Herpes zoster induced internuclear ophthalmoplegia a rare case report

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Abstract:
Internuclear ophthalmoplegia (INO) is caused by lesion involving the medial longitudinal fasciculus. Patients with INO are usually asymptomatic but they may complain of diplopia and occasionally oscillopsia. The most common causes of INO are ischemia and demyelination. Various infectious etiologies such as tuberculosis, AIDS, brucellosis, cysticercosis and syphilis may produce INO. Herpes zoster producing INO is a very rare presentation. The possible pathogenic mechanism could be demyelination or microinfarction in the brainstem. Our patient presented with a recent history of herpes zoster followed by double vision. On examination, he had right internuclear ophthalmoplegia. Varicella zoster IgM antibody was positive and he recovered fully after treatment with Acyclovir and steroids.

Keyword: Internuclear ophthalmoplegia, medial longitudinal fasciculus, varicella zoster virus, herpes zoster virus

Introduction:
Internuclear ophthalmoplegia is caused by damage to the medial longitudinal fasciculus between the third and sixth cranial nerve nuclei which impairs the transmission of neural impulses to the ipsilateral medial rectus muscle. It produces failure in adduction of the ipsilateral medial rectus and nystagmus in the contralateral abducting eye. INO can be caused by various etiologies. We are reporting a patient with herpes zoster vasculopathy, a rare presentation of herpes zoster virus. Herpes zoster is a relatively rare etiology of Internuclear ophthalmoplegia, till now only two cases have been described in world literature.

Case vignette: Mr. M, a 56 year old male presented with complaints of painful vesicular eruptions in the left maxillary region for 2 weeks. He was diagnosed to have herpes zoster and treated with oral acyclovir by his family physician. Two weeks after the onset of the eruptions he developed diplopia. The diplopia worsened while looking at distant objects. At this stage, he got admitted in our hospital. He did not have any other co-morbid illness.
Past history of chicken pox infection was present at 10 years of age. On examination he was afebrile, healed herpetic scars were present in the left maxillary region. He had right internuclear ophthalmoplegia as evidenced by restriction of adduction in the right eye with nystagmus on abduction in the left eye. His vertical eye movements and convergence were normal. The pupil and fundus examination were normal. The rest of the neurological examination was normal. Neck stiffness was not present. On evaluation his complete blood count, renal function test, electrolytes, chest x-ray and electrocardiogram were normal. MRI brain with contrast was normal. Cerebro-spinal fluid analysis showed pleocytosis and elevated protein with normal sugar level. Serum varicella zoster IgM antibody was positive. He was treated with intravenous acyclovir and intravenous methylprednisolone followed by oral Prednisolone. He symptomatically improved within 2 months.

Discussion:
Our patient had right internuclear ophthalmoplegia with a recent history of herpes zoster infection involving V2 distribution of left trigeminal nerve. The patient possibly had involvement of the right medial longitudinal fasciculus without significant brainstem lesions in the MRI brain. Serum herpes zoster IgM antibody was positive in our patient suggestive of recent reactivation of latent varicella zoster virus. The exact pathogenic mechanism by which varicella zoster caused internuclear ophthalmoplegia could not be elucidated. The probable hypothesis are: i) Demyelination in the brainstem; ii) Micro infarcts in the brainstem due to inflammatory meningovasculitis. Herpes zoster particles spread along trigeminal afferent fibers and causes small vessel vasculopathy. Vasculitis due to herpes zoster virus is characterized by vascular wall necrosis, inflammatory exudates and development of giant cells in
medium and small sized vessels.\footnote{Romero Lopez J, Sarasa Corral JL, Yanez Bana RM, Pareja Grande JA. Granulomatous angitis of the basilar artery related to herpes zoster of the 7th cranial nerve. Neurologia 1990;5(3):98-101.} Pathogenesis of vasculitis remains obscure, but has been found to be associated with immune complexes, mechanical factors and infection by varicella zoster virus.\footnote{S Cazabon, K Over and J Butcher. Horner’s syndrome and sixth nerve palsy due to herpes zosterophthalmicus arteritis. Eye (2005) 19,222-224.} Only two such cases of internuclear ophthalmoplegia following herpes zoster infection have been reported in world literature. In both of the patients MRI Brain was normal as in our case. In the first case the authors suggested the pathologic process was demyelination in the brain stem.\footnote{Jiunn -Horng Kang, Jau -Der Ho, Yi -Hua Chen and Herng -Ching Lin. Increased risk of stroke after herpes zoster attack: A population based follow up study. Stroke 2009;40:3443-3448.} In the second case it was herpes zoster vasculopathy mimicking giant cell arteritis.\footnote{Carroll WM, Mastaglia FL. Optic neuropathy and ophthalmoplegia in herpes zoster oticus. Neurology 1979;29:726-9.} Varicella zoster vasculopathy following primary infection or reactivation may involve large vessels causing unifocal granulomatous arteritis and small vessels causing multifocal vasculopathy.\footnote{Lapresle J, Faux N. Atteinte unilaterale des IX, X, XI et XII au cours d’un zona cervical. Contribution à la pathologie vasculaire des nerfs crâniens. J Neurol Sci 1981;52:351-7.} Autopsy done on patients with varicella zoster vasculopathy showing multifocal infarcts in MRI reveals ovoid mixed necrotic and demyelinating lesions with small vessel vasculopathy and demyelination. Cowdry A internuclear viral inclusions are present in glial cells at the edge of the smaller ovoid, demyelinating lesions.\footnote{Garg RK, Kar AM, Jain AK. Herpes zoster ophthalmicus with complete external ophthalmoplegia. J Assoc Phys India. 1992;40:486-497.} Although our patient had a definite clinical history of recent herpes zoster supported by positive serum varicella zoster virus IgM antibody, MRI brain with MRA was normal. It is possible that microinfarcts or small foci of demyelination in the brainstem were not detected in MRI.

Note: Informed consent was obtained from our patient for publication of photos and information, which is available with the author.

**Conclusion:**
A spectrum of neurologic complications may follow herpes zoster infection such as motor neuropathies of the cranial and peripheral nervous system, encephalitis, meningencephalitis, meningoencephalitis, myelitis and Guillain -Barre syndrome. Our patient had presented internuclear ophthalmoplegia following herpes zoster infection, which is a very rare neurological manifestation of varicella zostervirus, which recovered completely with appropriate treatment. We wanted to highlight this case for its rarity.

**References:**


10) Varicella zoster virus vasculopathies: diverse clinical manifestations, laboratory features, pathogenesis, and treatment. The Lancet neurology 2009; 8:731-40