Is it Possible Herpes Zoster Oticus Induced Diplopia?

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SUMMARY

Herpes zoster oticus (HZO) is a viral infection of the ear. Most patients with Herpes zoster oticus have diverse symptoms associated with dysfunction of the 7th and 8th cranial nerves. Ocular nervous system lesions can also be involved by herpes zoster, but isolated trochlear nerve lesion involvement is extremely rare. We discriminate other possible factors causing isolated trochlear nerve palsy and found a clear improvement of vertical diplopia after use of antiviral agent without systemic steroid. We report a case of delayed trochlear nerve palsy induced by Herpes zoster oticus in a 69-year-old male with a review of the literature.

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1. Introduction

A typical patient with Herpes zoster oticus (HZO) complaining of dizziness, will make most clinicians think that the main cause is dysfunction of the vestibulocochlear nerve involvement with HZO. However, when we found the aspect of dizziness is a different kind from peripheral vertigo, we thought the dizziness was derived from ocular nervous system lesions that can also be involved by HZO. What follows is our report on a rare case of a 69-year-old male who suffered from HZO without underlying diseases, and who subsequently developed dizziness because of diplopia induced by delayed trochlear nerve palsy.

2. Case report

A 69-year-old male without underlying diseases presented to hospital, with an acute onset of severe otalgia behind his right ear which began one day before admission. No remarkable physical findings were noted with the exception of auricular skin vesicles on the right ear. The patient was diagnosed with HZO in part, based on grouped tiny vesicles and crusted patch near the cymba concha of the right ear (Figure 1) accompanied by characteristic sharp or piercing pain on his right ear. Positive serology for varicella-zoster IgM antibody later corresponded with the diagnosis. On admission, the patient was initially given intravenous acyclovir (750 mg/day) and oral corticosteroid (30 mg/day) for 5 days, which in turn effectively resolved the pain and skin lesions. The patient was prescribed an antiviral medication famciclovir (750 mg/day) and corticosteroid (20 mg/day) for 7 days at discharge, with regular follow-up on an outpatient basis.

Two weeks after discharge, the patient subsequently developed substantial dizziness with double vision, nausea, gait disturbance,
VZV (Varicella zoster virus) usually occurs in elderly people and is caused by the reactivation of the virus. Isocoric pupils with symmetric and prompt reflexes to light were also observed. His right eye was typical of an underaction of the superior oblique muscle. Nine gaze ocular motility photography documented right hypertropia on gaze down and left (Figure 2) The Bielschowsky head tilt test revealed exotropia of 3 prism diopters (PD) and right hypertropia of 3 PD on right tilt and exotropia of 3 PD on left tilt. Alternate prism cover test showed right hypertropia of 3 PD and exotropia of 4 PD on rightward gaze. An isolated right trochlear nerve palsy was diagnosed for the patient. Thyroid function test for thyroid ophthalmopathy, routine complete blood cell count (CBC), HbA1c, blood pressure for diabetes mellitus (DM), hypertension induced microvasculopathy were performed to identify the responsible cause of the palsy. No remarkable findings were noted in laboratory tests including acetylcholine receptor (AchR) antibody for myasthenia gravis. Brain magnetic resonance imaging (MRI) showed no abnormalities.

The patient was given an antiviral agent famciclovir (750 mg/day), dexamethasone (100 mg/day), and diazepam (2 mg/day) for 7 days, presuming HZO was the underlying cause of the right trochlear nerve palsy. Patient’s presenting symptoms were markedly improved by the first week follow-up. Alternating prism cover test showed right hypertropia of 2 PD on left tilt. A follow-up examination 3 weeks later revealed no abnormalities, along with a complete resolution of diplopia and dizziness.

3. Discussion

HZO is viral disease by the reactivation of endogenous VZV (Varicella zoster virus). It usually occurs in elderly people and manifests as severe otalgia and associated cutaneous vesicular eruption, usually of the external canal and pinna. In HZO, 7th nerve and 8th nerve involvement is common. Isolated trochlear nerve palsy occurs rarely by HZO, particularly in the absence of Herpes zoster ophthalmicus. Considering the patient’s age, there were a number of other possible factors that might have caused the trochlear nerve palsy including microvasculopathy. However normal findings in HbA1c, thyroid function tests, acetylcholine receptor antibody test, and brain MRI led to our presumption that HZO was the responsible cause of the trochlear nerve palsy.

The pathogenic mechanism for extraocular nerve palsy in Herpes zoster ophthalmicus, some of possible hypotheses for pathogenesis include cytotoxic response to the viral infection, occlusive vasculitis derived from viral replication and circumscribed orbital myositis. However, rather than those hypotheses, we think it more likely that cranial nerves are interconnected neuroanatomically and functionally in the brainstem. In our case, we believe that the VZV, once reactivated, spread along the pathway of the brainstem reflexes connected with the affected cranial nerve in an anterograde direction, generating cranial polyneuropathy. Even though it is the nearest hypothesis to explain our patient’s symptoms, we need to consider only trochlear nerve involvement induced by HZO.

To the best of our knowledge, this is the first case of HZO related with delayed trochlear nerve palsy without symptoms of facial nerve palsy and vertigo. The only other case of HZO with delayed trochlear nerve palsy that has been reported in an article was accompanied by the symptoms of facial nerve palsy and vertigo. As most cases of HZO with cranial polyneuropathy have been reported and V, VII, VIII, IX and X are the ones most commonly affected, we are confident our case is very rare.

Through application of existing therapies for HZO, the anti-viral therapy and steroids, the patient’s otalgia and skin lesion showed improvement. However the vertical diplopia newly developed after 2 weeks. Upon eliminating other possible factors causing the isolated trochlear nerve palsy, we were able to observe a clear improvement in the patient after using an antiviral agent without systemic steroid. In some cases, the paralytic lesions improve spontaneously, but another prospective study found that administering acyclovir could prevent the ocular complication in Herpes zoster ophthalmicus. Steroids are known to help reduce post-herpetic neuralgia. However, since the patient’s otalgia has already decreased, we did not use steroids any more. So, in this case, we think the administration of antiviral agents also would be helpful for recovery from vertical diplopia.

We were able to find out through the case of a patient with HZO complaining of vertical diplopia, delayed trochlear nerve palsy could be caused by HZO. Vertical diplopia is not common to those patients with HZO and moreover it is an extremely rare case of isolated trochlear nerve palsy being involved with Herpes zoster infection. We suggest when physicians encounter patients who have symptoms of double vision and dizziness diagnosed HZO within few weeks without any underlying diseases, application of anti-viral agents could be very useful.

Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. For this type of study formal consent is not required.

Financial disclosure

No financial disclosure.
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