Oculomotor nerve palsy in a diabetic patient following an episode of Herpes Zoster Ophthalmicus

Diabetic patient with a recent episode of Herpes Zoster Ophthalmicus returns with new onset diplopia and unilateral ptosis. After extensive testing, he is diagnosed with an oculomotor nerve palsy likely secondary to Herpes Zoster.

I. Case Hx

45yo WM

CC: New onset diplopia with OD turned out and lid ptosis x1wk, headaches and neck pain x2wks

OHx: Mild NPDR, early senile cataracts, myopia

MHx: NIDDM x2yrs, dyslipidemia, PTSD, IBS, TBI with post-concussive headaches, and episode Herpes Zoster affecting V1, C2, and C3 dermatomes 2 weeks prior.

Meds: Amitriptyline, dicyclomine, gabapentin, gemfibrozil, glipizide, hydrocodone/acetaminophen, metformin, naproxen, prazosin, prednisone, sertraline, valacyclovir

Other: No known drug allergies. No tobacco, alcohol, or recreational drug use.

II. Pertinent findings

Clinical: Corrected DVA 20/25 OD and 20/20 OS. Pupils were equal, round, and reactive to light; no RAPD. In primary gaze, OD was out but not down with a moderate ptosis. Restricted elevation and adduction OD but able to move past the midline in the nasal and inferior nasal fields of gaze. Eye movements were full and unrestricted OS. Confrontations were full to finger counting OU. Anterior and posterior segment health were within normal limits.

Physical: Healing vesicular rash along right forehead and scalp, no Hutchinson’s sign.

Labs: HIV test and VDRL negative. CBC revealed low RBCs and mean platelet volume. Normal ESR and no organisms present on gram stain.

Radiology: Head CT w/o contrast revealed no mass or hemorrhage with well-preserved gray/white junctions. MRA brain negative for PCA aneurysm or any other vascular abnormality. Lumbar puncture had a normal opening pressure. The CSF was clear and colorless with elevated lymphocytes, protein, and glucose.

III. Differential diagnosis

Primary: Ophthalmoplegia is a known ocular sequelae of Herpes Zoster, with the oculomotor nerve most commonly being affected. On average, ophthalmoplegia occurs 2 months after the onset of rash.\(^1\)

Other: Posterior communicating artery aneurysm compresses the third nerve fibers, creating an oculomotor palsy with pupil involvement. The pupil may take days to become dilated, making a head CT or MRA essential to this diagnosis. Vascular disease such as diabetes, hypertension, and even giant cell arteritis may cause pupil-sparing third nerve palsies through ischemia. With ischemia, the peripheral pupillary fibers are spared while the inner muscular fibers are affected. Cavernous sinus syndrome caused by a mass lesion or thrombosis can cause an oculomotor palsy. However, additional involvement of cranial nerves IV, VI, V1, and the oculosympathetic pathway would be expected as all reside together in the cavernous sinus. Multiple sclerosis demyelination can cause isolated cranial nerve palsies. Although the trigeminal nerve is most frequently involved, MS should be a consideration in young adults with cranial nerve palsies. Cases of neurosyphilis and HIV have been reported to cause oculomotor palsies.\(^2\)

IV. Dx & discussion
Herpes Zoster Ophthalmicus is the reactivation of the varicella zoster virus in the V1 dermatome, the ophthalmic division of the trigeminal nerve. HZO ocular sequelae include keratitis, conjunctivitis, uveitis, and more rarely retinal necrosis and cranial nerve palsies. The exact mechanism between HZO and ophthalmoplegia is unknown, yet it has been hypothesized that axonal inflammation of the trigeminal nerve spreads through the cavernous sinus to the oculomotor nerve. Though the patient complained of head and neck pain, it was determined that this was from the H. Zoster as the timeline correlated with the onset of rash, and CT and MRA came back negative. Vascular disease is a more common cause of third nerve palsy than HZO. Yet, given the recent episode of HZO and absence of vascular abnormality on MRA, it was concluded that oculomotor involvement was most likely secondary to HZO rather than his fairly recent diagnosis of diabetes.

V. Tx, management

The patient was hospitalized and started on IV acyclovir and methylprednisone. Even though IV antivirals are typically reserved for secondary retinal necrosis, the neuro-ophthalmologist was concerned that there may be Herpes involvement of the patient’s central nervous system. He was taken off gabapentin and put on pregabalin for pain. He developed secondary hyperglycemia and was put on Insulin Lantus and Aspart. It was recommended that he patch his right eye to temporarily correct his diplopia. After 2 days on IV steroids, the patient was switched over to oral prednisone. He was then slowly tapered off prednisone and insulin. His IV acyclovir was discontinued after 10 days and he was discharged. At this time, the patient reported his diplopia, headaches, and neck pain were all improving. He was then referred back to optometry for Fresnel prism to correct his diplopia. The patient was found to still have a moderate ptosis with a 25PD right exotropia and 10PD right hypertropia on cover test. However, he reported single vision with just 7PD base down OD. He was seen on 2-3 week intervals and by his 7 week follow up, all Fresnel prism was removed from his glasses. Four months after the initial onset of his HZO-induced oculomotor palsy, he made a full recovery with no aberrant regeneration, ptosis, or diplopia, and only a slightly exophoria at distance and near. He corrected to 20/20 in each eye and no longer reported any neck pain or constant headaches.

VI. Conclusion

HZO-induced oculomotor palsies can be either pupil involved or sparing, thus require a thorough case history and extensive testing. With treatment, diabetic patients should be followed closely as they are at a greater risk of secondary hyperglycemia from steroid use.

References


