



Figure 1: Healing chicken pox lesions over the face with facial palsy

Isolated facial palsy in varicella

Sir,

Isolated facial palsy as a neurological complication of varicella is reported.^[1-5] As such neurological complications caused by varicella are estimated to occur in approximately 0.01-0.03% of infections. Frequent neurologic complications of varicella are cerebellar ataxia and encephalitis while the rare complications are transverse myelitis, aseptic meningitis, Guillain-Barré syndrome, meningo-encephalitis, ventriculitis, optic neuritis, delayed contralateral hemiparesis, peripheral motor neuropathy, cerebral angiitis, Reye's syndrome, and facial paralysis.^[1]

Ten days after varicella (chicken pox), a 26-year-old house wife presented with a left facial palsy without any neurological symptoms. While she was recovering from varicella, she developed inability to close her left eye and deviation of mouth to the right side. There was no clinical history of retroauricular pain, hyperacusis, decreased production of tears, and altered taste. On examination, there were multiple discrete crusted lesions all over the trunk, back, extremities, and healed eroded lesions over

the face. Complete neurological examination revealed lower motor neuron type of facial palsy without any other abnormal features [Figure 1]. Other systemic examination revealed no abnormalities. Vital signs are normal. Patient is not hypertensive. Routine investigations were within normal limits. Serological tests for syphilis and human immunodeficiency virus (HIV) infection are nonreactive. Varicella zoster virus IgG (13.2 NTU) and VZV IgM (13.7 NTU) were positive. Computed tomography (CT) scan of brain revealed no abnormality. The patient was treated with acyclovir 800 mg five times/day for 7 days and prednisolone 10 mg three times/day in tapering doses for 3 weeks. The patient was examined after 3 weeks and complete recovery of facial palsy was noticed.

Facial paralysis in varicella can be seen either prior to or after the appearance of exanthem. It may occur 5 days before exanthema appears or during 16-day period after varicella is diagnosed. Facial palsy as a complication of varicella can be a result of preeruptive hematogenous or neurogenous spread of varicella zoster virus^[1,3,4] Bilateral facial palsy can also occur rarely during varicella.^[3] Though in our case facial nerve palsy developed during varicella infection with elevated V-Z IgG and IgM antibodies, it should be differentiated from closely identical conditions of facial palsy like Bell's palsy and Ramsay Hunt syndrome.

Table 1: Differential diagnosis of our case

	Cause	Distinguishing features
Classical Bell's palsy	HSV Type 1	History of exposure to cold, no exanthematous, rash, no constitutional symptoms, sudden onset, prolonged course
Ramsay Hunt syndrome	Varicella zosteriform virus	Pronounced prodrome of pain, hyperacusis, vesicular eruption over the external pinna and in ear canal or pharynx
Our case	Varicella zosteriform virus	History of varicella, active and healed lesions over the body, elevated VZV antibodies (IgG and IgM)

Table 1 summarizes the differentiating features of our case and these two conditions. Classically Bell's palsy has been defined as idiopathic and the cause of the inflammatory process in the facial nerve remains uncertain. Recently attention has focused on infection with herpes simplex virus type 1 (HSV-1) as a possible cause because research has found elevated HSV-1 titers in affected patients. However, studies have failed to isolate viral DNA in biopsy specimens leaving the causative role of HSV-1 in question.^[6] Ramsay Hunt syndrome is due to reactivation of varicella zoster virus affecting geniculate ganglia and facial nerve. Many conditions can produce isolated facial nerve palsy like cholesteatoma, salivary tumors, Guillain Barre syndrome, Lyme disease, otitis media, sarcoidosis, and some influenza vaccines. These conditions usually have other distinguished features.^[6]

The prognosis of facial palsy due to varicella is generally good and 80% cases recover with or without treatment, but specific acyclovir and prednisolone therapy may accelerate the complete recovery like in our case.

**G. Raghu Rama Rao, A. Amareswar,
Y. Hari Kishan Kumar, Radha Rani**
Surya Skin Care and Research Center, Maharanipecta,
Visakhapatnam, AP, India

Address for correspondence: Dr. G. Raghu Rama Rao,
Surya Skin Care and Research Center, 15-1-2,
Nowroji Road, Maharanipecta,
Visakhapatnam - 530 002, AP, India.
E-mail: graghuramarao@hotmail.com

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