Review Article

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Herpes Zoster Virus Infection of Facial Nerve Treated Successfully without Corticosteroid

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Abstract

Herpes zoster virus (HZV) infection of the central and peripheral nervous systems can be of serious consequences. Most of the time it affects the respective dermatomes of the dorsal root ganglia without further invasion.

But in some cases it progresses to invade one or multiple cranial nerves in their motor (VI, VII) and /or sensory (V^{123}, VIII) distribution. The geniculate ganglion of the facial nerve (VII) as well as the trigeminal ganglion (V) are particularly vulnerable.

Rarely there may be a segmental radiculopathy, or myelopathy, too.

We present here a patient who presented with herpes zoster eruptions over face and neck unilaterally with ipsilateral VIIth. cranial nerve (facial) LMN palsy, collectively known as Ramsay Hunt syndrome.

He was treated conservatively without using corticosteroid and recovered within few weeks,without any residual neurological deficit.

Keywords: Herpes Zoster, Facial nerve, Lower motor neuron, Corticosteroid.

INTRODUCTION

Herpes zoster virus, also known as Vericella zoster virus (VZV) is a neurotropic DNA virus which can cause devastating central and peripheral nervous system infections.

After an episode of varicella (chicken-pox), the varicella-zoster virus (VZV) remains dormant in the nervous system, the sensory ganglia in particular. Herpes zoster typically occurs in adults or elderly with reactivation of dormant virus in the sensory ganglia and propagation of the virus down to the respective dermatomes. In case of cranial nerves

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namely, the sensory projections of trigeminal nerve, the skin along forehead, eyelids, malar and mandibular regions will be affected, while the auricle, neck and upper chest dermatomes are affected from involvement of dorsal root ganglia of cervical and upper thoracic nerves.

Moreover, the motor parts of cranial and peripheral nerves can be occasionally afflicted, as seen in Ramsay Hunt syndrome where the lower motor neuron (LMN) facial neuropathy[1] is the prominent manifestation.

In addition, the virus may occasionally invade the peripheral motor roots causing segmental radiculopathy.[2]

We depict a case of herpes zoster presenting with shingles along multiple dermatomes including cranial nerves - trigeminal (sensory) to cervical and uppermost thoracic dorsal root ganglia as well as LMN facial paresis.

CASE REPORT

A 52 yr old male presented with painful non-pruritic rashes over face and neck area of left side for last 3 days. He initially had a bout of low grade intermittent fever which lasted for 2 days and abated spontaneously without any treatment. This was followed by skin rashes over the left auricle, lower eyelid, malar and mandibular regions and left side of neck and upper chest.

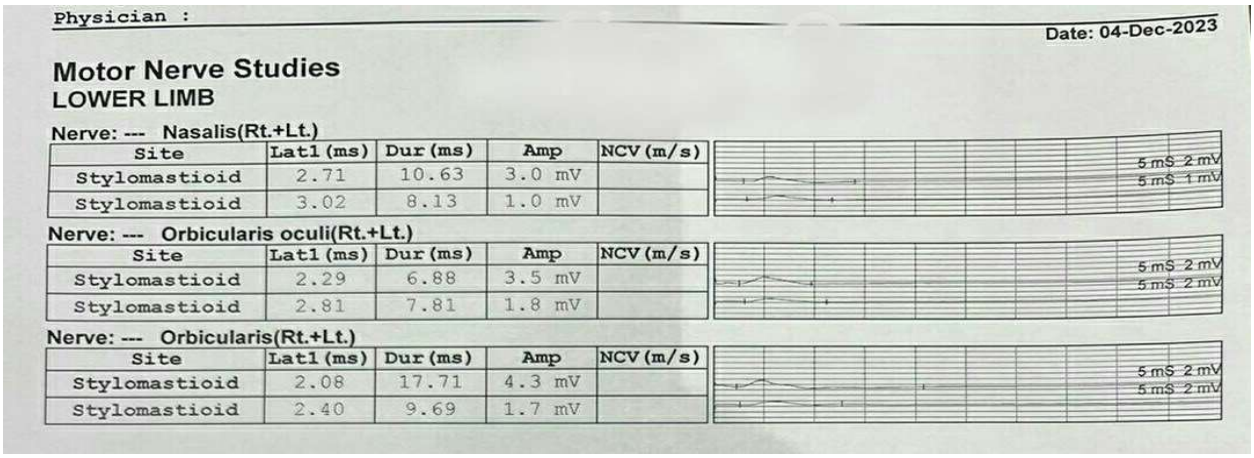
He then developed drooping with inability to blow his left cheek and seepage of saliva, water and food materials from left side of his mouth during eating. He also complained of increased tearing from his left eye.

Taste perception was impaired in left side of his tongue. However he denied having any vertigo, vomiting, tinnitus, deafness or hyperacusis. He also denied having visual blurring or double vision.

On examination, his mental status was normal with normal gait and motor, sensory and cerebellar functions.

Cranial nerves examination showed asymmetric deviation of angle of mouth towards right side, inability to blow his left cheek, incomplete closure of his left eye and absence of wrinkles in left forehead (fig. 1) indicating lower motor paresis of his left facial nerve. His jaw movements were normal bilaterally indicating no involvement of motor functions of mandibular nerves.

Fig. 1: Rash with incomplete closure of left eye



Physician :

Date: 04-Dec-2023

Motor Nerve Studies

LOWER LIMB

Nerve: --- Nasalis(Rt.+Lt.)

Site	Lat1 (ms)	Dur (ms)	Amp	NCV (m/s)
Stylomastioid	2.71	10.63	3.0 mV	
Stylomastioid	3.02	8.13	1.0 mV	

5 mS 2 mV
5 mS 1 mV

Nerve: --- Orbicularis oculi(Rt.+Lt.)

Site	Lat1 (ms)	Dur (ms)	Amp	NCV (m/s)
Stylomastioid	2.29	6.88	3.5 mV	
Stylomastioid	2.81	7.81	1.8 mV	

5 mS 2 mV
5 mS 2 mV

Nerve: --- Orbicularis(Rt.+Lt.)

Site	Lat1 (ms)	Dur (ms)	Amp	NCV (m/s)
Stylomastioid	2.08	17.71	4.3 mV	
Stylomastioid	2.40	9.69	1.7 mV	

5 mS 2 mV
5 mS 2 mV

Fig. 2: Decreased CMAP ampl in Left Orb. oculi, oris & nasalis

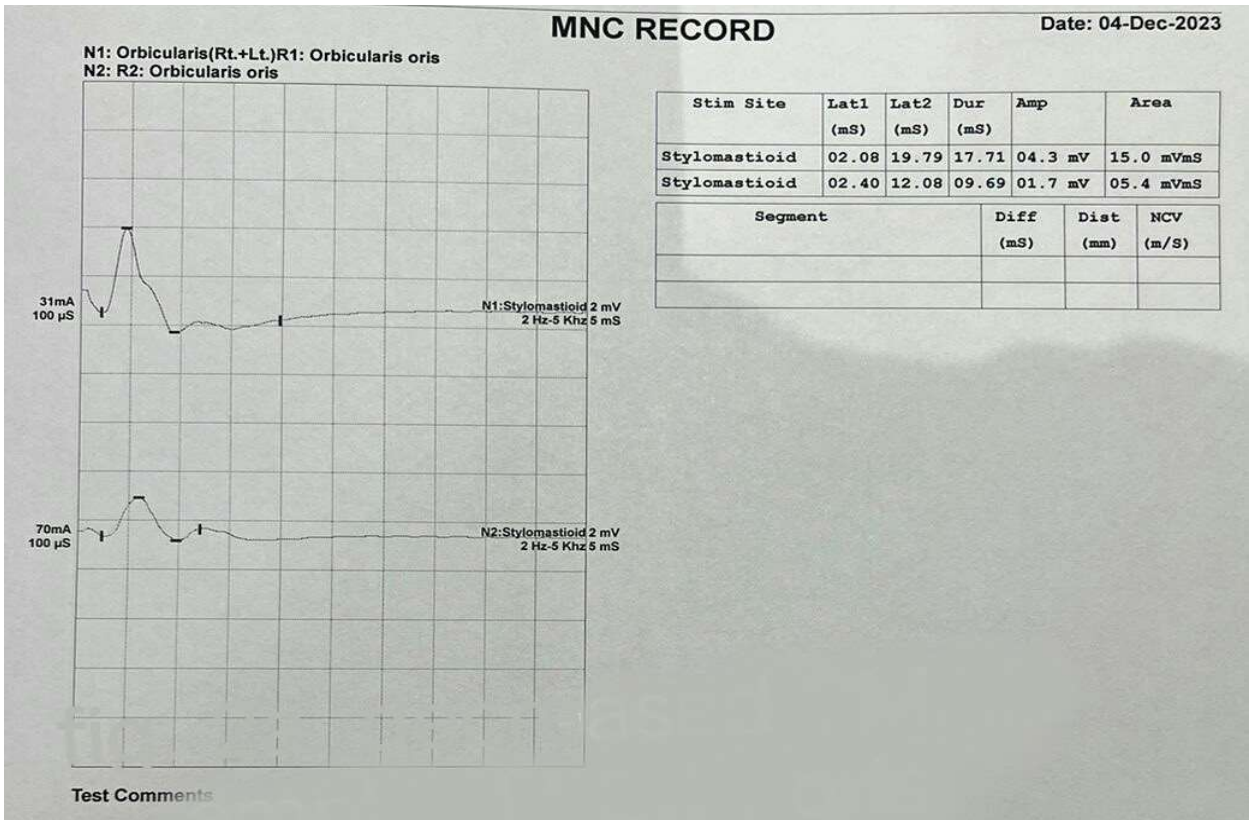


Fig. 2a: Decreased CMAP ampl in Left Orb. oris

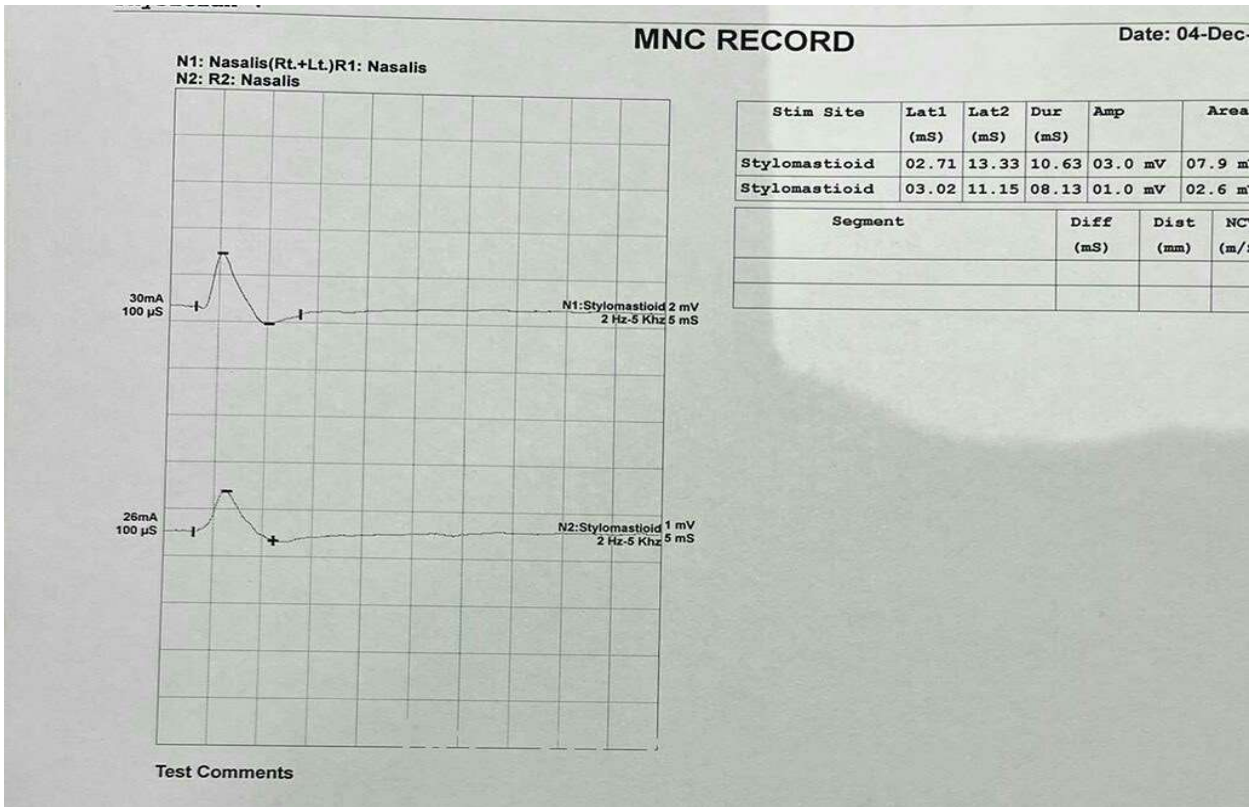


Fig. 2b: Decreased CMAP ampl in Left nasalis

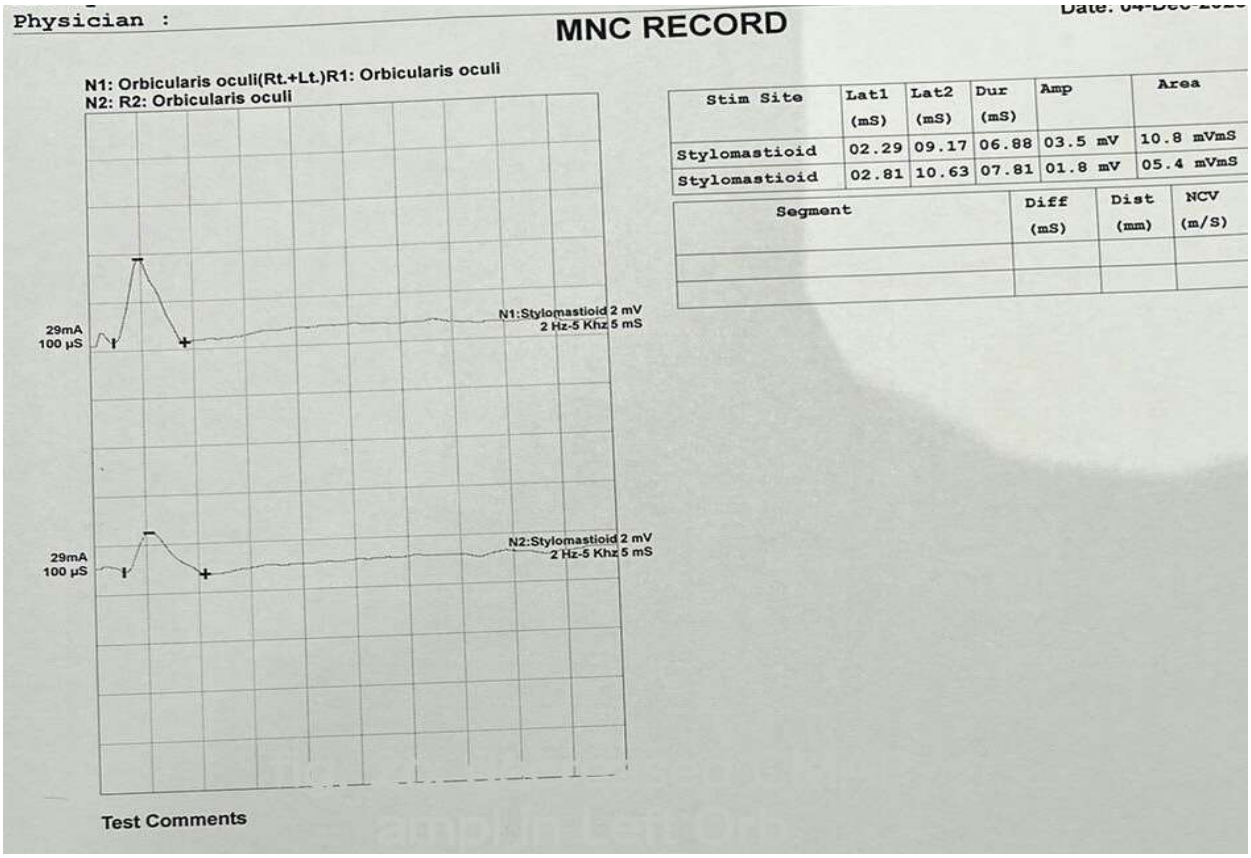


Fig. 2c: Decreased CMAP ampl in Left Orb. oculi

Vesicular eruptions were present in left trigeminal, cervical and uppermost thoracic dermatomes (fig. 1) suggestive of herpes zoster affecting dorsal root ganglia of left cervical and upper thoracic nerves as well as trigeminal ganglion. However, there was no lesions on the mucus membrane of mouth, cheek, nose, eye or external auditory meatus.

Thus the patient was diagnosed as left sided lower motor neuron facial palsy caused by herpes zoster (Ramsay Hunt syndrome) with trigeminal (sensory) neuropathy with cervical and upper thoracic dorsal root ganglionopathy.

NCV study of bilateral facial nerve showed decreased CMAP amplitudes from left orbicularis oculi, nasalis and orbicularis oris indicating axonopathy.

After consultation with dermatologist, he was treated conservatively with antiviral therapy valacyclovir 1000 mg 3 times daily for 7 days.

Physical therapy was instituted with a view to improve the tone and power of facial muscles.

Corticosteroid was not given though the patient was not diabetic or hypertensive.

The patient had uneventful recovery in next 3 weeks without having any residual neurological deficit.

DISCUSSION

About 1% of varicella-zoster infections can present with Ramsay Hunt syndrome phenotype.

Clinical features of this syndrome include otalgia, vesicular rash in the auricle or external auditory meatus with ipsilateral lower motor neuron facial palsy.[1] Occasionally there may be segmental radiculopathy.[2]

However, the diagnosis of herpes zoster needs demonstration of viral DNA from a Tzanck smear obtained from the skin lesions which is not widely available and does not preclude the diagnosis of Ramsay Hunt syndrome.

This patient had signs of LMN facial nerve palsy along with rashes along specific dermatomes consistent with the involvement of trigeminal nerve and dorsal root ganglia of cervical and upper thoracic nerves.

NCV study of facial nerves was consistent with left sided facial axonopathy.

Finally, though there are reports of successful treatment with acyclovir plus corticosteroid (3,4), we preferred treatment without steroid considering the risk of keratitis.

There was no residual neurological deficit on 3 months follow up.

CONCLUSION

Patients with Ramsay Hunt syndrome, when early diagnosed and treated with antiviral therapy should have high chance of complete recovery. Use of steroids in such situations, though advocated widely, may be judged cautiously not to induce any potential adverse effects.

Abbreviations: V^{123} = ophthalmic, maxillary and mandibular (sensory) div of Trigeminal nerve. VII = Facial nerve, VI=abducens nerve, VIII = Vestibulocochlear nerves; LMN = Lower motor neuron, NCV = nerve conduction velocity, CMAP = compound muscle action potential.

.Conflict of interest: There was no conflict of interest

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