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Case Report
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Acute Vestibular Neuritis in an 8-Year-Old Child with Chicken Pox

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Abstract

Vestibular neuritis secondary to an active *varicella zoster* virus infection in children is uncommon and need to be considered. We report a case of an 8-year-old Malay boy with an acute onset of dizziness, vomiting and vertigo, at day 5 of primary varicella-zoster virus infection. The child had normal hearing and no other cerebellar signs. Blood and MRI investigations were within normal range. The patient showed good progression in the ward with spontaneous full resolution of symptoms three weeks after discharge. Clinical diagnosis of vestibular neuritis was made based on symptoms and its clinical progression.

Keywords: *Varicella zoster*; Chicken pox; Vestibular neuritis; Vertigo

Introduction

Varicella zoster virus infection or chicken pox is a common infection seen in children. It is often diagnosed clinically. Available literature reports the incidence of varicella-related hospitalizations in children worldwide to be from 0.9 to 29.4/100,000 [1]. While neurological complication is rare, it is a common reason for hospitalization and includes cerebellar ataxia, meningoencephalitis, convulsion and optic neuritis [1]. Children infected with varicella rarely needs admission and admissions are inherently due to complications. This case highlights a case presented with neurological complications related to *varicella zoster* infection that warrant admission for further investigations and observation.

Case Presentation

An 8-year old Malay boy presented with acute onset of dizziness, vertigo and vomiting for one day. There is no associated photophobia or confusion. There was no history of ear pain, ear discharge, hearing problem or tinnitus. He was having on going primary *varicella zoster* infection, which was clinically diagnosed five days earlier by a primary care doctor when he presented with typical appearance of vesicular rash and fever. Two of his siblings were also recently diagnosed with chicken pox. The patient was born preterm at 28-week gestation following a caesarean section for a triplet delivery but otherwise has no other significant past medical problems.

General examination showed an alert boy with normal speech. There was no neck stiffness. His weight was 46kg (95^{th} centile), height was 137cm (50^{th} centile) and his BMI was 24.5kg/m^2 (>95^{th} centile). His temperature was 36.6^{0}C, his blood pressure was 123/60 mmHg (50^{th} centile), pulse rate was 80 beats per minute, respiratory rate was 24 breaths per minute and SpO_2 was 100% on room air. There were multiple vesicles, some with crusted top, distributed over his face, scalp, trunk and limbs. His tonsils were mildly injected. Both his tympanic membranes were normal. No vesicular rashes were present within the ear canals.

Visual acuity was not performed as patient was not able to keep his eyes opened. There was painless, spontaneous left horizontal abduction gaze nystagmus. No vertical or pendulous nystagmus were noted. Headshake illicit a horizontal gaze nystagmus beating to the left and Dix hall-pike test showed a left horizontal gaze nystagmus. No abnormality was noted on right eye examination. Other cerebellar signs were negative. Tandem gait was not performed as the patient refused to walk due to dizziness. Muscle tone, deep tendon reflexes and plantar responses were normal.

Patient was promptly admitted. On arrival to hospital, immediate supportive and symptomatic treatment with intravenous fluids and prochlorperazine were given with good symptoms resolution. In-hospital investigations revealed a haemoglobin of 13.5 (normal range: 11.1–14.1 g/dL), white cell count of 15.5 (normal range: 5–11x10^9/L) and platelet of 265 (normal range: 110–450x10^9/L).

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Renal function test, liver function test, magnesium, phosphate and calcium were within normal range. MRI and MRA of the brain were done and showed no evidence of intracranial abnormalities to suggest infarction, aneurysm or hemorrhage. There was no hyperintense signal of the cerebellar gray matter in T2-weighted sequences to suggest presence of acute cerebellitis. Lumbar puncture to look for early meningoencephalitis was not performed as clinical symptoms improved following the symptomatic treatment.

This child was alert and afebrile throughout admission and showed good progression in the ward. He was discharged after 10 days of admission. Upon discharge, he was able to walk without any gait abnormalities, his vomiting and dizziness had abated but lateral gaze nystagmus persisted. Three weeks later, upon follow up, he had full spontaneous recovery.

Discussion and Conclusion

In children, the prevalence of vertigo and dizziness due to vestibular disorders is less frequent compared to adult and was reported to be between 0.5 and 5.3% [2]. Vestibular neuritis is an inflammation of the vestibular branch of the vestibulocochlear nerve. Labyrinthitis on the other hand, occurs when both branches of the vestibulocochlear nerve is affected and is associated with hearing loss. Prevalence of vestibular neuritis within the age group of 7 to 12 years old has not been reported [2].

A clinical diagnosis of vestibular neuritis attributed to *varicella zoster* infection was made by correlating the acute presentation of vertigo, horizontal, horizontal nystagmus at rest, normal hearing test [3] and the active *varicella zoster* infection. Absence of vertical nystagmus and other cerebellar signs such as ataxic gait and impaired heel-shin test makes central causes of vestibular disorder very unlikely [4,5].

Infection from the vestibular ganglia reservoir resulting in reactivation of *herpes simplex* type 1 have been reported to lead to vestibular neuritis [6,7]. This is unlikely in this case as there was no past history to suggest *herpes simplex* infection. Other possible viral causes of acute vestibular neuritis cannot be indubitably excluded in this case as specific confirmatory serological cerebrospinal fluids or blood test for specific viruses were not performed. Ideally, serological or molecular test of cerebrospinal fluids (CSF) to confirm *varicella zoster* and other potential causative viruses should be tested. CSF also is useful in identifying early meningoencephalitis. In this case, CSF was not performed due to the clinical improvement observed in the ward and that CSF investigations rarely yield correlative findings [6,8]. Other investigations to look for mildly inflamed VIII nerve is the auditory test, which within this case showed a normal result. However, this is not a reliable method as compared to brainstem auditory evoked test which is better at looking for mildly inflamed nerve.

Treatment for vestibular neuritis is mainly symptomatic. As per needed doses of prochlorperazine were offered to the patient. Literature on the role of antiviral treatment is limited and intravenous antiviral treatment is recommended only for severe neurological complications [9]. Similarly, there is limited evidence to support the use of corticosteroid for symptomatic recovery [10], especially when patient has normal hearing. Recovery from vestibular neuritis usually occurs in approximately two to four weeks 3 with small percentage of children (5%) having persistent nystagmus at 2 years [11].

In conclusion, vestibular neuritis presented with vertigo is a rare complication of an acute *varicella zoster* virus infection in children. Clinical suspicion and timely admission is crucial for investigation, supportive measures and close monitoring of symptoms progression.

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