessity for tailored diagnostic and therapeutic approaches [8].

History and Physical

The clinical manifestations of herpes encephalitis can present acutely or sub acutely, often preceded by a prodromal phase marked by fever, malaise, headache, and nausea. Typical features associated with HSV-1 encephalitis include a persistent alteration in mental status exceeding 24 hours, accompanied by other indications of cerebral inflammation such as fever, headache, seizures, and focal neurological deficits. Changes in cognition, behavior, and personality have been extensively documented. Additional associated symptoms may encompass urinary and fecal incontinence, aseptic meningitis, dermatomal rashes, and Guillain-Barré syndrome. Benign recurrent meningitis represents an atypical manifestation. Behavioral syndromes, including hypomania, Klüver-

Bucy syndrome, and varying levels of amnesia, are commonly observed, with behavioral and personality changes frequently misinterpreted as psychiatric conditions. Prominent physical findings typically include fever and altered mental status. Although meningeal signs may be present, meningism us is observed in fewer than 50% of cases. Neurological deficits often emerge acutely, lasting less than one week, and commonly present as focal cranial nerve palsies, hemiparesis, dysphasia, aphasia, ataxia, visual field abnormalities, or papilledema. Anterior opercular syndrome has occasionally been identified as an initial presentation linked to encephalitis [16].
A substantial study highlighted the prevalence of specific symptoms in herpes simplex encephalitis, including fever (80%), confusion (72%), abnormal behavior (59%), headache (58%), decreased mental acuity (58%), seizures (54%), focal neurological deficits (41%), nausea and vomiting (40%), aphasia or altered speech (40%), coma (33%), and meningism us (28%) [8]. In pediatric cases, clinical presentations such as fever, lethargy, behavioral alterations, drowsiness, and focal seizures or neurological abnormalities are prevalent. Neonatal herpes simplex encephalitis typically manifests within the first three weeks of life, with symptoms including irritability, lethargy, poor feeding, tremors, or seizures. Herpetic skin lesions are frequently observed in neonatal cases, along with altered liver function tests and thrombocytopenia when multi-organ involvement occurs. HSV-2 infections may rarely present with concurrent myelitis. Immunocompromised individuals often exhibit atypical or subtle presentations, characterized by fewer prodromal symptoms and focal neurological deficits compared to immunocompetent patients, complicating the diagnostic process [8]. Certain medications, particularly those influencing T-cell activity, further predispose individuals to herpes simplex encephalitis.

Evaluation

There are no pathognomonic clinical features specific to herpes simplex encephalitis, necessitating expedited diagnostic evaluations to avoid treatment delays. Maintaining a high index of suspicion is critical, especially in immunocompromised individuals presenting with febrile encephalopathy. Historically, brain biopsy was considered the diagnostic gold standard; however, cerebrospinal fluid (CSF) analysis through lumbar puncture now serves this role and should be conducted promptly. Contraindications for lumbar puncture include brain shift, herniation, or other indicators of elevated intracranial pressure [8]. Typical CSF findings include elevated opening pressure, increased protein concentration, normal glucose levels, and lymphocytic pleocytosis. Elevated CSF white blood cell counts exhibit 95% sensitivity but may be absent during early infection, in pediatric patients, or in immunocompromised individuals. Temporal hemorrhage commonly results in elevated red blood cell counts in the CSF. Polymerase chain reaction (PCR) testing for HSV-1 and HSV-2 demonstrates sensitivities and specificities of 96% and 99%, respectively [8]. PCR results are generally positive during the initial week of acyclovir therapy and tend to normalize after 10–14 days of treatment.
False-negative PCR results may arise due to low viral loads within the first 72 hours, blood presence in the CSF interfering with the test, or prolonged acyclovir administration exceeding 48 hours. When clinical suspicion remains high despite an initially negative PCR obtained within three days of symptom onset, retesting is recommended. The 2008 Infectious Diseases Society of America (IDSA) guidelines advise continued acyclovir therapy with repeated PCR analysis within 3–7 days under such circumstances. PCR positivity for HSV in the bloodstream has been noted in neonates and immunocompromised patients, offering an indirect diagnostic alternative when lumbar punctures are temporarily unfeasible. The intrathecal synthesis of HSV-specific IgM antibodies can be pivotal in diagnosing HSV encephalitis, particularly when CSF samples are unavailable or acquired more than one week after treatment initiation [17]. Sole reliance on serum serology proves ineffective due to the widespread prevalence of HSV IgG antibodies in the general population. Detecting CSF antibodies against purified HSV glycoprotein B yields a sensitivity of 97% and a specificity of 100%. Although viral antibody titers typically rise four-fold during illness, detection is only possible after 10–14 days, thus aiding in delayed diagnoses.
Routine hematological assessments, including complete blood count, coagulation studies, and basic metabolic panels, may reveal lymphocytosis or remain within normal limits. Imaging studies frequently commence with a computerized tomography (CT) scan to assess midline shifts, hemorrhage, or increased intracranial pressure. While less sensitive than magnetic resonance imaging (MRI), CT scans can identify hypodensities, hemorrhage, and edema, with contrast enhancement appearing after approximately one week. MRI offers superior diagnostic accuracy, with more than 90% sensitivity in detecting abnormalities associated with herpes simplex encephalitis. Classic MRI findings involve the temporal lobes, occasionally extending to the limbic system, inferior frontal lobes, and insular cortex. Atypical patterns, including extra-frontotemporal involvement, are more common in pediatric and immunocompromised populations. Electroencephalograms (EEGs) are often abnormal, reflecting the epileptogenic nature of herpes simplex encephalitis. Recurrent sharp-and-slow complexes originating in the temporal lobes, periodic lateralized epileptiform discharges (PLEDs), or prominent high-amplitude slow waves are characteristic EEG findings [18].

Differential Diagnosis

Conditions that mimic encephalopathy or encephalitis should be thoroughly considered in differential diagnoses. Among the potential mimicking conditions are:

1. **Viral Causes:** Encephalitis caused by other viruses can present with similar symptoms. Examples include herpesviruses such as Epstein-Barr virus (EBV), cytomegalovirus (CMV), varicella-zoster virus (VZV), and human herpesviruses 6 and 7 (HHV-6 and HHV-7). Arboviral infections like West Nile virus and St. Louis encephalitis, along with Eastern and Western equine encephalitis, California encephalitis, Japanese encephalitis, Rubulavirus (mumps), enterovirus, adenovirus, and dengue virus, are noteworthy.

2. **Bacterial and Other Pathogenic Causes:** Infections caused by bacteria, mycobacteria, prions, fungi, or parasites may lead to primary or secondary central nervous system (CNS) infections, mimicking encephalitis.
3. **Hypoxemic and Septic Encephalopathies:** These conditions can produce similar mental status changes and focal neurological symptoms.
4. **Noninfectious Causes:** Autoimmune or paraneoplastic encephalitis are critical noninfectious causes that may resemble encephalitis.
5. **Metabolic Disorders:** Conditions such as hepatic or uremic encephalopathies, Wernicke encephalopathy, mitochondrial disorders, and electrolyte imbalances (e.g., hypo- or hypernatremia, hypo- or hypercalcemia) should also be considered.
6. **Neurovascular and Other Systemic Causes:** Brain tumors, seizure disorders, vasculitis, neurosyphilis, and traumatic brain injuries may mimic encephalitic presentations. Systemic diseases like systemic lupus erythematosus (SLE) and Behçet's disease, as well as rare conditions like progressive multifocal leukoencephalopathy due to JC virus, are potential differential diagnoses.
7. **Rare Neurological Disorders:** Subacute sclerosing panencephalitis, though rare with current vaccination efforts, can occur years after measles infection in children, presenting with progressive neurological deterioration.

Pertinent Studies and Ongoing Trials

A prospective, multinational, randomized, placebo-controlled trial assessed the efficacy of adjuvant oral valacyclovir in reducing neuropsychological sequelae in herpes simplex encephalitis patients. In the trial involving 87 participants, valacyclovir (6 g daily) was administered for three months following standard intravenous acyclovir therapy. Results indicated no significant benefit of this extended treatment [21]. The utility of adjuvant corticosteroids in herpes simplex encephalitis remains a contentious issue. A retrospective study involving 45 patients examined the role of corticosteroids administered alongside acyclovir. Through stepwise logistic regression, the study identified advanced age, low Glasgow Coma Scale (GCS) scores at acyclovir initiation, and the absence of corticosteroid therapy as predictors of poor outcomes [25]. The GACHE trial, a multicenter, randomized, double-blind study, evaluated 40 mg dexamethasone for four days alongside two weeks of acyclovir therapy. Unfortunately, recruitment challenges led to inconclusive results [27]. The ongoing Dex-Enceph trial aims to determine the effects of a 4-day dexamethasone regimen on verbal memory scores in herpes simplex encephalitis patients [28].

Treatment Planning

Acyclovir requires careful preparation to avoid complications. For intravenous administration, the drug should be diluted to a concentration below 7 mg/mL. For a 70 kg individual, a dose of 700 mg should be diluted in at least 100 mL of solution and infused over an hour, accompanied by fluid boluses to prevent crystalluria and renal dysfunction. Administration via intramuscular or subcutaneous routes is contraindicated due to the drug's alkaline nature, which can cause inflammation and phlebitis. Dose adjustments based on renal function are detailed as follows:

- **CrCl >50 mL/min:** No adjustment needed.
- **CrCl 25–50 mL/min**: Administer the usual dose every 12 hours.
- **CrCl 10–25 mL/min**: Administer the usual dose every 24 hours.
- **CrCl ≤10 mL/min**: Administer half the usual dose every 24 hours.
- **Intermittent hemodialysis**: 5 mg/kg after dialysis sessions.
- **Peritoneal dialysis**: 5 mg/kg daily without supplementation.
- **Continuous renal replacement therapy (CRRT)**: 10 mg/kg every 12 hours.

Prognosis

Herpes simplex encephalitis in adults carries significant morbidity and mortality rates, even with timely diagnosis and treatment. Mortality estimates range between 20–30% [22, 23]. Factors such as an APACHE score exceeding 27 and delays exceeding 48 hours in initiating acyclovir therapy correlate with poor outcomes [20]. Neonates and children, particularly those untreated, experience heightened morbidity and mortality risks [3]. Immunocompromised individuals are especially vulnerable, with mortality rates reaching 36%, compared to 7% in immunocompetent patients [28]. Long-term complications include cognitive and behavioral abnormalities, anterograde amnesia, and symptoms resembling Klüver-Bucy syndrome. Survivors often exhibit difficulties with memory and learning, even when standard mental status examinations yield normal results. A Swedish study highlighted high rehospitalization rates among survivors, primarily due to seizure episodes, neuropsychiatric symptoms, and thromboembolic events [7].

Complications

Short-term complications encompass cerebral edema, status epilepticus, elevated intracranial pressure, aspiration pneumonitis, cerebral venous thrombosis, cerebral infarction, and diabetes insipidus. Long-term sequelae include neurological deficits such as aphasia, ataxia, dysphasia, and amnesia, along with cognitive, behavioral, and neuropsychiatric abnormalities. Approximately 70% of patients report enduring cognitive and behavioral challenges [5].

Possible Link Between HSV and Alzheimer's Disease

Evidence suggests that herpes simplex virus (HSV) infection may predispose individuals to Alzheimer's disease (AD) later in life. The temporal lobes and basal forebrain, regions primarily affected by HSV, are also implicated in AD pathology [29]. Survivors of herpes simplex encephalitis often exhibit anterograde and retrograde memory loss akin to AD symptoms. Moreover, a significant correlation exists between the APO-E4 allele and HSV genome loads, suggesting that HSV latency coupled with APO-E4 presence may increase susceptibility to AD [29].

Consultations

Management of encephalitis necessitates interdisciplinary collaboration. Neurology consultations are essential for diagnosis and treatment planning, while infectious disease specialists are invaluable when no infectious etiology is identified, or the patient's condition deteriorates. Neurosurgeons may be consulted for cases involving significant brain involvement or when a biopsy is warranted. Rehabilitation specialists play a critical role in addressing short- and long-term neurorehabilitation needs, and psychological or psychiatric support may be required to manage neuropsychiatric sequelae. Pediatric and neonatology experts are indispensable for managing younger patients, often in conjunction with pediatric infectious disease and neurology specialists.

Patient Education

Despite advances in detection and treatment, herpes simplex encephalitis is associated with considerable morbidity and mortality. Survivors often experience persistent neurological or neuropsychiatric disorders. Preventative strategies are currently unavailable for older children and adults. Neonatal transmission can be minimized through cesarean delivery for pregnant women with active herpes labialis and by preventing contact between neonates and individuals with active HSV infections.

Nursing Intervention Plans

Effective nursing intervention plans are essential in managing patients with complex medical conditions, including herpes simplex encephalitis (HSE). These plans should be structured to address both the immediate needs of the patient and long-term complications, ensuring holistic care. Initially, the nursing focus is on stabilizing the patient and preventing further complications. This includes continuous monitoring of vital signs, neurological status, and signs of increased intracranial pressure. Nurses must ensure timely administration of prescribed antiviral therapy, such as intravenous acyclovir, adhering strictly to dosage guidelines to prevent nephrotoxicity. For patients with impaired renal function, the dose adjustments must align with creatinine clearance levels, as specified in the treatment protocol. Preventive measures, such as adequate hydration and avoidance of extravasation during drug infusion, are critical in minimizing complications.

Another key component involves managing acute symptoms like seizures or altered mental status. This requires close collaboration with the medical team to administer anticonvulsants or sedatives as necessary. Airway management is essential, particularly for patients with reduced consciousness or at risk of aspiration. Nurses should ensure the availability and readiness of equipment for suctioning and oxygen therapy, maintaining adequate oxygenation levels at all times. Long-term care interventions are equally vital, focusing on rehabilitation and psychosocial support. Nurses play a pivotal role in coordinating multidisciplinary consultations, including neurology, infectious diseases, and rehabilitation services, to facilitate recovery. Educating patients and families about potential cognitive and behavioral changes, such as memory loss or emotional disturbances, fosters preparedness and compliance with follow-up care. Emotional support through counseling and psychological interventions aids in coping with the long-term effects of the condition. By integrating comprehensive monitoring, acute symptom management, and psychosocial support, nursing intervention plans ensure that patients receive holistic and patient-centered care, ultimately enhancing outcomes and quality of life.

Conclusion

Herpes simplex encephalitis (HSE) represents a critical challenge in neurological and infectious disease care. The condition, predominantly caused by HSV-1, affects individuals across all age groups and presents nonspecific symptoms such as fever, headache, and altered mental status. These manifestations, coupled with their potential for rapid progression to life-threatening complications, highlight the importance of early diagnosis and intervention. Advanced diagnostic modalities, particularly cerebrospinal fluid polymerase chain reaction (CSF-PCR), have become the cornerstone of HSE identification, demonstrating high sensitivity and specificity. However, the risk of false negatives underscores the need for clinical vigilance. Treatment with intravenous acyclovir has markedly reduced HSE-associated mortality, yet significant morbidity persists, particularly neurological sequelae. This emphasizes the need for comprehensive care approaches that address both acute treatment and long-term recovery. Nursing interventions play a pivotal role in managing HSE patients. These include monitoring neurological status, managing seizures and other acute symptoms, preventing complications such as pressure ulcers, and providing psychosocial support. Patient and caregiver education are vital to enhance adherence to follow-up care and mitigate anxiety related to the disease's severe outcomes. HSE also poses challenges for immunocompromised individuals and neonates, necessitating tailored diagnostic and therapeutic strategies. Multidisciplinary collaboration involving neurologists, infectious disease specialists, and nursing teams is critical for holistic care. Future research should focus on novel therapeutic agents, early biomarkers for disease progression, and strategies to enhance neuroprotection and functional recovery. In summary,

advancements in antiviral therapy and diagnostic tools have improved HSE management, early recognition, prompt treatment, and comprehensive nursing care remain integral to optimizing patient outcomes. Increased awareness and continuous education among healthcare providers are essential to mitigate the devastating impact of this disease.

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التهاب الدماغ الناتج عن عدوى الهربس: نظرة عامة على التشخيص والعلاج والإدارة والتدخلات التمريضية

الملخصz:

الخلفية :التهاب الدماغ الناتج عن الهربس (HSE) هو اضطراب خطير في الجهاز العصبي المركزي تسببه فيروسات الهربس البسيط 1-HSV) و .(2-HSVعلى الرغم من التقدم في العلاجات المضادة للفيروسات، إلا أن هذه الحالة ترتبط بمعدلات عالية من الممراضة والوفيات. يعد التشخيص والإدارة السريعة أمرين حاسمين للحد من النتائج السلبية.

الهدف :تقدم هذه الدراسة نظرة عامة على التهاب الدماغ الناتج عن الهربس، بما في ذلك علم الأوبئة، والأسباب، والفيزيولوجيا المرضية، والمظاهر السريرية، وطرق التشخيص، واستراتيجيات العلاج، والتدخلات التمريضية.

المنهجية :تم إجراء مراجعة شاملة للأدبيات لتحليل التهاب الدماغ الناتج عن الهربس من وجهات نظر تاريخية وسريرية وعلاجية. تم جمع البيانات حول الفيزيولوجيا المرضية، وأدوات التشخيص مثل تحليل البوليميراز المتسلسل للسائل النخاعي(CSF-PCR) ، وفعالية العلاج المضاد للفيروسات. تم التركيز بشكل خاص على رعاية التمريض في إدارة مرضى التهاب الدماغ الناتج عن الهربس.

النتائج :يحدث التهاب الدماغ الناتج عن الهربس بشكل أساسي بسبب فيروس1-HSV ، مع معدل حدوث عالمي يتراوح بين 2-4 حالات لكل مليون شخص سنويًا. تشمل الأعراض النموذجية الحمى، وتغير الحالة العقلية، والنوبات، والعجز العصبي الموضعي. يعتمد التشخيص على تحليلCSF-PCR ، والتصوير العصبي، والتقييم السريري. يقلل العلاج باستخدام أسيكلوفير من معدل الوفيات بشكل كبير، ولكنه لا يقضي على خطر العجز العصبي طويل الأمد. تركز التدخلات التمريضية على إدارة الأعراض، وتثقيف المرضى، والدعم النفسي الاجتماعي.

الخلاصة :على الرغم من التقدم الكبير في استراتيجيات التشخيص والعلاج، لا يزال التهاب الدماغ الناتج عن الهربس حالة تهدد الحياة. يعتبر التعرف المبكر والإدارة الشاملة، بما في ذلك الرعاية التمريضية المصممة، ضروريين لتحسين نتائج المرضى. يُوصى باتباع نهج متعدد التخصصات لتحقيق رعاية مثلى.

الكلمات المفتاحية: التهاب الدماغ الناتج عن الهربس البسيط، 1-HSV، العلاج المضاد للفيروسات، تحليل السائل النخاعي، رعاية التمريض، العجز العصبي.