Herpes Zoster Ophthalmicus with Third Nerve Palsy

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Introduction

Herpes zoster is a recurrent infection of the varicella-zoster virus. When it infects the periorbital region, the virus often involves the ocular structures, and may lead to more disastrous sequelae, including blindness. Despite advancements, including immunization and antiviral therapy, zoster ophthalmicus persists in the population.[1] Therapy mitigates, but does not obviate, many of the complications of infection.[2] Our paper describes a patient with herpes zoster ophthalmicus that presented from the community.

Case Presentation

The patient is an 85 year-old male with a past medical history of HTN, BPH, and open-angle glaucoma who was in his usual state of health until approximately two weeks prior to admission, when he began having episodes of nausea and emesis. A few days later, the nausea and emesis resolved, and he developed rhinorrhea. The next day, he noticed a series of "scabby," non-painful sores on the left side of his nose. At that time he also noted an intermittent stabbing headache in the area of his left eye and a yellowish, purulent discharge from his left eye with left eye swelling. He was unable to voluntarily open his left eyelid. Vision was preserved in the left eye. Five days prior to presentation he saw his primary care physician who prescribed a regimen of antihistamine, levofloxacin, and ketorolac eye drops. However, his nose lesions and eye swelling worsened, and he then visited an ophthalmologist on the day of presentation to the hospital. The ophthalmologist's exam revealed elevated intraocular pressure and possible anterior chamber involvement.

On admission he reported no facial trauma and no recent sick contacts. He also denied any headache or eye pain in the last few days. He did report continuing eye discharge and inability to voluntarily open his left eye. Further review of the patient's past medical history was significant for a history of rectal carcinoma, prostate cancer, hyperlipidemia, bronchitis, chickenpox as a child, and prior basal cell carcinoma of his left eye. Surgical history included a skin cancer removal and "prostate surgery." Patient reported no known medication allergies. His outpatient medications were felodipine, simvastatin, and finasteride. He was not taking any over-the-counter or herbal medications. Social history was negative for tobacco, alcohol, or substance abuse. He lives with his wife with no pets. He is an army veteran of World War II and is currently retired. He reported no recent travel or occupational exposures. On review of systems, he reported no fever, chills, genitourinary, or musculoskeletal complaints. On exam, he appeared to be in no acute distress. His temperature was 99.8° F, his heart rate 80 beats per minute, his blood pressure 142/76 mmHg, and respiratory rate 20. He appeared comfortable. Head was atraumatic, however his left periorbital region was swollen and mildly erythematous, and the patient, despite encouragement, was unable to open his eyelid voluntarily. Upon raising the eyelid on exam, the patient had purulent discharge from the medial canthus, the sclera was minimally injected, and the left pupil was 3.5-4 mm and non-reactive to light. The right pupil was 2.5- 3 mm and reactive to light. Visual acuity was preserved bilaterally. Extraocular movements of the right eye were intact, while the left eye could abduct, but could not adduct, elevate, or move downward. The patient had lesions on the left forehead, eyelid, and tip of the nose that were scab-like. They were not painful, nor were they actively bleeding (Figure 1). The remainder of the patient's physical exam was unremarkable. Admission laboratory work showed that the patient had no leukocytosis or anemia, and aside from a serum potassium of 3.1, laboratory evaluation was normal.

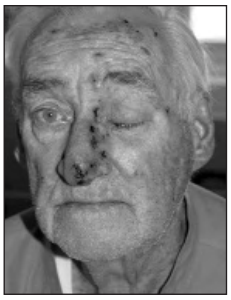

Figure 1.

The patient had already failed outpatient presumptive therapy for bacterial conjunctivitis. The dermatomal distribution of his lesions, corresponding to more than one branch of the ophthalmic division of the trigeminal nerve, the cranial nerve III palsy, along with the acuity and the time course of the illness, were consistent with herpes zoster ophthalmicus infection.

Upon admission, brimonidine ophthalmic drops were started to decrease the intraocular pressure in the setting of open-angle glaucoma. Anterior chamber involvement was noted, for which steroid eye drops were started. Systemic antiviral therapy with acyclovir was also initiated. MRI/MRA of the brain was performed and ruled out aneurysm as an etiology of CN III palsy. The day after admission the patient was again seen by ophthalmology, and artificial tears and timolol were added to the patient's medication regimen for his increased intraocular pressure and history of open-angle glaucoma. The patient did well on the above prescribed course, and was discharged to home with outpatient follow-up four days after initial presentation. Our last report of follow-up was sixteen days after initial presentation to the VAMC, when the patient was noted to have slow improvement. At that time, he was able to raise his eyelid and maintain it voluntarily for increasing periods of time. His visual acuity remained unaffected by the Herpes Zoster Ophthalmicus, and the edema around his eye was noted to be significantly improved at that time.

Discussion

Herpes zoster is an extremely common neurological affliction, which often occurs at least one time in a person's life. Estimates have placed the incidence at approximately 2.2-7.1 cases per 1000 person-years.[4] Herpes zoster generally occurs in the elderly, and is caused by reactivation of the varicella-zoster virus. Patients are initially infected by age ten, and the presentation is commonly recognized as chickenpox. The virus then becomes latent in sensory ganglia, and later reactivates, spreading via spinal or cranial nerves to a dermatome. Patients thereafter present with the typical unilateral exanthem, and concomitant acute pain due to inflammation of sensory neurons and skin insults.[5]

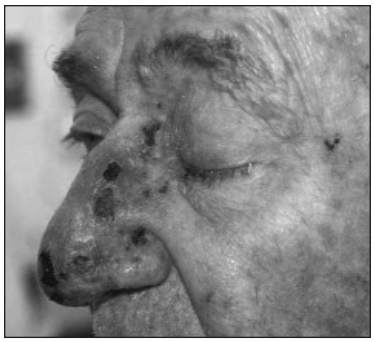

Figure 2- Hutchinson's sign, as seen in our patient, involving the nasociliary branch of the ophthalmic division of trigeminal nerve.

Reactivation is often seen in the setting of diminished cell-mediated immunity, common to the elderly as well as the immuno-compromised patient. Prospective studies have shown that the most common location of zoster reactivation is the ophthalmic nerve.[4] In fact, ophthalmic herpes zoster is seen in 10-20% of all zoster case.[5] Often, affected patients have severe and persistent pain. Other features include keratitis, uveitis, and optic neuritis of the affected eye, all of which require immediate attention to prevent vision impairment. Decreased corneal sensitivity may result as well, leading to dry eyes, and eventual corneal ulceration.[5]

In the early stages of herpes zoster ophthalmicus, patients report malaise, pain, pruritis, low-grade fever, and photophobia. Skin hypersensitivity occurs on the forehead, followed by erythematous macules, which evolve to papules and vesicles in the affected dermatome. Generally the skin rash precedes ocular lesions by several days. Periorbital edema is seen early in the course of the disease. Conjunctivitis, episcleritis, and corneal epithelial defects are all commonly observed phenomena. As with our patient, anterior chamber involvement is observed, with the release of viral antigens causing a mild uveitis with concomitant elevation in intraocular pressure.[5]

And, while the frontal branch of the ophthalmic division of the trigeminal nerve almost always is involved, involvement of the nasociliary branch is quite rare. The appearance of skin lesions at the side of the nose, known as Hutchinson's sign, has been considered a prognostic feature for ocular inflammation in patients with acute herpes zoster ophthalmicus.[4] (See Figure 2) Our patient presented with complete ptosis and essentially paralysis of the oculomotor nerve, which is particularly rare in herpes zoster ophthalmicus. An extensive review of the literature returned only one recent case review found in French literature, reporting two cases of oculomotor paralysis.[3] Furthermore, it has been postulated that involvement of cranial nerves other than the trigeminal nerve occurs by secondary vasculitis in the orbital apex, resulting in diplopia.[5]

Early and immediate therapy with oral acyclovir is the cornerstone of treatment of this disorder, and has been proven to significantly decrease the incidence of negative sequelae of eye disorders. Patients should be evaluated within one week of starting acyclovir. Patients who present with Hutchinson's sign or visual complaints merit referral to an ophthalmologist.[5] ■

References

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Fool

They say God protects fools and the innocent
They say God protects the innocent and the souls of children
Unfortunately the years of childhood has passed me
I doubt that God looks upon me as innocent
So I pray to be the fool

God protects the fools
The fools that don't know any better
Those who live by folly
Those who are compared to, throughout his scripture
Why does God protect those who he mocks throughout his book

The fool in the book of wisdom is the enemy
The fool is the ass in Psalms
The fool in Sirach is the Anti
The fool is what you don't want to be
The fool is what God tells you not to be
But he protects them
He understands them
He created them

If that is what I need in order to have his shield
I pray to be the fool
I pray to be protected
Maybe that is why ignorance is bliss at times
Maybe that is why they say that
Maybe a fool said that
And see how his words still last?

But maybe he didn't last
Because with that statement
I think he lost his ignorance
Because he knew it was bliss
His ignorance, no longer folly, but purpose
And I don't even know his name
But was that fool protected?
I hope he was
For my sake....
For our sake.

Marshall Fleurant, MD