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REPORTE DE CASO

Varicella zoster: uncommon stroke mimic in a young patient without cerebral ischemia - A case report

Luisa Rodríguez-Rosero[1,*], Raúl Andres Vallejo-Serna[2], Jenny Patricia Muñoz-Lombo[3]

Abstract

Varicella-zoster virus (VZV) is a neurotropic alpha-herpesvirus that, after primary infection, remains latent in nerve ganglia and can reactivate, affecting the central nervous system. The manifestations include meningitis, encephalitis, cranial neuropathies, and vascular events. We present the case of a previously healthy 32-year-old woman who presented with sudden onset of severe headache, followed by right peripheral facial palsy and ipsilateral hemiparesis. Neurological examination revealed hearing loss, right VII (facial) and VI (abducens) cranial nerve palsy, and decreased strength and sensory deficit on the right side of her body. Computed tomography and CT angiography results were normal, and no ischemic lesions were identified. Brain magnetic resonance imaging revealed inflammatory changes in the sixth and seventh cranial nerves. Cerebrospinal fluid (CSF) analysis showed lymphocytic pleocytosis and elevated protein levels, and the meningoencephalitis panel was positive for VZV. Additional autoimmune, cardiovascular, and infection-related causes were ruled out. Intravenous acyclovir (10 mg/kg every 8 h for 14 days) and oral prednisolone (60 mg/day for 10 days) were initiated, resulting in significant clinical improvement. This case highlights the importance of suspecting VZV reactivation in young patients with multifocal neurological deficits.

Palabras clave: Virus Varicela-Zoster; Accidente cerebrovascular; Neuroinfección

Varicela Zoster: un simulador infrecuente de evento cerebrovascular agudo en paciente joven, reporte de caso

Resumen

El virus Varicela zoster (VVZ) es un alfa-herpesvirus neurotrópico que, tras la primoinfección, permanece latente en ganglios nerviosos y puede reactivarse, afectando el sistema nervioso central, las manifestaciones incluyen meningitis, encefalitis, neuropatías craneales y eventos vasculares. Se presenta el caso de una mujer de 32 años, previamente sana, que consultó por cefalea intensa de inicio súbito, seguida de parálisis facial periférica derecha y hemiparesia ipsilateral. Al examen neurológico presentaba hipoacusia, paresia facial y del VI nervio craneal derechos, disminución de fuerza y sensibilidad en hemicuerpo derecho. La tomografía y angiotomografía fueron normales, no se identificaron lesiones isquémicas; la resonancia magnética cerebral mostró cambios inflamatorios en VI y VII pares craneales. El líquido cefalorraquídeo (LCR) evidenció pleocitosis linfocitaria e hiperproteinorraquia; el panel de meningoencefalitis fue positivo para VVZ. Se descartaron causas autoinmunes, cardiovasculares e infecciosas adicionales. Se inició aciclovir intravenoso 10 mg/kg cada 8 h por 14 días y prednisolona oral 60 mg/día por 10 días, logrando mejoría clínica significativa. Este caso destaca la importancia de sospechar reactivación de VVZ en pacientes jóvenes con déficit neurológico multifocal.

Keywords: Varicella-zoster virus; Stroke mimic; Neuroinfection

Introduction

The varicella-zoster virus (VZV) is an alpha herpesvirus of the Herpesviridae family, composed of double-stranded DNA that infects only humans[1]. Primary infection with the virus causes the disease known as chickenpox, which mainly manifests in children under 10 years of age and is more frequent in countries where the vaccine is not widely available[2]. Following the primary infection, this microorganism remains latent in the nervous tissue, especially in the peripheral autonomic ganglia, from where spontaneous reactivation, or in response to predisposing factors, can occur[3].

Viral reactivation, known as herpes zoster, leads to the spread of the microorganism, which can occur in the central nervous system, skin, or both. It most frequently manifests as a pain-

1 Internal Medicine Department, Internal Medicine Program, Universidad del Valle, Cali, Colombia. https://orcid.org/0009-0008-1070-4657
2 Internal Medicine Department, Infectious Diseases Program, Universidad del Valle, Cali, Colombia. https://orcid.org/0000-0002-0692-5077
3 Internal Medicine Department, Internal Medicine Program, Universidad del Valle, Cali, Colombia. Internal Medicine Department, Infectious Diseases Program, Universidad del Valle, Cali, Colombia. Infectious Diseases Department, Hospital Universitario del Valle "Evaristo García"; Professor, Universidad del Valle, Cali, Colombia. https://orcid.org/ 0000-0002-9281-2844
* Autor para correspondencia:
Correo electrónico: rodriguez.luisam04@gmail.com

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ful vesicular skin eruption with characteristic dermatomal distribution[1]. However, multiple neurological complications have been described, with zoster being a risk factor for stroke development[4].

We present the case of a young woman who consulted for an acute neurological deficit, in search of etiological causes, and was finally diagnosed with a neuroinfection by VZV, notably without the presence of skin lesions, showing improvement with the targeted treatment.

Case description

A 32-year-old woman with no relevant past medical history presented to our emergency department with a three-day history of sudden-onset severe frontoparietal headache, complicated with right-sided peripheral facial paralysis, and a ten-hour evolution of ipsilateral hemiparesis at admission with persistent headache.

The patient's general condition was good, vital signs were within normal limits, neurological assessment revealed no impairment of higher mental functions, right-sided peripheral facial paralysis (Figure 1), ipsilateral sixth cranial nerve palsy, right-sided hearing loss, right upper extremity strength 3/5 and right lower extremity 4/5 according to the MRC scale, decreased sensation in the right side of her body, gait disturbances, and normal myotendinous reflexes.

Computed tomography and CT angiography of the head and neck were normal, and no ischemic lesions were identified. Brain magnetic resonance imaging (MRI) with and without contrast, along with magnetic resonance angiography (MRA) of the brain and neck, revealed inflammatory changes in cranial nerves VI and VII, likely related to viral infection, without other relevant findings (Figure 2).

The complete blood count and renal function tests were normal, and diabetes mellitus and dyslipidemia were ruled out. The infectious disease profile (HIV, syphilis, hepatitis B and C, and HTLV 1/2) was negative (Table 1). Lumbar puncture was performed with an opening pressure of 23 cmH_2O. Cerebrospinal fluid (CSF) analysis showed lymphocytic pleocytosis and elevated protein levels. The meningoencephalitis panel (BioFire® FilmArray®) was positive for *Varicella-zoster virus* (VZV), and other CSF studies were negative (Table 2). Complementary studies for cerebral ischemic vascular events in a young patient (transesophageal echocardiogram, 24-hour Holter, and autoimmunity profile) were negative.

With the diagnosis established, an attempt was made to identify the presence of skin lesions in the patient; however, none were found. Intravenous acyclovir 10 mg/kg every 8 h was initiated; due to House-Brackmann grade III facial paralysis (moderate dysfunction) and the exclusion of ischemic stroke, oral prednisolone 60 mg/day was added. Upon

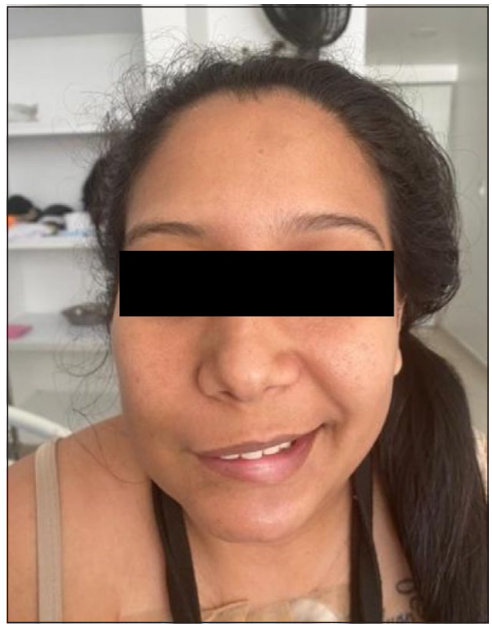

Figure 1. Clinical presentation: Right-sided peripheral facial paralysis.

further questioning, the patient reported a history of chickenpox in childhood, and the serological profile for VZV was positive for IgG and negative for IgM.

Treatment was completed with intravenous acyclovir for 14 days and oral prednisolone for 10 days, with marked motor function improvement (strength from 3/5 to 4+/5 on the affected side and House-Brackmann grade from III to II). The patient was discharged with a prescription for pregabalin for pain management, an outpatient rehabilitation plan, and immunodeficiency testing. No further follow-up information was available.

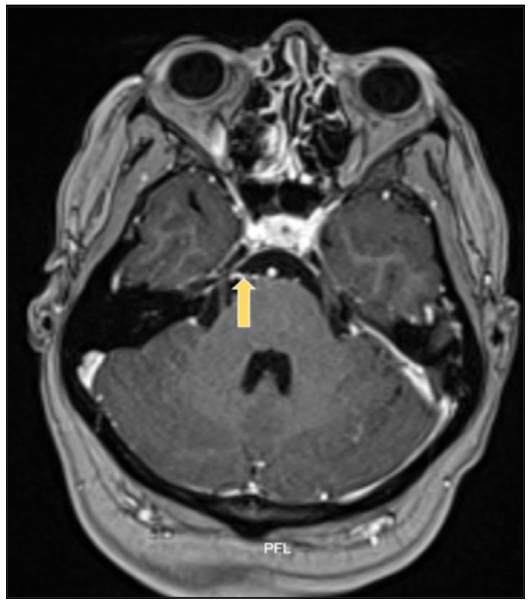

Figure 2. Brain MRI with contrast: T1+Gadolinium sequence: Enhancement along the path of the right cranial nerve VII (indicated by yellow arrow), no other associated findings.

Table 1. Laboratory tests

Laboratory or parameter	Result	Reference range
White blood cells	6.800 x 10^3/uL	3.980 - 10.04 x 10^3/uL
Neutrophils	4.300 x 10^3/uL	1.56 - 6.13 x 10^3/uL
Lymphocytes	1.880 x 10^3/uL	1.18 - 3.74 x 10^3/uL
Hemoglobin	12.9 g/dL	11.2 - 15.7 g/dL
Platelets	340.000 x 10^3/uL	182.000 - 369.000 x10^3/uL
Sodium	138 mmol/L	136 - 145 mmol/L
Potassium	4.1 mmol/L	3.5 - 5.1 mmol/L
Chloride	105 mmol/L	98 - 107 mmol/L
Serum creatinine	0.7 mg/dL	0.51 - 0.95 mg/dL
Blood urea nitrogen (BUN)	11 mg/dL	8 - 23 mg/dL
Glycemia	107 mg/dL	70 - 100 mg/dL
Glycated hemoglobin (HbA1c)	5.19 %	5.7 - 6.4 %
Total cholesterol	193 mg/dL	101 - 239 mg/dL
High-density lipoprotein cholesterol (HDL)	47 mg/dL	< 100 mg/dL
Low-density lipoprotein cholesterol (LDL)	121.7 mg/dL	> 50 mg/dL
Triglycerides	123 mg/dL	< 150 mg/dL
C-reactive protein (CRP)	<5 mg/L	0 - 5 mg/L
Thyroid-stimulating hormone (TSH)	2.2140 IU/mL	0.4-4.05 mUI/mL
Free thyroxine	1.03 ng/dL	0.78-2.19 ng/dL
HIV antibodies 1 and 2	Negative	
Hepatitis B surface antigen	Negative	
HTLV 1/2	Negative	
Non-treponemal test	Non-reactive	
Treponema pallidum (TP-PA)	Negative	
Varicella-zoster IgM antibodies	10.1 U/mL	<20 U/mL
Varicella-zoster IgG antibodies	>1500 mIU/mL	<50 mUI/mL
Folic acid	4.64 ng/ml	2.76 - 20 ng/mL
Vitamin B12	342 pg/ml	239 - 931 pg/mL
Complement C3	139 mg/dL	88 - 165 mg/dL
Complement C4	32.4 mg/dl	14 - 44 mg/dL
Anticardiolipin antibodies IgG	1.3 GPL-U/mL	<10 GPL-U/mL
Anticardiolipin antibodies IgM	0.8 U/ml	<10 MPL-U/mL
Anti-beta-2 glycoprotein I antibodies IgG	1.8 U/ml	<5 U/mL
Anti-beta-2 glycoprotein I antibodies IgM	0.9 U/ml	<5 U/mL
Antinuclear antibodies	Negative	Biological reference interval in healthy individuals: Reactive: 1:40 from 20 to 30 % Reactive: 1:80 from 10 to 15 % Reactive: 1:160 up to 5 % Reactive 1:320 up to 2 %.
Anti-extractable nuclear antigen antibodies	Negative	

Discussion

The Varicella-zoster virus (VZV) is an alphaherpesvirus of the Herpesviridae family that contains double-stranded DNA and infects only humans[1]. Infection caused by the virus is found worldwide, with epidemiological variations between temperate and tropical or subtropical regions. In temperate countries, such as the United States and the United Kingdom, up to 90% of the population seroconverts before the age of 15 years, with a higher incidence in winter and early spring. In tropical regions, infection is less frequent in children, leaving more adults susceptible to infection[2].

Primary VZV infection causes chickenpox, a highly contagious disease that mainly affects children under 10 years of age. Since the introduction of the vaccine, the incidence of this disease has decreased. The main characteristic of VZV is a pruritic vesicular rash that occurs mainly on the trunk and face, where the virus resides. It can infect other susceptible individuals through contact and aerosol transmission[3]. Following the primary infection, the virus remains inactive in the nerve tissue, with the possibility of reactivation in adulthood, which can lead to shingles. It is estimated that approximately one-third of people will develop it at some point in their lives[1].

Neurological involvement by VZV can occur through hematogenous dissemination or retrograde transaxonal transport from skin lesions to the sensory ganglia. The route of viral infection depends largely on the mechanism and location of exposure, whereas viral pathogenesis depends on environmental factors and the host's immunological susceptibility[4]. Therefore, the risk of central nervous system involvement after the viral latency period increases with age and in immunosuppressed conditions[1]. During reactivation, neuronal dissemination can affect any point along the neuroaxis, involving the brain parenchyma, spinal cord, subarachnoid space, blood vessels, and eyes[4]. Among the most frequently involved sites are the ophthalmic branch of the trigeminal nerve and the facial nerve via the geniculate ganglion[1].

Neurological manifestations are varied and include meningitis, encephalitis, cerebellitis, myelitis, radiculitis, and single or multiple cranial neuropathies. Cerebellitis, observed in 1 out of every 4,000 cases of *varicella-zoster*, is reported to present with ataxia, nystagmus, headache, and altered mental status. In Ramsay Hunt syndrome, reactivation of the virus in the geniculate ganglion produces an auricular or oral vesicular rash, peripheral facial palsy, and vestibular symptoms[5]. Another relevant manifestation is VZV-associated vasculopathy, which can cause ischemic or hemorrhagic cerebrovascular events, aneurysms, arterial dissection, and subarachnoid hemorrhage. The risk of these events increases with age and in immunosuppressed patients[6]. Diagnosis usually relies on characteristic clinical manifestations and a history of prior exposure but can be complemented by laboratory methods. Direct detection of the virus by culture is impractical because of its marked lability; therefore, techniques such as direct immunofluorescence, polymerase chain reaction (PCR), and antibody detection in cerebrospinal fluid (CSF) are preferred. In cases of vasculopathy, CSF PCR has a sensitivity of approximately 30%, which increases to 93% with the detection of IgG and IgM against VZV. Calculating the intrathecal/serum antibody ratio ([CSF VZV IgG ÷ serum VZV IgG] / [CSF total IgG or albumin ÷ serum total IgG or albumin]) can help differentiate local production from serum contamination, with a cutoff point of > 1.5[4].

In VZV infection, involvement of multiple cranial nerves has been described, with lower cranial nerves being less frequent, except for cranial nerve VIII[7]. Multi-nerve paralysis can be caused by mechanisms such as meningitis or direct spread of the virus affecting the anatomically related nerves[8]. Another known mechanism is vasculopathy, which involves an inflammatory infiltrate with T cells and macrophages, primarily affecting the adventitia and intima, with its main manifestation being ischemic or hemorrhagic stroke, although it can also include aneurysms, thrombotic events, and arterial dissection[3]. However, it has been postulated that vasculopathy may be one of the mechanisms that can also explain cranial neuropathies[4].

Table 2. Cerebrospinal fluid studies

Laboratory or parameter	Result	Reference range
	Opening pressure 23 cm H2O	Opening pressure: 5 - 15 cm H2O
Cytochemical analysis	Aspect: transparent, colorless. Glucose: 44 mg/dL (serum glucose 98 mg/dL). Proteins: 83 mg/dL. Red blood cells: 15/ mm³. Polymorphonuclear cells: 14/mm³. Lymphocytes: 495/ mm³	Glucose: 45 - 70 mg/dL Proteins: 15 - 5 mg/dL Red blood cells: 0 /mm3 Polymorphonuclear cells: 0 / mm3 Lymphocytes: 0 /mm3
Gram stain	No leukocytes or bacteria observed	
Bacterial culture	Negative	
Treponemal test	Negative	
VDRL	Non-reactive	
India ink stain	Negative	
Acid-fast bacilli smear	Negative	
Mycobacterium tuberculosis culture	Negative	
Molecular panel for meningitis*	Varicella-zoster virus detected	

* Panel includes: Citomegalovirus, enterovirus, Herpes simplex 1 y 2, Varicela Zoster virus, *Escherichia coli*, *Haemophilus influenzae*, *Listeria monocytogenes*, *Neisseria meningitidis*, *Streptococcus agalactiae*, *Streptococcus pneumoniae*, *Cryptococcus neoformans/Gatti*.

In this case, the patient's MRI showed inflammatory changes in the sixth and seventh cranial nerves. The inflammatory process disrupts the blood-brain barrier and causes diffusion of contrast material into the endoneurial space, resulting in enhancement of the affected nerves[7]. Studies have demonstrated a positive correlation between facial nerve inflammation and increased signal intensity on contrast-enhanced MRI, primarily affecting the meatal and labyrinthine segments, and the inflammatory process usually resolves with the onset of clinical recovery[9].

The patient's treatment consisted of intravenous acyclovir for 14 days and oral prednisolone for 10 days, as recommended in the literature, showing better results than monotherapy with antiviral[3], resulting in significant clinical improvement (strength from 3/5 to 4+/5 on the affected side of the body and House-Brackmann grade from III to II). However, the main limitation is the lack of follow-up on the patient's progress; the final clinical improvement or possible recurrences are unknown.

The high seroprevalence of anti-VZV IgG antibodies (up to 99% in adults, as reported in population-based studies) reinforces the importance of considering this etiology, even in immunocompetent patients, when faced with a compatible acute neurological syndrome. Including VZV in the differential diagnosis of neurological syndromes with suggestive findings on neuroimaging and CSF is key to timely treatment, which can improve functional outcomes of patients[10].

In conclusion, VZV reactivation should be considered in the differential diagnosis of acute multifocal neurological deficits, even in immunocompetent young adults. Confirmation using molecular techniques and early initiation of intravenous acyclovir, combined with corticosteroids in selected cases, can improve the functional prognosis. This case underscores the need for a comprehensive and timely approach to prevent permanent neurological sequelae in such patients.

Ethical considerations

Protection of persons and animals. The authors declare that no experiments were conducted on humans or animals for this manuscript.

Protection of vulnerable populations. Not applicable.
Confidentiality. The authors declare that no data that could identify patients appears in this document. The patient was informed about this case report and their informed consent was obtained, respecting their confidentiality.

Privacy. The authors guarantee the protection of the identity of the patient included in the case report. No names, initials, or medical record numbers that could identify participants were used.

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Conflict of interest. The authors declare no conflict of interest.

Statement on the use of artificial intelligence. The authors state that no artificial intelligence tools were used in the preparation of this case report.

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