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Case Report

Unexpected Stroke in a Child Post-Chickenpox: Unveiling Critical Medical Imaging Insights

El Mabrouk Fatma*, Lahlou Ihssane, Hlioui Kamal, Nazik Allali, Latifa Chat and Siham El Haddad

Department of Radiology, Ibn Sina University Hospital Center, Rabat, Rabat-Sale-Kenitra, Morocco

***Corresponding Author:** El Mabrouk Fatma, Department of Radiology, Ibn Sina University Hospital Center, Rabat, Rabat-Sale-Kenitra, Morocco.

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Abstract

This study reports the case of an 11-year-old child who developed acute ischemic stroke (AIS) following varicella infection. The patient had no significant medical history and initially recovered well from the varicella episode. However, four months later, he presented with sudden right upper limb monoplegia, vomiting, and headaches. Neurological examination revealed motor and sensory deficits in the right upper limb and weakness (3/5) in the left upper limb. Imaging studies, including brain CT and MRI, confirmed a hypodense pontine lesion consistent with AIS. Laboratory tests indicated an inflammatory response, and viral serology showed the presence of VZV DNA in the cerebrospinal fluid. Treatment with antivirals and corticosteroids led to significant improvement over three months, with complete recovery after seven months. This case underscores the potential for severe neurological et vascular complications, including AIS, following varicella infection in immunocompetent children. Understanding the pathophysiology and early recognition of such complications are crucial for timely intervention and optimal outcomes.

Keywords: *Acute Ischemic Stroke (AIS); Varicella-Zoster Virus (VZV) Complications; Pediatric Stroke*

Introduction

Infection with the varicella-zoster virus (VZV) is a prevalent condition, particularly among unvaccinated pediatric populations. Although varicella is typically regarded as a benign pediatric disease, severe complications can arise, including neurological and vascular pathologies leading to ischemic stroke. In this article, we present a case of an 11-year-old immunocompetent child experiencing acute post-varicella IS, underscoring the clinical and radiological implications of this rare yet significant and debilitating complication.

Case Report

This case concerns an 11-year-old child with no notable personal or family medical history, born from a non-consanguineous marriage, the eldest of two siblings, who demonstrates normal psychomotor development and growth. The patient experienced a varicella episode four months ago and received symptomatic treatment consisting of rest, hydration, hygiene of scratching lesions, and paracetamol for fever, with good improvement.

The patient presented to the emergency department with a sudden onset of right upper limb monoparesis, vomiting, and headaches. The medical history did not reveal any trauma or similar previous episodes.

Clinical examination found a conscious patient, well-oriented in time and space, with a fever of 39°C. All other vital constants were normal. There was a loss of motor and sensory function in the right upper limb, a 3/5 muscle weakness in the contralateral upper limb, no meningeal syndrome, no dysarthria, and no visual disturbances. Additionally, there were no skin lesions like chickenpox or other.

A biological workup, including a complete blood count (CBC) and C-reactive protein (CRP) test, was initiated, and a brain CT scan was requested to look for signs indicative of intracranial hypertension (ICH) or meningitis.

The brain CT scan was performed with 64-slice helical cuts, both with and without contrast injection, revealing a well-defined, round hypodense pontine lesion that did not enhance after contrast injection and showed no mass effect or perilesional edema. No pathological contrast enhancement was observed, and the vascular structures were well opacified (Figure 1).

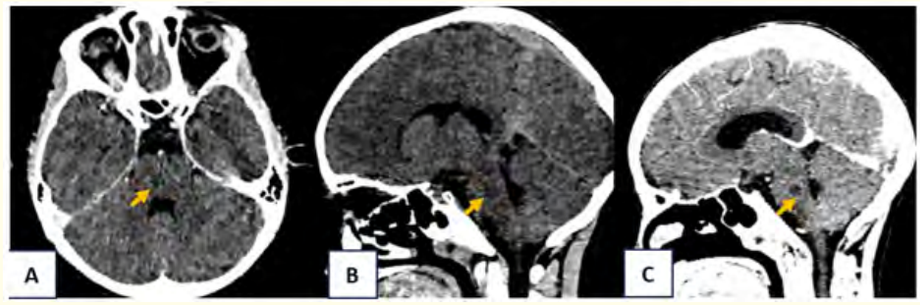

Figure 1: *Axial slice without contrast injection of a brain CT scan (A) and sagittal slice (B) showing a hypodense, rounded pontine lesion that is fairly well defined and better delineated after contrast injection as seen in the sagittal slice with contrast (C), which does not enhance.*

The brain CT scan also ruled out signs indicative of intracranial hypertension (ICH) as well as any other contraindications to a lumbar puncture, such as a potential infection.

As for the laboratory tests, the complete blood count (CBC) showed lymphocytosis and a biological inflammatory syndrome, and the lumbar puncture findings were consistent with the CBC, revealing lymphocytic-predominant pleocytosis. Consequently, a viral serology study was initiated.

In front of the atypical presentation and for better characterization of the lesion, a brain MRI was performed, revealing a poorly defined right pontine lesion with hypointensity on T1, hyperintensity on T2 and FLAIR, diffusion hyperintensity with low ADC (Figure 2) and a supplemental injection was performed, revealing a lack of opacification in the basilar artery and an interruption of flow on the arterial TOF sequences, confirming the vascular origin of the pontine lesion (Figure 3).

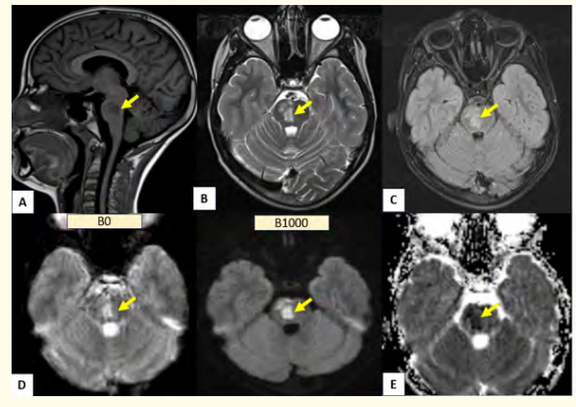

Figure 2: *A right unilateral pontine lesion is observed, appearing as a hypointense signal on T1-weighted imaging (A) and as a hyperintense signal on axial T2-weighted and FLAIR sequences (B and C), well demarcated. It presents as a hyperintense signal on diffusion-weighted imaging (D) with a low ADC coefficient (E).*

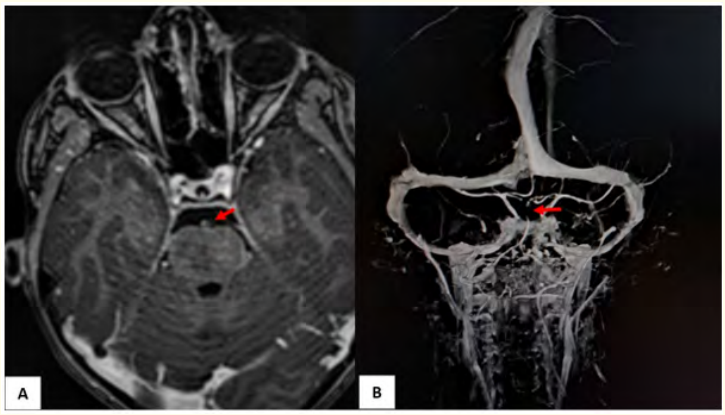

Figure 3: *On the axial T1 post-contrast sequence (A), the red arrow indicates a lack of opacification of the basilar artery, which is better visualized on the arterial/venous TOF sequence, highlighting the interruption of flow.*

All findings suggest a post-varicella ischemic stroke (AVCI), confirmed with serologies showing the presence of VZV DNA in the cerebrospinal fluid (CSF), representing the case of an 11-year-old immunocompetent child with a post-varicella AIS.

The treatment included intravenous antivirals and corticosteroid boluses, resulting in significant improvement after 3 months and full recovery after 7 months.

Discussion

Chickenpox is a widespread infectious disease caused by the varicella-zoster virus (VZV).

In the absence of vaccination, this infection typically occurs during childhood. However, cases of chickenpox are observed even in individuals with up-to-date vaccination records due to its highly contagious nature. The incubation period is 14 to 16 days post-exposure, and transmission occurs via indirect airborne route (through droplets in the air) and direct contact with skin lesions [1].

According to the World Health Organization, chickenpox affects approximately 140 million people worldwide each year, resulting in 4.2 million severe cases requiring hospitalization and causing 4,200 deaths [2].

The varicella-zoster virus (VZV) is a neurotropic alpha herpesvirus that exclusively infects humans. Primary infection causes chickenpox, after which the virus remains latent in human body, residing in ganglionic neurons throughout the nervous system. Occasionally, cell-mediated immunity against VZV diminishes, leading to virus reactivation and the development of shingles, characterized by skin lesions [3].

Other manifestations are also described: the VZV infection can manifest through various cardiovascular, muscular, hematological, urogenital, and hepatic complications. Respiratory and neurological complications are the most frequently reported, according to meta-analysis and systematic study conducted by Hiral Anil Shah, Anne Meiwald in January 2024, with 70 and 69 studies respectively [4].

Besides, among the neurological complications, common findings include encephalitis and cerebellar ataxia, Guillain-Barré syndrome, facial paralysis, transverse myelitis, aseptic meningitis, cerebral vasculitis, optic neuritis, meningoencephalitis, ventriculitis, delayed contralateral hemiparesis, and peripheral motor neuropathy [5].

Vasculopathy and varicella-zoster virus induced stroke syndrome have been recognized in medical literature since the early 1970s [6]. Recent research has highlighted that individuals affected by herpes zoster (shingles) may face an elevated risk of both ischemic and hemorrhagic strokes. This association underscores the virus's potential to trigger vascular complications, possibly through mechanisms involving inflammation and vascular damage.

However, the findings across studies have not been entirely consistent. While some studies support a heightened stroke risk following shingles, others have not found significant long-term associations. There is also ongoing debate regarding the duration of heightened stroke risk post-zoster infection. Some researchers suggest that the risk may be acute or short-term, while others explore the possibility of prolonged vascular implications over a more extended period.

Understanding these dynamics is crucial for developing targeted prevention strategies and optimizing patient care, particularly for individuals recovering from shingles who may require vigilant monitoring for potential vascular events. Further research is needed to clarify the exact mechanisms and long-term implications of varicella-zoster virus-related vascular complications, aiding in more precise risk assessment and management strategies for affected patients [7].

The first meta-analysis on the risk of stroke following herpes zoster infection was indeed conducted by Hiral Anil Shah and Anne Meiwald in January 2024. By synthesizing data from 9 observational studies, they found that the increased risk of stroke is highest shortly after the acute episode of shingles, gradually decreasing over time but remaining significant beyond the first year. This risk is particularly pronounced among patients with ophthalmic zoster [4].

It appears that our patient developed AIS 4 months after the acute phase of chickenpox without ocular involvement.

The occurrence of acute ischemic stroke in this case is inflammation triggered by systemic infection, which can increase blood clotting susceptibility. Due to the presence and replication of VZV within cerebral arteries, the release of cytokines such as TNF-alpha and interleukin-2 during inflammation or stress can lead to endothelial dysfunction, plaque rupture, and hypercoagulability, thereby accelerating vasculopathy of small and large vessels [8].

Upon reviewing both acute phase and recent assessments, our patient showed no clinical or biological signs of coagulation disorders. Given their young age, atherosclerosis is highly unlikely.

The most commonly reported vasculopathy following VZV infection is giant cell arteritis of the temporal artery, which occurs in 80% of cases with associated ocular involvement. This condition primarily affects adults, particularly those with diabetes [9].

While our patient did not exhibit any clinical or radiological signs indicative of ocular involvement or optic neuropathy, brain MRI remains an indisputable diagnostic tool for detecting such complications.

In their 2017 article, Maria A. Nagel and Don Gilden [10] reported that the average interval between shingles and the onset of focal neurological deficits is 4 months, exactly matching the timeline of our patient's presentation. They emphasized that abnormal brain MRI findings are present in 97% of cases, often revealing lesions at the gray-white matter junction. Angiography typically shows involvement of both small and large arteries in nearly 70% of cases. However, in our case, the stroke occurred in the right pons, and the low ADC value confirms the acute nature of the lesion. Arteriography revealed stenosis in the cerebral artery.

Nagel and Gilden [10] also noted that the definitive diagnosis of VZV vasculopathy is established by detecting anti-VZV IgG antibodies in the cerebrospinal fluid (CSF), which are more frequently positive than amplifiable VZV DNA in the CSF. None of these details contradict the findings observed in our case.

Because VZV vasculopathy results from an active viral infection, it is usually recommended to launch intravenous acyclovir. The standard dosage is typically 10 - 15 mg/kg administered every 8 hours for 7 - 10 days. The effectiveness of corticosteroids in addition to antiviral treatment is uncertain due to limited research.

However, a short course of oral prednisone is recommended at 1 mg/kg daily for 5 - 7 days is often given due to the specific type of artery inflammation seen in VZV vasculopathy within the brain [11].

Conclusion

In summary, our case illustrates the potential for varicella-zoster virus (VZV) infection to lead to acute ischemic stroke (AIS) despite the absence of typical ocular manifestations. Diagnostic tools such as brain MRI and angiography were instrumental in confirming the acute nature of the stroke and identifying cerebral artery involvement. The presence of anti-VZV IgG antibodies in cerebrospinal fluid further supports the diagnosis of VZV-related vasculopathy. These findings underscore the importance of comprehensive evaluation in managing VZV-associated neurological complications, guiding tailored treatment strategies for better patient outcomes.

Informed Consent

Written informed consent was obtained from the parents of patients for the publication of this case report.

Ethical Approval

Our institution does not require ethical approval for reporting individual cases or case series.

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I have no funding.

Conflict of Interest

This work has no conflict of interest.

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