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Title:
Oculomotor Nerve Ophthalmoplegia with Pupillary Involvement Secondary to Herpes Zoster Ophthalmicus

Abstract:
Ocular motor cranial nerve palsies are rare manifestations of herpes zoster ophthalmicus (HZO). A patient with HZO presented with ipsilateral mydriasis a week after the cutaneous rash that eventually evolved into an oculomotor nerve palsy.

Outline:
I. Case History
   a. 47 year-old African female
   b. Presents to the eye clinic for a 7 day follow-up of a HZO with vesicular rash on the right side of her face, reports improvement in the rash, but has new symptoms of mild sensitivity to light OD
   c. Past ocular and medical histories are unremarkable; the patient is relatively new to the country and does not have a regular eye or medical care provider
   d. Current medications include valacyclovir 1000 mg PO TID, erythromycin 0.5% ophthalmic ung BID to cover the lesions on the forehead and face, and artificial tears QID OD
   e. The patient was seen 7 days prior in the emergency department (ED) with a chief complaint of itchy skin lesions on the right side of her face. The patient reported a right-sided headache 2 days prior to vesicular eruptions. The patient was diagnosed with herpes zoster ophthalmicus affecting the first (V1) and second (V2) divisions of the trigeminal nerve. A complete eye examination was performed by an ophthalmologist on-call during the ED visit and was negative for ocular manifestations of HZO. The patient was instructed to follow-up in the eye clinic in 1 week.

II. Pertinent findings
   a. Vesicular rash was improving with evidence of crusting and healing along the trigeminal dermatomes of V1 and V2 of the right side of the face, negative Hutchinson’s sign; otherwise patient appeared to be of good physical health
   b. Clinical examination
      i. Uncorrected vision was 20/40 OD without improvement with pinhole and 20/25 OS.
      ii. Pupils were round, dilated, and minimally reactive to light and accommodation OD; but round and reactive to light and accommodation OS; there was no afferent pupillary defect OU.
      iii. Extraocular motilities were full and smooth OU.
      iv. Anterior segment findings
         1. Lids and lashes: OD vesicles along upper eyelid; OS clear
2. Cornea: OD mild diffuse punctate epithelial staining secondary to mechanical irritation from vesicular lesions; OS clear

3. Anterior Chamber: OD deep with grade 2 to 3+ cells/1+ flare; OS deep and quiet

v. Posterior segment findings

1. Vitreous: clear OD and OS, no cells OU
2. Fundus: flat and intact 360 degrees OU, no snowballs or snowbanking OU

c. Laboratory studies

i. Rapid oral swab HIV screening yielded a positive result
ii. ELISA and Western Blot confirmation of HIV disease
iii. Viral load = 31,121 copies; CD4 cells/UL = 489

d. On further follow-up 5 days later

i. Vision stable OU
ii. Pupil sizes unchanged: OD still dilated at 6 mm, OS 3 mm
iii. No evidence of supersensitivity to diluted pilocarpine in dilated pupil in order to rule out Aide’s pupil
iv. New ptosis OD
v. Limitation of elevation OD
vi. Decreased corneal sensitivity OD

III. Differential Diagnosis

a. CN III palsy with pupillary involvement with the following possible etiologies:

i. Varicella zoster virus (VZV) infiltration or immunological response to the virus in the oculomotor nerve fibers and ciliary ganglion
ii. Aneurysm of posterior communicating artery
iii. Space-occupying tumor
iv. Acute ischemia

b. Pharmacological dilation

c. Aide’s tonic pupil

IV. Diagnosis and Discussion

a. Because an immunocompromised status was suspected in this young female patient from Africa who presented with herpes zoster ophthalmicus, HIV testing was performed. Patients with HIV have a 15 to 25 times increased risk for developing HZO.6

b. Same-day consultation with neuro-ophthalmology on the initial visit led to the diagnosis of HZO-associated pupillary mydriasis. The patient subsequently developed a partial CN III (superior branch) palsy on follow-up 5 days later.

c. There are <20 cases in the literature of various forms of ophthalmoplegia associated with HZO. The prevalence of ophthalmoplegia of some form secondary to HZO (either palsies of cranial nerve III, IV, VI and even complete ophthalmoplegia) is between 5%-31%. Cranial nerve III is the most-affected ocular motor nerve while cranial nerve VI is the least-affected. Theories for the development of isolated or multiple neuropathies of the cranial nerves innervating the eye include direct cytopathic effect of VZV and a host inflammatory response to VZV that can spread to the cavernous sinus and/or superior orbital fissure. Ocular motor nerve palsies may precede or appear subsequent to the cutaneous manifestation of HZO. Pupillary involvement is likely due to VZV infiltration of pupillary fibers at the ciliary ganglion. Fortunately, prognosis for ocular motor
nerve palsies associated with HZO is good with most cases recovering within a few months.

V. Treatment and Management
   a. Cutaneous HZO is improving: patient is taking valacyclovir 1000mg PO TID, erythromycin oph ung to cover the skin lesions BID
   b. HZO anterior uveitis OD: Pred Forte 1% Q2H OD
   c. HZO partial CN III palsy: continual frequent evaluation and follow-up with neuro-ophthalmology service for further ocular manifestations of HZO
   d. Refer patient for HIV counseling to obtain HAART/anti-viral medications; request for HIV testing for her children

VI. Conclusion
   a. Suspect immunocompromised status in young patients who present with herpes zoster
   b. Important to check pupils and extraocular motilities for evidence of ocular motor cranial nerve involvement in patients with HZO
   c. A negative Hutchinson’s sign does not rule out the risk of ocular complications from HZO
   d. VZV can be a great masquerader. Eye care professionals need to be aware that ophthalmoplegia is a rare complication of HZO and can involve cranial nerves III, IV and VI separately or together simultaneously.

References: