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Case Report

Trigerminal Herpes Zoster with Encephalitis in a HIV Seronegative Child: A Case Report

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Abstract

Background: Herpes zoster (HZ) - the reactivation of latent varicella virus infection, is usually seen in the immunocompromised. Complicated Trigeminal nerve involvement is rare in childhood.

Aim: To report a case of Trigeminal herpes zoster with encephalitis in a 19 month old HIV seronegative boy.

Case Summary: AP presented in August 2019 with a 2 week history of painful, blistering and ulcerating lesions on the right side of the face, 12 days of fever and 8 days of convulsions. His mother had varicella zoster (VZ) at 8 months of gestation and did not receive antiviral therapy nor VZV Immunoglobulin. No history of vesicular rashes in the neonatal period or previous VZ infection. On examination, he had multiple weeping vesicles, ulcerations, scabs and crusts all localized to the V1, V2 trigeminal dermatomes on the right half of the face. He was conscious, alert with normal tone and reflexes. There were no visual or auditory deficits. He was seronegative to HIV. Full blood count showed leukocytosis, CSF analysis was normal. He received meningitis doses of IV acyclovir, IV ceftriaxone, topical lidocaine, oral paracetamol with remarkable clinical improvement.

Conclusion: HZ is an unusual presentation in immunocompetent children but may occur following in-utero exposure from maternal VZV infection. Advocacy for antenatal care, vaccination/immunization against VZV, use of Post exposure VZV Immunoglobulin in pregnant women and neonates is key.

Keywords: *Morel-Lavallee Lesion; Bleomycin; Sclerotherapy; Axilla; Child*

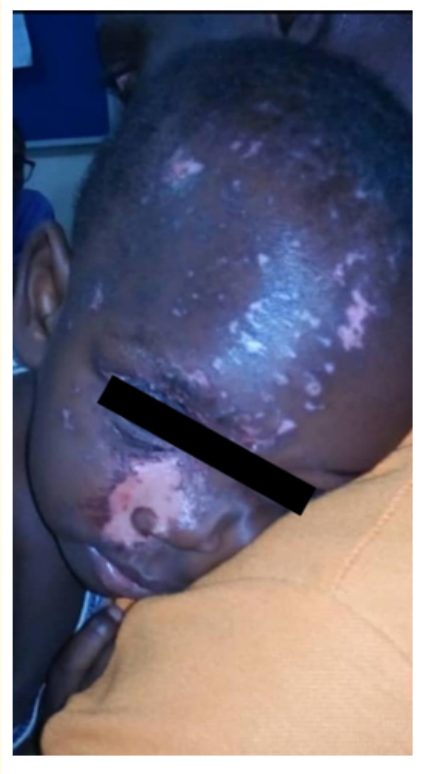
Introduction

Trigerminal herpes zoster (shingles) is an acute viral disease affecting the trigeminal nerve. It occurs following reactivation of the varicella zoster virus (VZV) that remained dormant in the trigeminal nerve root [2] ganglion following exposure to VZV. Herpes zoster may affect any sensory ganglia and its cutaneous nerve: the most common sites affected are thoracic dermatomes (56%), followed by cranial nerves (13%) and lumbar (13%), cervical (11%) and sacral nerves (4%) [1]. Among cranial nerves, the trigeminal and facial nerves are the most affected due to reactivation of VZV latent in gasserian and geniculated ganglia [1]. The 1st division of the trigeminal nerve is commonly affected, whereas the 2nd and the 3rd are rarely involved [1]. It is a disease of the immunocompromised and elderly, its occurrence in young children may be a sign of immunosuppression [2] or in utero exposure to VZV from maternal infection in pregnancy. Trigeminal

herpes zoster is a potentially devastating clinical occurrence, and is associated with severe long-term neurological sequelae, including encephalitis, vision loss and postherpetic neuralgia [3].

Case Summary

AP is a 19 month old male who presented to CHEW with a 2 week history of Right sided facial rashes, fever of 12 days and 8 day history of convulsions. His mother had generalized papular and vesicular rashes with fever for 1 week, at 8 months of gestation. She did not receive antiviral therapy nor VZV Immune globulin. She was seronegative to HIV. At presentation, he was underweight (74% of expected), he had multiple facial scabs, hypo pigmented and erythematous macules and excoriations at the frontal, peri orbital, temporal, maxillary and nasolabial aspect of the face. Lesions were pruritic, tender and all localized to the right half of the face, beginning from the midline. He was conscious and alert with normal tone and reflexes. There was no visual or auditory deficits, no obvious craniopathies. He was seronegative to HIV I and II. He had leukocytosis with abnormal lymphoblast and monocytosis. Cerebrospinal fluid biochemistry, microscopy and culture were essentially normal. He received IV Acyclovir 20 mg/kg, IV Ceftriaxone 100 mg/kg, Topical Lidocaine, Oral Paracetamol.



Discussion

Herpes zoster is a viral infection which signals background suppression in cellular immunity which leads to reactivation of dormant VZV in nerve roots, resulting in cutaneous manifestations. The leading cause of immunosuppression in sub-Saharan Africa remains the HIV/AIDS Pandemic [5], however our patient was seronegative to HIV. Other possible explanations for reduced immunity in our patient

include age less than 18 months and malnutrition. Clinical manifestations of VZV in children without antecedent chicken pox exanthems signals in utero exposure [6]. This cannot be ruled out in our patient, whose mother had suspected chicken pox at 8 months of gestation but did not receive antivirals nor varicella immunoglobulin VZIG. VZIG or IVIG prophylaxis in pregnancy is advocated within 72 to 96 hours of exposure and reduces the risk of neonatal and early childhood complications of VZV infection [6]. Central and peripheral nervous system complications, including encephalitis, vision loss and postherpetic neuralgia may be severe and reduce quality of life. The management of the condition depends upon the initiation of antiviral therapy at an early stage within the first 48 - 72 hours of the beginning of lesions [7]. CNS manifestations may have been averted if therapy was commenced promptly. First line antiviral medication is acyclovir and this was administered at 20 mg/kg in our patient due to CNS involvement. Other antivirals used in the treatment of this condition include Famciclovir [7], Valacyclovir and Foscarnet. Foscarnet is recommended for immunocompromised patients with acyclovir-resistant varicella zoster virus [4].

Conclusion

Trigeminal herpes zoster with encephalitis is an unusual encounter in immunocompetent children but can occur following in -utero exposure from maternal VZV infection. Focused antenatal care, advocacy for vaccination/immunization against VZV, availability and use of Post exposure VZIG in pregnant women and neonates are needed to prevent morbidity and mortality.

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