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CASE REPORT

Intracranial hypertension due to Epstein-Barr virus encephalitis during the postpartum period

Hipertensión intracraneal debido a encefalitis por virus de Epstein-Barr durante el período posparto

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Palabras clave
Adulto; edema cerebral; líquido cefalorraquídeo; infecciones por el virus de Epstein-Barr; presión intracraneal; hipertensión intracraneal; resultado del tratamiento; período posparto; encefalitis.

Abstract

Case Description:

A 28-year-old postpartum woman presented with severe headache, fever, and decreased level of consciousness. Cerebrospinal fluid PCR detected 185,000 copies of Epstein-Barr virus DNA.

Clinical Findings:

Neuroimaging revealed diffuse cerebral edema consistent with intracranial hypertension in the absence of hemorrhagic lesions, confirming Epstein-Barr virus encephalitis complicated by objectively documented elevated intracranial pressure.

Treatment and Outcomes:

The patient received intravenous acyclovir, phenytoin, and hypertonic saline, achieving complete neurological recovery after 21 days of therapy.

Clinical Relevance:

To our knowledge, this is the first documented case of non-hemorrhagic Epstein-Barr virus encephalitis with objectively confirmed intracranial hypertension in the postpartum period. The most plausible mechanism is inflammation-induced diffuse cerebral edema leading to impaired cerebrovascular autoregulation and subsequent intracranial pressure elevation. Clinicians should include Epstein-Barr virus infection in the differential diagnosis of postpartum patients presenting with encephalitis and signs of increased intracranial pressure, even when neuroimaging does not reveal hemorrhagic findings.

CRediT authorship contribution statement

MFG: Conceptualization, Investigation, Writing - original draft, Writing - review & editing. **FG**: Conceptualization, Supervision, Writing - review & editing, Validation. **NR**: Investigation, Resources, Writing - review & editing. **JJ**: Investigation, Resources, Writing - review & editing. **DP**: Visualization, Formal analysis, Writing - review & editing.

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Resumen

Descripción del caso:

Mujer de 28 años en período posparto que consultó por cefalea intensa, fiebre y disminución del nivel de conciencia. La PCR en el líquido cefalorraquídeo detectó 185,000 copias de ADN del virus de Epstein-Barr.

Hallazgos clínicos:

La neuroimagen evidenció edema cerebral difuso compatible con hipertensión intracraneal y ausencia de lesiones hemorrágicas, lo que confirma encefalitis por virus de Epstein-Barr complicada con elevación objetivamente documentada de la presión intracraneal.

Tratamiento y desenlaces:

La paciente recibió aciclovir intravenoso, fenitoína y solución salina hipertónica y logró una recuperación neurológica completa tras 21 días de tratamiento.

Relevancia clínica:

A nuestro conocimiento, este es el primer caso documentado de encefalitis no hemorrágica por el virus de Epstein-Barr con hipertensión intracraneal objetivamente confirmada en el período posparto. El mecanismo más probable es el edema cerebral difuso inducido por inflamación, que conduce a una alteración de la autorregulación cerebrovascular y a una elevación subsecuente de la presión intracraneal. Los clínicos deben incluir la infección por el virus de Epstein-Barr en el diagnóstico diferencial de pacientes puérperas con encefalitis y signos de hipertensión intracraneal, incluso cuando la neuroimagen no muestre hallazgos hemorrágicos.

Introduction

Encephalitis is an inflammatory process of the brain parenchyma associated with neurological dysfunction, caused by viruses (69%), bacteria (20%), prions (7%), parasites (3%), and fungi (1%). It is more frequently observed in infants under 1 year of age (11.1 cases per 100,000 population) and in individuals over 65 years of age (13.2 cases per 100,000 population) [1].

Epstein-Barr virus remains latent in over 90% of adults and can cause infectious mononucleosis, a relatively common illness among young adults that is typically self-limiting. However, approximately 1% of patients with infectious mononucleosis may develop central nervous system complications. Compared to central nervous system complications from enteroviruses, echoviruses, coxsackieviruses, or herpes simplex virus, Epstein-Barr virus-related central nervous system manifestations are rare and predominantly observed in Human immunodeficiency virus-positive individuals [2].

To our knowledge, only one case of Epstein-Barr virus encephalitis complicated by intracranial hypertension has been reported, which was associated with hemorrhagic encephalitis [3]. The present case is particularly relevant, as it describes a non-hemorrhagic intracranial hypertension secondary to Epstein-Barr virus encephalitis in a 28-year-old postpartum woman.

Case report

We present the case of a mestiza woman between 20 and 30 years of age in the postpartum period following her first pregnancy, delivered at 36 weeks of gestation by cesarean section. The patient initially developed a holocranial headache with a 15-day evolution, associated with fever and progressive alteration of consciousness. Due to symptom persistence, she was assisted at a primary health care center, where she received intravenous analgesia (paracetamol 1 g intravenously, single dose), with symptomatic improvement, after which she was discharged.

Twenty-four hours later, her symptoms worsened, and she returned to the emergency room. Neurological examination revealed somnolence with a Glasgow Coma Scale score of 11/15 (E3V3M5), right-sided brachiocrural hemiparesis, and no cranial nerve deficits. Pupils were isochoric and reactive to light. No signs of meningeal irritation were observed, including neck stiffness, Kernig, or Brudzinski signs. Fundoscopic examination could not be adequately performed at the time of admission due to the patient's clinical condition. The patient reported persistent severe headache, with no episodes of emesis at presentation.

During her stay in the emergency room, the patient experienced multiple episodes of emesis, followed by a generalized tonic-clonic seizure characterized by ocular deviation and sphincter relaxation. In the postictal period, she exhibited acute neurological deterioration, with a decrease in the Glasgow Coma Scale score from 11/15 to 8/15 (E2V2M4).

Neurological examination revealed a depressed level of consciousness and persistence of right-sided brachiocrural hemiparesis. Pupils were isochoric and reactive to light, and no signs of meningeal irritation were identified, including neck stiffness, Kernig, or Brudzinski signs. The patient continued to report severe, persistent headache, suggestive of intracranial hypertension; however, fundoscopic examination could not be reliably performed due to her clinical condition.

In view of the acute neurological deterioration, airway protection with invasive mechanical ventilation was initiated, and a loading dose of intravenous phenytoin (1 g infused over 30 minutes) was administered. Given the rapid clinical decline and the need for continuous hemodynamic and neurological monitoring, the patient was subsequently transferred to the intensive care unit.

On the first day of admission to the intensive care unit, laboratory studies revealed an elevated C-reactive protein level of 203 mg/L, consistent with marked systemic inflammation. Given the clinical suspicion of intracranial hypertension, a non-contrast cranial computed tomography scan was obtained. Axial sections demonstrated decreased differentiation between gray and white matter, sulcal effacement, and ventricular compression, findings consistent with diffuse cerebral edema (Figure 1). Based on clinical deterioration, neuroimaging findings, and concern for elevated intracranial pressure, the neurosurgery team placed an intracranial pressure monitor, which initially showed a pressure of 20 mmHg, confirming the diagnosis of intracranial hypertension.

Management included hyperosmolar therapy with hypertonic saline (3% sodium chloride, administered as intermittent boluses of 2-5 mL/kg and titrated to maintain serum sodium levels between 145 and 155 mEq/L), along with standard neuroprotection measures. These included head-of-bed elevation at 30 degrees, adequate sedation and analgesia (sedation with propofol at 2 mg/kg/h and analgesia with fentanyl at 2 µg/kg/h by continuous infusion), avoidance of hyperthermia, maintenance of normoglycemia and normovolemia, and controlled ventilation to maintain normocapnia.

On the second day of admission to the intensive care unit, previously instituted hyperosmolar therapy and neuroprotection measures were continued. Antiepileptic treatment was maintained with intravenous phenytoin at a maintenance dose of 100 mg every 8 hours, following the initial loading dose, to prevent recurrent seizures. During this period, intracranial pressure decreased progressively, reaching values between 16 and 17 mmHg, indicating a favorable response to

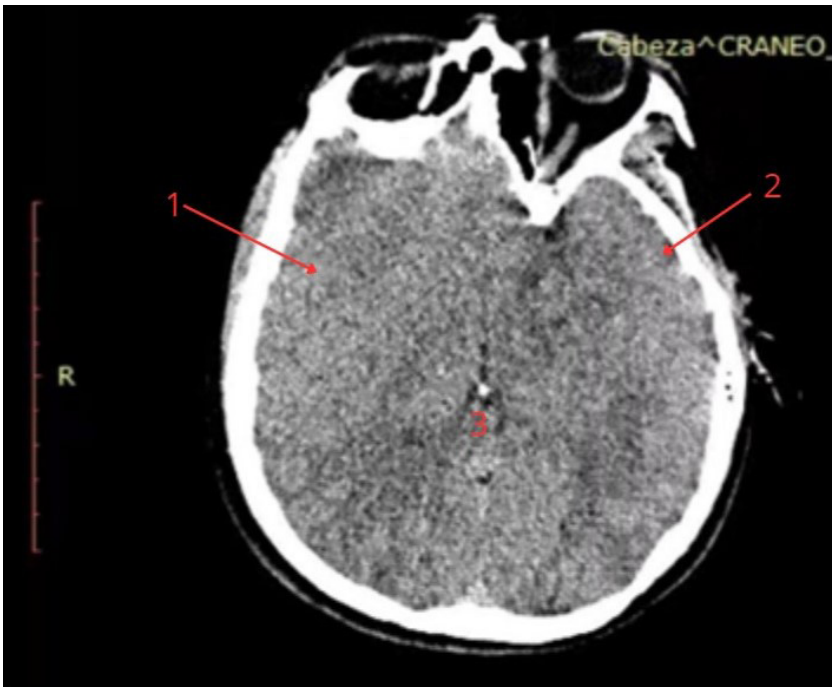

Figure 1. Axial Simple Head CT Scan Showing Diffuse Cerebral Edema and Features of Intracranial Hypertension at the lateral ventricles. Findings from the axial simple head CT scan include: 1) Decreased differentiation between gray and white matter is observed, suggesting the presence of diffuse cerebral edema associated with increased intracranial pressure. 2) Effacement of the cortical sulci is noted, which suggests increased intracranial pressure due to cerebral swelling. 3) A reduction in the size of the lateral ventricles is observed, consistent with the presence of intracranial hypertension, indicating displacement of cerebrospinal fluid due to cerebral edema.

therapy. Clinically, manifestations suggestive of intracranial hypertension, such as recurrent emesis, gradually diminished; however, the patient continued to exhibit neurological findings consistent with encephalitis, with a persistently altered level of consciousness corresponding to a Glasgow Coma Scale score of 12/15, as well as focal neurological deficits.

On the third day of admission to the intensive care unit, after intracranial pressure was stabilized at 13 mmHg, the intracranial pressure monitoring device was safely removed. Subsequently, a lumbar puncture was performed to investigate the underlying etiology of the encephalitic process. Cerebrospinal fluid analysis revealed a positive polymerase chain reaction for Epstein-Barr virus, with a viral load of 185,000 copies/mL. At this time, clinical manifestations of intracranial hypertension, including vomiting, had resolved; however, the patient continued to exhibit persistent neurological impairment, with an altered level of consciousness corresponding to a Glasgow Coma Scale score of 13/15.

Given the persistence of encephalitic features despite adequate control of intracranial pressure, brain magnetic resonance imaging was obtained to further characterize parenchymal involvement and support the etiological diagnosis. MRI demonstrated hyperintense lesions on FLAIR, diffusion-weighted imaging, and apparent diffusion coefficient sequences involving the bilateral medial temporal lobes, predominantly on the left, as well as the medial frontal regions (Figure 2). This imaging pattern was highly suggestive of viral encephalitis, supporting the diagnosis of Epstein-Barr virus encephalitis rather than a purely pressure-related or vascular process.

The final diagnosis was Epstein-Barr virus encephalitis occurring during the postpartum period. The patient was treated with intravenous acyclovir at a dose of 10 mg/kg every 8 hours for a total duration of 21 days. After 10 days of antiviral therapy in the intensive care unit, marked clinical stabilization was achieved, characterized by sustained control of intracranial pressure, resolution of seizures, absence of emesis, and improvement in the level of consciousness.

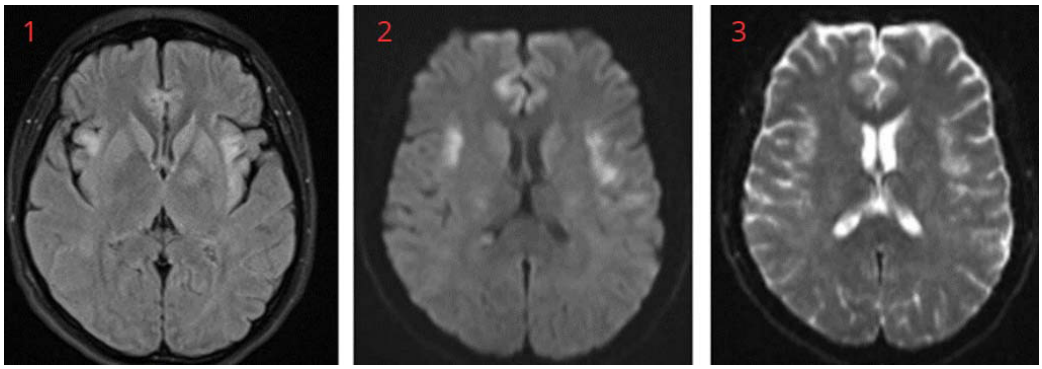

Figure 2. Magnetic Resonance Imaging of the brain in axial view at the level of the basal ganglia and lateral ventricles, showing signs of intracranial hypertension and cerebral edema. The axial Magnetic Resonance Imaging scans demonstrate the following findings: 1) FLAIR Sequence: A hyperintense lesion is evident at the bilateral medial temporal level, predominantly on the left, suggesting a possible inflammatory or ischemic process affecting this region. 2) Diffusion-Weighted Imaging (DWI): The lesion demonstrates restricted diffusion in the same area. 3) Apparent Diffusion Coefficient (ADC): The sequence confirms the presence of low values within the lesion in the affected regions, including the medial frontal area.

Given this favorable evolution, the patient was transferred from the intensive care unit to the general ward to complete the remaining antiviral course. By day 15 of antiviral treatment, complete resolution of symptoms associated with intracranial hypertension and encephalitis was documented, with recovery of consciousness to a Glasgow Coma Scale score of 15/15 and no residual focal neurological deficits. The patient completed a total of 21 days of antiviral therapy with acyclovir and was subsequently discharged home on oral antiepileptic therapy with phenytoin at a maintenance dose of 300 mg per day, divided into three doses of 100 mg every 8 hours, to prevent seizure recurrence. Outpatient neurological follow-up was scheduled for four weeks, with clinical reassessment and consideration of antiepileptic tapering depending on clinical status.

Given the patient's critical neurological condition and need for intensive care management, breastfeeding was temporarily suspended during the acute phase of the illness. Lactation management was addressed once neurological stabilization was achieved.

Ethical declarations

This case was deemed exempt from ethical committee review as it describes a single clinical case without experimental intervention. Written informed consent was obtained directly from the patient for the publication of this case and any accompanying images. Dr. Daniel Peñaherrera Vasquez is the guarantor of this article.

Discussion

Epstein-Barr virus remains latent in more than 90% of adults. Central nervous system involvement is uncommon, occurring in approximately 1% of cases, and is considerably rarer when compared with neurological complications caused by enteroviruses, echoviruses, coxsackieviruses, or herpes simplex virus [2].

Among the neurological abnormalities associated with Epstein-Barr virus infection, only four cases in the literature have described cerebral edema or hemorrhage [3-6], and to date, only one case has documented intracranial hypertension in the setting of hemorrhagic Epstein-Barr virus encephalitis [3]. Intracranial hypertension in Epstein-Barr virus encephalitis is therefore exceptionally rare and, when reported, is most commonly associated with hemorrhagic complications. This phenomenon is thought to result from immune-mediated vascular injury, including vasculitis, endothelial dysfunction, and microvascular damage, which predispose to intracerebral hemorrhage when the inflammatory response is sufficiently intense to disrupt vascular integrity and significantly increase intracranial pressure [3,7].

In contrast, Epstein-Barr virus is not primarily neurotropic; central nervous system involvement is more frequently mediated by lymphocytic infiltration, activation of proinflammatory cytokines, and disruption of the blood-brain barrier rather than direct neuronal cytopathic effects.

These mechanisms promote a combination of vasogenic and cytotoxic edema by increasing capillary permeability, impairing cerebrovascular autoregulation, and altering cerebrospinal fluid dynamics. However, inflammatory edema alone is usually limited in extent and insufficient to generate clinically significant intracranial hypertension [7,8]. The development of marked intracranial hypertension in the absence of hemorrhage, as observed in the present case, suggests a predominant role of diffuse inflammation-induced cerebral edema and autoregulatory failure without overt vascular rupture, representing an exceptionally uncommon pathophysiological presentation.

The ideal diagnostic approach for suspected viral encephalitis complicated by intracranial hypertension involves early recognition of encephalopathy and seizure activity, followed by prompt neuroimaging and cerebrospinal fluid analysis. Magnetic resonance imaging is the modality of choice, as it is more sensitive than computed tomography for detecting early parenchymal involvement, particularly in limbic encephalitis, and for differentiating inflammatory, vascular, and demyelinating processes [9].

Lumbar puncture with comprehensive cerebrospinal fluid analysis, including polymerase chain reaction testing for neurotropic viruses, should be performed as early as safely possible [10]. In cases with clinical or radiological signs of raised intracranial pressure, invasive intracranial pressure monitoring may be required before lumbar puncture to avoid secondary neurological injury [7].

In the presented case, the diagnostic approach was guided by disease severity, prioritizing initial cranial computed tomography and intracranial pressure monitoring for suspected intracranial hypertension, thereby delaying cerebrospinal fluid analysis. Once pressure was controlled, lumbar puncture confirmed Epstein-Barr virus infection, and magnetic resonance imaging supported the diagnosis of Epstein-Barr virus encephalitis, underscoring the importance of a stepwise, safety-driven approach.

The differential diagnosis of this presentation was broad and particularly challenging, given the rarity of Epstein-Barr virus-associated encephalitis complicated by isolated cerebral edema and intracranial hypertension. Initial considerations included herpes simplex virus encephalitis, autoimmune encephalitis, acute disseminated encephalomyelitis, cerebral venous sinus thrombosis, and postpartum-related cerebrovascular disorders such as reversible cerebral vasoconstriction syndrome or posterior reversible encephalopathy syndrome [7,8]. The presence of focal neurological deficits, seizures, and temporal lobe involvement on imaging strongly suggested an encephalitic process; however, the prominent intracranial hypertension without evidence of hemorrhage or venous thrombosis made the diagnostic process especially complex.

Optimal therapy for Epstein-Barr virus encephalitis remains controversial and is not standardized, as no antiviral agents are officially approved for this indication. In published case series, acyclovir has been the most frequently used antiviral agent, often initiated empirically because of overlap with protocols for herpes simplex virus encephalitis. In a systematic review of nearly 100 Epstein-Barr virus encephalitis cases, acyclovir was used in approximately 70% of treated patients [7]. Ganciclovir has been considered a potentially more effective option in Epstein-Barr virus infection due to its greater ability to penetrate the blood-brain barrier and inhibit viral replication, and small clinical reports have supported its use when acyclovir fails or is insufficient. In some individual reports, patients with Epstein-Barr virus encephalitis showed clinical improvement after switching from acyclovir to ganciclovir, supporting its consideration in refractory cases [9]. Corticosteroids and immunomodulatory therapies have also been used to control inflammation and cerebral edema, although high-quality evidence is lacking [11].

In the present case, the patient was treated with intravenous acyclovir, intracranial pressure targeted management, and supportive care, as ganciclovir was not available at our institution at the time of treatment. This case represents the first reported instance of non-hemorrhagic Epstein-Barr virus encephalitis occurring in the postpartum period and complicated by intracranial hypertension secondary to cerebral edema alone. Owing to the extreme rarity of this presentation, delays in diagnosis and mismanagement are common. Recognizing Epstein-Barr virus as a rare but plausible cause of intracranial hypertension due to non-hemorrhagic encephalitis is therefore crucial for early detection, as timely etiological diagnosis and appropriate neurocritical care may prevent potentially fatal complications.

Conclusion

Epstein-Barr virus remains latent in more than 90% of adults and primarily causes infectious mononucleosis. However, it is important to consider the potential development of central nervous system complications, such as intracranial hypertension, which is rarely reported in the medical literature. The medical community should be aware of and attentive to this condition to ensure early and appropriate treatment, thereby preventing further complications.

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