

- 5.Spinner, R.J., Atkinson, J.L.D., Tiel, R.L. Peroneal intraneural ganglia: the importance of the articular branch. A unifying theory. *J Neurosurg.* 2003,99,2,330–343.
- 6.Spinner, R.J., Desy, N.M., Amrami, K.K. The cystic transverse limb of the articular branch: a pathognomonic sign for peroneal intraneural ganglia at the superior tibiofibular joint. *Neurosurgery.* 2006,59,1,157–166.
- 7.Visser, L.H. High-resolution sonography of the common peroneal nerve: detection of intraneural ganglia. *Neurology.* 2006,67,8,1473–1475.
- 8.Young, N.P., Sorenson, E.J., Spinner, R.J., Daube, J.R. Clinical and electrodiagnostic correlates of peroneal intraneural ganglia. *Neurology.* 2009,72,5,447–452.
- 9.Panwar, J., Mathew, A., Thomas, B.P. Cystic lesions of peripheral nerves: are we missing the diagnosis of the intraneural ganglion cyst? *World J Radiol.* 2017,9,5,230–244.
- 10.Spinner, R.J., Desy, N.M., Rock, M.G., Amrami, K.K. Peroneal intraneural ganglia. Part I. Techniques for successful diagnosis and treatment. *Neurosurg Focus.* 2007,22,6,:E16.
- 11.Spinner, R.J., Desy, N.M., Rock, M.G., Amrami, K.K. Peroneal intraneural ganglia. Part II. Lessons learned and pitfalls to avoid for successful diagnosis and treatment. *Neurosurg Focus.* 2007,22,6,:E27.
- 12.Wilson, T.J., Mauermann, M.L., Rock, M.G., Spinner, R.J. Outcomes following surgery for peroneal intraneural ganglion cysts. *Muscle Nerve.* 2018,57,6,989–993.
- 13.König, R.W., Wirtz, C.R., Friedrich, S., Pauly, M., Antoniadis, G. Clinical outcome after decompression of intraneural peroneal ganglion cyst and its morphologic correlation to postoperative nerve ultrasound. *J Neurosurg.* 2020,133,1,233–239.
- 14.Wilson, T.J., Stone, J.J., Howe, B.M., Rock, M.G., Spinner, R.J. Joint outcomes following surgery for superior tibiofibular joint-associated peroneal intraneural ganglion cysts. *Neurosurgery.* 2020,86,3,383–390.
- 15.Kankam, H.K.N., Duraku, L.S., Hundepool, C.A., George, S., Chaudhry, T., Power, D.M. Outcomes following surgery for common peroneal nerve intraneural ganglion cysts: a case series and systematic review. *Eplasty.* 2023,23:e39.
- 16.Broekx, S., Van Der Straeten, R., D'haen, B., Vandevenne, J., Ernon, L., Weyns, F. Intraneural ganglion cyst of the common peroneal nerve causing foot drop in a 12-year-old child. *Clin Neurol Neurosurg.* 2021,209,106915.
- 17.Pemmari, A., Mäkelä, K., Niemi, J., Kiekara, T., Himanen, S.L. Large intraneural ganglion cyst in the peroneal nerve. *Case Rep Neurol.* 2022,14,1,44–50.
- 18.della Vecchia, G., Baldi, A., Passavanti, M.B., Lucariello, A., De Luca, A., De Blasiis, P. Acute foot drop caused by intraneural ganglion cyst of the peroneal nerve: literature review and case report. *J Pers Med.* 2023,13,7,1137.
- 19.Adil, A., Basener, C., Checketts, J. Intraneural synovial cyst of the common peroneal nerve: an unusual cause of foot drop with four-year follow-up. *Case Rep Orthop.* 2019,2019:8045252.
- 20.Smith, B.W., Howe, B.M., Amrami, K.K., Spinner, R.J. High-resolution MRI of a peroneal intraneural ganglion cyst arising from the knee joint: illustrative case. *J Neurosurg Case Lessons.* 2021,1,21,:CASE21130.

MULTICRANIAL NEUROPATHY IN REACTIVATED VARICELLA ZOSTER VIRUS INFECTION—A CASE REPORT

Garima Shant¹, Deepak Jain ², Manisha Dolia³

¹ Department of Medicine, PGIMS, Rohtak, India,

² Department of Medicine, PGIMS, Rohtak, India

³ Department of Medicine, PGIMS, Rohtak, India,

ABSTRACT

Herpes zoster typically presents with dermatomal rash and radicular pain. Although rare, but it can cause various neurological complications such as meningitis, encephalitis, myelitis, cerebral angiitis, subacute amyotrophy and cranial nerve palsy. We report a case of a 63-year-old male who presented with otalgia, facial asymmetry, hearing loss, and subsequently developed dysphagia and hoarseness. Clinical examination revealed involvement of multiple cranial nerves (VII, VIII, IX, and X), consistent with polyneuritis cranialis. Notably, there were no characteristic vesicular eruptions. Cerebrospinal fluid analysis demonstrated lymphocytic pleocytosis with elevated protein, and polymerase chain reaction confirmed VZV DNA. A diagnosis of herpes zoster meningitis with cranial polyneuropathy was established. The patient was treated with intravenous acyclovir and corticosteroids, resulting in complete

recovery of neurological deficits on follow-up. This case underscores that Varicella zoster virus can be an important and often underrecognized cause of polyneuritis cranialis, highlighting the need for high clinical suspicion to enable timely diagnosis and management and to prevent long-term sequelae

KEYWORDS

Varicella zoster virus; Polyneuritis cranialis; Herpes zoster meningitis

INTRODUCTION

Varicella Zoster Virus is a neurotropic virus which cause two distinct clinical syndromes: varicella and herpes zoster. Herpes zoster is caused by spontaneous reactivation of VZV which become latent in sensory ganglia, predominately in trigeminal and thoracic ganglion cells during primary infection with chickenpox. The waning of immunity with ageing, sys-

temic illness or drugs can lead to reactivation of VZV presenting as painful vesicular eruptions in dermatomes, most common sites being trunk (T5-T10) followed by craniocervical region. Involvement of craniocervical region is frequently associated with meningeal signs and high diseases severity [1]. Among the cranial nerves 5, 7 and 8 nerves are involved more frequently manifesting in the form of herpetic neurologic syndromes like ophthalmic herpes and Ramsay Hunt syndrome (RHS). The classical presentation of RHS is herpetic eruptions at external auditory canal, otalgia and ipsilateral lower motor neuron facial nerve palsy. Cranial nerves like 9 and 10 can also be involved presenting as herpes occipitocollaris. Rarely 3, 4 and 6 cranial nerve involvement is also reported in literature [2]. Although inclination towards these particular sites is not fully understood but may be explained to some extent that herpetic neurologic syndromes are more likely to occur if the distance of the ganglia from the skin is short. Apart from the typical dermatomal rash, two variants zoster sine herpete (absence of rash) and cutaneous dissemination can also be seen in herpes zoster.

Approximately 2–23% of unilateral facial palsy cases without vesicles are due to herpes zoster sine herpete and may be erroneously diagnosed as Bell's palsy [3]. Furthermore, vesicular eruptions may appear after the onset of facial weakness in 12–34% of patients rendering Ramsay Hunt syndrome indistinguishable from Bell's palsy [2,4]. RHS when untreated has poor prognosis and low recovery rate in contrary to Bell's palsy so an early diagnosis and treatment have a crucial role for full recovery of cranial nerve palsy [5].

CASE REPORT

A 63-year-old man with no known comorbidities presented with a 10-day history of right ear pain with discharge, 7 days of facial asymmetry, and 5 days of headache, photophobia and vomiting. He also reported a history of diminished hearing in the right ear for past 4 days, without associated tinnitus or vertigo. He had initially been managed elsewhere as a case of complicated otitis media with antibiotics. However, his symptoms progressively deteriorated. He subsequently presented to us with acute-onset dysphagia and hoarseness of voice for 1-day. There was no history of cutaneous eruptions, and his family history was also noncontributory. Higher mental functions, sensory and motor examination was normal. Cranial nerve examination revealed right lower motor neuron facial nerve palsy. [Figure 1,2] Rinne test was positive on right side (BC > AC) and weber test showed localization to left ear indicating right mixed hearing loss. Oropharyngeal examination revealed deviation of uvula to left side and absent gag reflex on right side stipulating involvement of 9 & 10 cranial nerves on right side. Rest of cranial nerves examination was normal. There was no neck rigidity, Brudzinski or Kernig sign. His cardiovascular, respiratory and abdominal examination was unremarkable. Otoscopy showed erythema, crusting in right ear canal and perforation of right tympanic membrane. Indirect laryngoscopy exhibited immobility of right vocal cord. A pure tone audiogram showed right profound mixed hearing loss. Fundus examination was normal in both eyes. MRI brain reveals normal study [Figure 3]. In view of persistent headache and vomiting, CSF analysis was sent which

showed 167 cells predominately lymphocytes, protein 117 mg/dl, ADA 6.3, sugar 70 mg/dl and positive for VZV PCR. Routine blood investigations showed Hb 14.3 mg/dl, TLC 15,000/mm³ and platelet count of 1.4 lakh/mm³. Blood sugar was 108 mg/dl. Rest liver function tests, renal function tests and thyroid profile was normal. The diagnosis of herpes zoster meningitis with cranial polyneuropathy involving seventh, eighth, ninth and tenth cranial nerve was made. Patient was given intravenous acyclovir for 10 days along with steroids. Exposure keratoconjunctivitis was managed with eye patching and artificial tears. At one month, the patient's facial nerve palsy resolved completely. Three months later, his dysphagia and hoarseness subsided fully. Follow-up laryngoscopy demonstrated bilaterally mobile vocal cords. —

DISCUSSION

Herpes zoster can occur at all ages with highest prevalence in sixth decade of life (5–10 case per 1000 person per year) [6]. CNS involvement may follow localized herpes zoster in the form of meningoencephalitis, myelitis, cerebral angiitis, cranial nerve palsies, amyotrophy etc. Although trigeminal, facial and vestibulocochlear nerves are more commonly involved, rarely involvement of other cranial nerves can also be seen. This case was unique as involvement of multiple cranial nerves (7, 8, 9 and 10) is rarely seen. Only few cases of VZV infection leading to cranial polyneuropathy are reported in literature [7,8]. Also, Herpes zoster meningitis is uncommon neurological complication which can occur before or after the onset of vesicular rash along the dermatomal distribution. It can occur even without cutaneous eruptions (Zoster Sine Herpete) as seen in our case, although with a higher incidence in immunocompromised.

Various theories have been hypothesized for coexistence of multiple cranial polyneuropathies. The reactivation of latent VZV in the cranial sensory ganglia (geniculate/trigeminal) may activate cellular immune responses resulting in collateral damage to adjacent cranial motor nerves due to the close anatomical proximity between them [2]. Other proposed theories include transmission via anastomotic connections between the facial nerve and other affected cranial nerves, simultaneous activation of virus in multiple ganglia or spread through CSF [9,10]. Haanpää et al. demonstrated VZV in the cerebrospinal fluid of 21% of patients with cutaneous lesions, despite the absence of clinical manifestations of meningoencephalitis.

The detection of virus by PCR in mononuclear cells of external auditory canal fluid, blood, CSF is the gold standard test in diagnosis of VZV infection. Serological test detecting antibodies have lower sensitivity.

Use of FDA approved antivirals such as acyclovir, valacyclovir, famciclovir etc. along with corticosteroids remains the mainstay of treatment with higher recovery rate when initiated within 3 days of symptoms onset [4]. When antiviral plus steroid therapy was compared with steroid alone therapy or antiviral alone therapy, it showed better nerve function recovery [11,12].

CONCLUSION

This presentation is rarely seen in clinical practice. Apart from common causes of polyneuritis cranialis like vascular, tumor, chemotherapy, diabetes, vasculitis, rare causes like

VZV infection should be considered and CSF analysis should be encouraged. We emphasize the need of high suspicion to diagnose the syndrome early as delay in treatment have unfavourable outcomes.

Declaration of patient consent

The authors declare that written, informed consent was taken after discussion with the patient and family members for possibility of publication of the case along with clinical data and investigations. The patient and their family have been assured of maintaining anonymity during the process.

Financial support and sponsorship - Nil

Conflict of interest – Nil

Acknowledgment - Nil

REFERENCES

1. Viral infections of nervous system and prion disease. In Ropper H. Allan, Samuels A. Martin, Klein P. Joshua, Prasad Sashank, editors. Adam and Victor's Principles of Neurology. 11th ed. New York: McGraw Hill; 2019.p.775-778
2. Tsau, P.W., Liao, M.F., Hsu, J.L., Hsu, H.C., Peng, C.H., Lin, Y.C., Kuo, H.C., Ro, L.S. Clinical Presentations and Outcome Studies of Cranial Nerve Involvement in Herpes Zoster Infection: A Retrospective Single-Center Analysis. J Clin Med. 2020 Mar 30;9,4,946.
3. Furuta, Y., Ohtani, F., Aizawa, H., Fukuda, S., Kawabata, H., Bergström, T. Varicella-zoster virus reactivation is an important cause of acute peripheral facial paralysis in children. Pediatr Infect Dis J. 2005 Feb;24,2,97-101.
4. Murakami, S., Honda, N., Mizobuchi, M., Nakashiro, Y., Hato, N., Gyo, K. Rapid diagnosis of varicella zoster virus infection in acute facial palsy. Neurol. 1998;51,4,1202-1205.
5. Monsanto, R.D., Bittencourt, A.G., Bobato Neto, N.J., Beilke, S.C., Lorenzetti, F.T., Salomone, R. Treatment and Prognosis of Facial Palsy on Ramsay Hunt Syndrome: Results Based on a Review of the Literature. Int Arch Otorhinolaryngol. 2016 Oct;20,4,394-400.
6. Al-Ani, R.M. Ramsay Hunt Syndrome With Cranial Polyneuropathy and Delayed Facial Nerve Palsy: A Case Report. Cureus. 2022 Jul 29;1,7 :e27434.
7. Stornaiuolo, A., Iodice, R., De Simone, R., Russo, C., Rubino, M., Braca, S., Miele, A., Tozza, S., Nolano, M., Manganeli, F. Multiple cranial neuropathy due to varicella zoster virus reactivation without vesicular rash: a challenging diagnosis. Neurol Sci. 2023 Oct;44,10, 3687-3689.
8. Tecellioglu, M., Kamisli, S., Erbay, M.F., Kamisli, O., Ozcan, C. A Rare Presentation of Cranial Polyneuropathy Without Rash Caused by Varicella Zoster Virus. Med Arch. 2017 Aug;71,4,293-295.
9. Haanpa, M., Dastidar, P., Weinberg, A., Levin, M., Miettinen, A., Lapinlampi, A., et al. CSF and MRI findings in patients with acute herpes zoster. Neurology 1998;51,1405-11.
10. Worme, M., Chada, R., Lavalley, L. An unexpected case of Ramsay Hunt syndrome: case report and literature review. BMC Res Notes. 2013 Aug 28;6,,337.
11. Uscategui, T., Dorée, C., Chamberlain, I.J., Burton, M.J. Antiviral therapy for Ramsay hunt syndrome (herpes zoster oticus with facial palsy) in adults. Cochrane Database Syst Rev. 2008. pp. 1-2
12. De Ru, J.A., van Benthem, P.P. Combination therapy is preferable for patients with Ramsay hunt syndrome. Otol Neurotol. 2011;32,5, 852-855.



Figure 2. Deviation of angle of mouth on left side



Figure 3. Normal MRI brain (T2 image)

