CASE REPORT

Varicella Meningitis presenting with Neurogenic Dysphagia

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Abstract

Varicella zoster virus (VZV) infection is common, but central nervous system involvement is rare. We report a patient with herpes zoster oticus and aseptic meningitis with 8–10th cranial nerve involvement who responded well to treatment with aciclovir.

Keywords: Aseptic meningitis, Lower cranial nerve involvement, Varicella.

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Introduction

Neurological manifestations of VZV infection are well known. Varicella neurological manifestations include encephalitis, seizures, meningitis, etc.[1,2] Cranial nerve involvement with varicella meningitis is known, but 9th and 10th cranial nerve palsies have rarely been reported. We present a patient with herpes zoster oticus and bulbar palsy.

Case Description

A 50-year-old gentleman presented with a headache for 10 days, numbness around the right ear for a week, and difficulty in swallowing, and nasal regurgitation of liquids for 3 days. He had no fever, vomiting, or altered sensorium. On clinical examination, he was found to have vesicles in the right external ear and involvement of right 8–10th cranial nerves. He had decreased hearing in the right ear, Rinne test was normal (air conduction was better than bone conduction), and there was lateralization to the left ear on Weber test. There was dropping of soft palate on the right side, a decrease in palatal movements with deviation of the uvula to the left side, and the gag reflex was sluggish on the right side. Laryngoscopic examination revealed vesicular lesions over the right tonsillar pillars and posterior pharyngeal wall. Vocal cords and laryngeal mucosa were normal. He was diagnosed to have herpes zoster oticus with probable meningitis. His audiogram revealed mild sensory neural deafness. Magnetic resonance imaging (MRI) brain with gadolinium contrast did not reveal any significant abnormality (Fig. 1). Cerebrospinal fluid (CSF) analysis revealed 70 cells with lymphocytic predominance, normal sugar, and slightly elevated protein level (Table 1). CSF gram stain, nucleic acid amplification test for tuberculosis (GeneXpert MTb/Rif Ultra), acid fast bacilli (AFB) stain, and fungal stain were negative. CSF bacterial culture and AFB cultures did not reveal any growth. CSF herpes simplex polymerase chain reaction (PCR) was negative, but CSF varicella DNA PCR was positive. He was empirically started on intravenous acyclovir 750 mg three times daily soon after admission prior to CSF analysis. He also received steroids, supportive medication, and nasogastric tube feeds. However steroids are not recommended in varicella meningitis. His dysphagia improved within 5–6 days of therapy, and he was able to swallow both liquids and solids without difficulty. He was given 2 weeks of intravenous acyclovir; steroids were tapered and stopped after 2 weeks.

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Discussion

Neurological manifestations may occur with both primary varicella infection or reactivation of infection (herpes zoster). Neurological complications include encephalitis, aseptic meningitis, peripheral motor neuropathy, myelitis, Guillain-Barre syndrome, and stroke syndromes.[3] Ramsay hunt syndrome in association with herpes zoster oticus has been described, however, lower cranial nerves, especially the involvement of 9th and 10th cranial nerves, has rarely been reported.[4–6] There have been case reports of varicella meningo rhombencephalitis or rhombencephalomyelitis.[7]

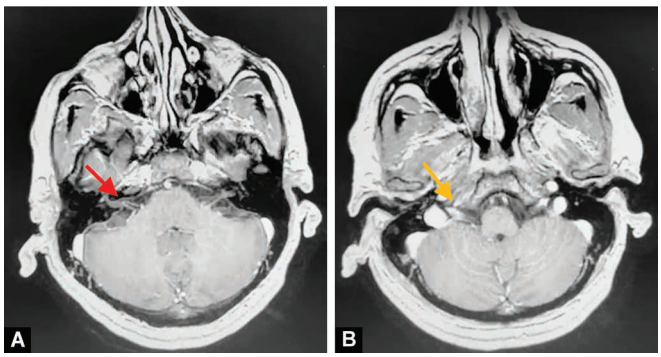

Fig. 1: Postcontrast T1 axials at the level of pons and medulla showing normal 7th–8th nerve complexes (red arrow) and 9th–10th nerves (yellow arrow)

Table 1: CSF findings

CSF parameter	
White blood cell count	70 cells/cc (100% lymphocytes)
Protein	50 mg/dL
Sugar	85 mg/dL
Gram stain, AFB, and fungal stain	negative
GeneXpert Mtb/Rif Ultra	not detected
AFB culture	sterile
Herpes simplex PCR	negative
Varicella zoster DNA PCR	positive

Our patient had CSF pleocytosis and findings suggestive of aseptic meningitis and no abnormalities on MRI brain. The presence of vesicles in the ear (herpes zoster oticus) and CSF varicella PCR positivity confirmed the diagnosis. The presence of exanthem was an easy clinical clue in our patient, but some patients may present with aseptic meningitis without exanthem.

Centers for Disease Control and Prevention estimates that about 30% of people from the United States of America will experience herpes zoster in their lifetime.[8] We do not have data on the incidence of chicken pox or herpes zoster in India . A multicenter, cross-sectional study to estimate seroprevalence in India revealed antibodies against VZV in 62% of the study population.[9] Herpes zoster ophthalmicus was seen in 10–15% of patients with herpes zoster.[10] Neurological complications other than post herpetic neuralgia and ophthalmic nerve involvement are uncommon. Diagnosis of varicella meningitis is based on the presence of typical exanthem, CSF finding of pleocytosis with normal sugar and protein levels (aseptic meningitis), and CSF for VZV PCR positivity. However, a significant proportion of patients may not have skin rash and CSF pleocytosis. More than a third of patients with varicella meningitis did not have cutaneous lesions in case of the series published by Persson and Lozano et al.[11,12] Absence of CSF pleocytosis was reported in 5% of patients by Persson, and 45% by Lozano et al.[11,12] Treatment of choice is intravenous aciclovir for 10–14 days.[13]

Conclusion

Varicella meningitis is uncommon but needs prompt diagnosis and treatment to reduce morbidity. A 9th and 10th cranial nerve involvement is rare, and the absence of cutaneous lesions does not rule out the possibility of varicella meningitis.

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