Case Report

A Rare Presentation of Post-Herpes Zoster VIII and XII Cranial Nerve Palsies

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Abstract
Herpes zoster (shingles) is a sporadic disease that results from reactivation of latent Varicella Zoster Virus (VZV) from dorsal root ganglia. Central Nervous System involvement may follow localised herpes zoster. Many patients without signs of meningeal irritation have Cerebro Spinal Fluid (CSF) pleocytosis and moderately elevated levels of CSF protein. Multiple cranial nerve involvement is seen post-Herpes zoster but involvement of XII cranial nerve has been rare. In this paper, we present a case, who is immunocompromised with post-Herpes V, VIII and XII cranial nerve palsies

Introduction:
VZV causes chickenpox (Varicella), after which the virus becomes latent in cranial nerve, dorsal root and autonomic nervous system ganglia along the entire neuraxis1. Decades later, a declining VZV-specific host immunity leads to virus reactivation, usually resulting in herpes zoster, which is characterized by pain and rash restricted to 1 to 3 dermatomes2,3. Histopathologic examination of representative dorsal root ganglia during active herpes zoster demonstrates hemorrhage, edema, and lymphocytic infiltration. It occurs at all ages, but its incidence is highest (5–10 cases per 1000 persons) among individuals in the sixth decade of life and beyond. Herpes zoster is more severe in immunocompromised than immunocompetent individuals.

Active replication of VZV in other organs, such as the lung or the brain, can occur during herpes zoster but is uncommon in the immunocompetent host. Herpes zoster is characterized by a unilateral vesicular dermatomal eruption, often associated with severe pain. The dermatomes from T3 to L3 are most frequently involved. The onset of disease is heralded by pain within the dermatome, which may precede lesions by 48–72 h; an erythematous maculopapular rash evolves rapidly into vesicular lesions. In the normal host, these lesions may remain few in
number and continue to form for only 3–5 days. When branches of the trigeminal nerve are involved, lesions may appear on the face, in the mouth, in the eye, or on the tongue. Infection of CNS caused by herpes zoster may manifest as encephalitis, neuritis, myelitis, VZV vasculopathy, or ophthalmic herpes. Symptomatic meningoencephalitis is characterized by headache, fever, photophobia, meningitis and vomiting. Many patients without signs of meningeal irritation have CSF pleocytosis and moderately elevated levels of CSF protein. CSF pleocytosis is seen in 40% of patients with HZ without signs of meningitis. Multiple cranial nerve involvement is seen post-Herpes zoster but involvement of XII cranial nerve has been rare. XII cranial nerve palsy in Herpes zoster infection have been reported in few case reports.

**Case Report**

A 35 year old Hindu gentleman, right handed person, farmer by occupation hailing from Shimoga presented to our hospital with complaints of headache and on & off fever since 3 months easy fatiguability since 1 ½ months. Fever was mild to moderate grade, not associated with chills or rigors, partly relieved by medication. Headache was diffuse, holocranial. No history of nausea or vomiting. Patient was admitted in our hospital in Feb 2013. He is a known case of HIV positive since September 2012 and was started on Anti Retroviral Therapy (ART) with initial CD4 count of 308/µl. He was on Zidovudine+ Lamivudine + Nevirapine regimen. In January 2013, he developed anaemia, hence the regimen was changed to Tenofovir + Lamivudine+ Nevirapine. Patient also gave history of deviation of angle of mouth to right- 5 years back, along with difficulty in closing left eye. He took Ayurvedic medication for the same and had partial improvement in few days. He also gave H/O vesicular eruptions suggestive of Herpes zoster lesions over the left lower face, left oral mucosa and over left lateral border of tongue 7 months back. These lesions had appeared overnight. For which he took Ayurvedic medication and 2 days later he noticed decreased hearing in left ear, difficulty in speaking and deviation of tongue to left on protruding it out.

On examination, Patient was moderately built and nourished. He was afebrile, with a heart rate of 96/min, with a respiratory rate of 12/min, & Blood Pressure was 110/70 mmHg. Pallor(+++). Evidence of post herpes zoster healed lesions on face and oral cavity was present. CNS- Higher mental function- normal, Cranial nerves examination: Left LMN facial palsy & Left sensorineural hearing loss was present. Left XII nerve palsy (Figure 1).

**Motor system**- Hypotonia & hyperreflexia- all 4 limbs; Plantar – bilateral flexor, Brisk abdominals

**Sensory system**: Decreased touch, pain in left lower face;

**Fundus**: ? HIV retinopathy, resolving papilledema. No signs of meningeal irritation

Cerebellar signs- absent
**Investigations**
Complete blood count: Hemoglobin- 3g%, WBC count- 4700, MCV- 78, Platelet count- 2.4 lakh/mm3, Peripheral Smear- microcytic hypochromic anemia, Random Blood Sugar-137mg/dl, Liver Function Test- within normal limits, HIV(ELISA)- reactive
Chest X-ray, USG abdomen, MRI brain(plain + contrast): normal study
CSF analysis: protein- 0.022g/dl (220mg/l);Sugar- 40mg/dl; Cells- 2 cells/mm³; Culture- sterile; VZV DNA (polymerase chain reaction)- Negative

Patient was transfused with blood. Iron, Vitamin B₁₂ and Folic acid tablets were given. Anemia was corrected. Physiotherapy was given for facial palsy. He had symptomatically improved at the time of discharge.

**Discussion:**
The presence of post-Varicella lesions on the skin in our patient made us think of the possibility of Varicella-induced cranial nerve palsies. We kept a differential diagnosis of Herpes Zoster meningoencephalitis causing multiple cranial nerve palsy. As MRI(Plain & Contrast) was normal and CSF for VZV DNA (PCR) was negative, Herpes Zoster meningoencephalitis was ruled out. After herpes zoster, VZV can also spread to blood vessels of the brain, producing a unifocal or multifocal vasculopathy, particularly in immunocompromised individuals. Virological analyses of patients who died from VZV vasculopathy have revealed both VZV DNA and VZV antigen in cerebral arteries, indicating active virus infection⁴. Detection of VZV DNA or antibody in CSF
confirms a diagnosis of VZV vasculopathy\(^6\). In our patient, CSF protein, sugar, cells were within normal limits, CSF culture was negative and CSF VZV DNA(Polymerase chain reaction) was negative. CSF antibody was not done. Neurological complications of Varicella can be due to direct invasion of the virus or by immunological mechanisms. Our patient presented in the resolving phase of Varicella infection thus explaining the absence of Varicella active lesions. Varicella zoster virus is associated with multiple cranial neuropathies, the most common being facial nerve paralysis.\(^{10}\) Most of the cases described in the literature are due to Varicella zoster reactivation.

In conclusion, although Varicella is generally a mild and self-limiting illness, it can have complications like multiple cranial nerve palsies. This case alerts us to the occurrence of rare complications of common diseases.

**References**