

**Short Report**

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**Afebrile intracranial hypertension revealing varicella meningitis***S Mrhar<sup>1</sup>; Nozha Radi<sup>1\*</sup>; K Elfakiri<sup>1</sup>; N Rada<sup>1</sup>; G Draiss<sup>1</sup>; N Sora<sup>2</sup>; M Bouskraoui<sup>1</sup>*<sup>1</sup>Pediatrics Department A, Mother and Child Hospital Center, Hospitalier Mohammed VI, Marrakech, Morocco.<sup>2</sup>Microbiology Laboratory, Mohammed VI Hospital Center, Marrakech, Morocco.**\*Corresponding Author: Nozha Radi**Pediatrics Department A, Mother and Child Hospital  
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**Introduction**

The varicella and shingles virus VZV is a strictly human virus which belongs to the family of herpesviridae which is transmitted by air or in contact with skin lesions rich in virus, it is a virus with dermo-neurological tropism whose primary infection corresponds to chicken pox and reactivation after stagnation in the sensory ganglia, caused by shingles during a drop in immunity. VZV can cause various CNS central nervous system infections including meningitis and encephalitis.

We report the case of an immunocompetent child who presents with varicella meningitis by reactivation without dermatological signs or meningeal syndrome.

**Observation**

This is a 14-year-old boy with a history of cutaneous chickenpox at the age of 4, well vaccinated according to the Moroccan national vaccination program, admitted for an apyretic intracranial hypertension syndrome made of helmet headaches. intensely resistant to analgesics with vomiting of food in a jet

and photophobia, evolving for 6 days before admission, without other associated signs, in particular no convulsion or disturbance of consciousness. On clinical examination, the patient was conscious, hemodynamically and respiratory stable, afebrile at 36.7°C; no meningeal stiffness no signs of kernig or brudzinski. Brain scan was normal, fundus was normal. The intracerebral pressure measurement was normal at 17.5 mmHg.

The biological assessment showed a lymphopenia at 990/mm with a hemoglobin of 12.9 g/dl, white blood cells at 10,290/mm<sup>3</sup> and platelets at 324,000/mm<sup>3</sup>, as well as a CRP at 1.90 mg/l. the HIV serology was negative and the primary immunodeficiency assessment was normal.

The treatment was based on intravenous acyclovir at a dose of 20 mg/kg/8H for 10 days with oral corticosteroid therapy based on prednisone 1 mg/kg/day for 5 days orally and analgesic based on paracetamol 15 mg/kg/6h. The evolution was good.

## Discussion

Chickenpox is the first clinical manifestation of VZV infection. Among the 8 human herpesviruses, VZV is the only one to be transmitted by aerosol, the respiratory tract constituting the portal of entry, it replicates locally in the airways and lymph nodes before spreading to various organs during a second viremia. This incubation period, which lasts about 14 to 16 days, is followed by a generalized skin rash which is in most cases quickly controlled by the immune system [1]. A recent epidemiological study carried out in France confirms that nearly 90% of 8-year-old children have encountered VZV and are naturally immune [2].

The virus has neurotropism like herpes simplex virus (HSV), and it can also lay dormant in sensory ganglia. Reactivation of the virus results in clinical manifestations of herpes zoster and sometimes disseminated disease in immunocompromised children [3].

Three different CNS presentations are most common: meningitis, encephalitis and Acute Disseminated Encephalomyelitis (ADEM) [4]. Several cases of fatal neurological disease caused by reactivation of VZV without rash in immunocompromised children and adults have been reported. Older immunocompetent patients may also present with VZV reactivation, including neurological disease, without a rash [5]. Varicella zoster meningitis is rare. Sometimes the CNS manifestation may appear before the rash in the case of chickenpox and herpes zoster [6]. In meningitis the demonstration of the presence of DNA or viral antigens/particles in the CSF or on the increase in antibody titer between paired serum samples. In 1992, Shoji et al [7] used PCR on CSF samples from a group of meningitis patients, some without rash, and noted that VZV was the causative agent. Echevarria et al [8] studied eight patients with aseptic meningitis, half of whom had no rash. They determined VZV-specific antibodies in serum and CSF. In cases of meningitis due to VZV, possible treatment with intravenous acyclovir for 7 to 10 days has been recommended for immunocompetent children [9].

## Conclusion

What is unique in our patient is the occurrence of varicella meningitis caused by reactivation of VZV without skin lesions and without febrile meningeal syndrome in an immunocompetent child. This is a rare event, although cases are likely to be overlooked because VZV is not routinely suspected on CSF analysis in aseptic meningitis and PCR is not systematically practiced.

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