

positive results. The results of a chest, abdomen, and pelvis CT scan and a serum tumour marker test ruled out a paraneoplastic origin. The patient was diagnosed with non-paraneoplastic anti-NMDA receptor encephalitis and treated with corticosteroids, immunoglobulins, and rituximab. Clinical progression was unstable; language impairment and neuropsychiatric manifestations persisted, motor symptoms (myoclonus and rigidity, with no clear epileptogenic focus) worsened, and the patient developed symptoms of dysautonomia. Neurological symptoms improved progressively with cyclophosphamide. A follow-up brain SPECT scan performed 5 months after the initial symptoms showed near-complete resolution of the alterations (Fig. 1B and D). Other successfully treated patients with this type of encephalitis have also shown disappearance or improvement of abnormal findings in brain perfusion SPECT^{3,4} or ¹⁸F-FDG-PET.⁷ Brain perfusion SPECT played a crucial role in the diagnosis of this type of encephalitis given the atypical initial symptoms; language impairment of subacute onset suggested other diagnoses.

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Varicella-zoster virus meningitis in an immunocompetent paediatric patient^{☆☆}



Meningitis causada por el virus varicela-zóster en un niño inmunocompetente

Dear Editor:

Herpes zoster is the localised manifestation of varicella-zoster virus (VZV) infection. It occurs as a result of reactivation of the virus, which remains latent in sensory ganglia of cranial and spinal nerves once the infection resolves. Neurological complications of herpes zoster are

relatively infrequent, especially in immunocompetent children.

We present the case of a 7-year-old boy who was admitted due to headache, vomiting, photophobia, and somnolence. The day before admission, the child's parents noticed that the patient had an erythematous exanthem on the left scapula, which was initially micropapular and subsequently vesicular; the exanthem was not pruriginous or painful. Two weeks previously, the patient had experienced upper respiratory tract infection. He had no recent history of foreign travel, contact with sick people, or insect bites. The patient had been vaccinated according to the Portuguese vaccination calendar; he had not received the varicella vaccine but had developed the condition at the age of 2 years. His parents were non-consanguineous.

During examination, the child was afebrile, haemodynamically stable, and drowsy (but easily awakened); he displayed no meningeal or focal neurological signs. Eye fundus examination revealed no abnormalities. The left scapular region showed a vesicular exanthem on an erythematous base, suggestive of herpes zoster. A complete blood count yielded 6000 leukocytes/mm³, 90.2% neutrophils, and normal C-reactive protein levels. A CSF analysis revealed 480 leukocytes/mm³ (96.7% mononuclear cells), a protein level of 90 mg/dL, and low glucose levels (41 mg/dL in the

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CSF and 85 mg/dL in the serum). Results from Gram staining and a CSF culture were negative for pathogenic bacteria. A polymerase chain reaction (PCR) study detected VZV in the CSF. No alterations were detected in cell-mediated and humoral immunity. The patient received IV aciclovir (10 mg/kg every 6 hours) for 14 days, progressing favourably. After 4 years of follow-up, our patient remains asymptomatic.

Two factors may increase the risk of herpes zoster in paediatric patients: immunosuppression^{1,2} and varicella in early childhood.^{3–5} Although the underlying mechanisms are yet to be understood, the inability of children below the age of 2 years to develop a specific cell-mediated and humoral immune response to VZV may explain why healthy paediatric patients with a primary VZV infection occurring in early childhood may develop herpes zoster and aseptic meningitis.^{6,7} Herpes zoster lesions in children are less frequently associated with localised pain, paraesthesia, pruritis, or fever.^{8,9} VZV is a neurotropic human herpesvirus⁸ that may cause a wide range of neurological disorders if it reactivates.^{1–4} Neurological involvement is usually accompanied by the exanthem that characterises herpes zoster,^{6,10} except in immunosuppressed patients.^{1,3} However, the literature also includes cases of meningitis secondary to VZV infection and no exanthem in immunocompetent patients,^{3,5,11,12} since the reactivated virus can travel directly to the central nervous system without travelling to the epidermis.^{2,5} CSF profiles of patients with meningitis secondary to VZV infection are clinically undistinguishable from those of patients with other types of viral meningitis.^{3,5,6,13} Low CSF glucose levels have been observed in immunocompetent children and adults with meningitis due to VZV infection.^{3,12} PCR analysis of the CSF for VZV DNA is recommended in cases of unexplained aseptic meningitis, especially in children who develop varicella in early childhood.⁴ PCR analysis, especially in the first week after onset of acute symptoms of meningitis (with or without exanthem), is essential for diagnosis and treatment. Negative PCR results do not rule out meningitis due to VZV infection, however.¹² In these cases, anti-VZV IgM antibodies may be detected in the CSF. Presence of IgM antibodies in the CSF may point to CNS diseases since these antibodies do not easily cross the blood-brain barrier.¹ During the second week after disease onset, viral DNA disappears and anti-VZV IgG antibodies can be found in the CSF.¹²

IV aciclovir dosed at 10–15 mg/kg every 8 hours for 10–14 days is the treatment of choice for encephalitis due to VZV infection.¹ Most paediatric patients recover fully from viral CNS infections.⁸ VZV should be considered as a possible cause of meningitis in children with vesicular exanthem and a history of varicella in early childhood.

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