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Herpes Zoster Ophthalmicus Associated with Ipsilateral Bell's Palsy

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Abstract

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Background: Herpes zoster is caused by reactivation of the varicella zoster virus with spread along the dorsal root ganglion and sensory nerve to a dermatome. In herpes zoster ophthalmicus (HZO), involvement of the ophthalmic (V_1) branch of the trigeminal nerve results in ocular sequelae and often presents with a characteristic pseudodendrite. Reactivation within cranial nerve VII can lead to the neurologic complication of facial nerve palsy on the affected side. **Case Report:** This case report describes a patient diagnosed with HZO and subsequent same-sided facial nerve palsy (Bell's palsy) and discusses the potential link between the two conditions. **Conclusion:** Treatment for HZO and Bell's palsy is fluid and conservative treatment must be weighed against issues with polypharmacy and the severity of the disease. The prognosis of HZO and Bell's palsy is dependent on how well symptoms are managed, including close observation beyond resolution.

Keywords

herpes zoster, herpes zoster ophthalmicus, facial nerve palsy, Bell's palsy

INTRODUCTION:

The human varicella zoster virus (VZV) primarily infects individuals in what is commonly known as "chicken pox."[1] The virus then remains dormant within the dorsal root or neurosensory ganglion. Reactivation of the herpes virus, triggered by advanced age and immune stress, results in shingles varicella, or herpes zoster, occurring along the dermatome innervated by the ganglion.[2] Herpes zoster (HZ) manifests as unilateral pain in a dermatomal distribution with a vesicular rash that does not cross the midline.[3] It is associated with a prodrome of fever, malaise, headache, and dermatomal pain.[2] Shingles appears most commonly as a rash along the body's thoracic dermatomes.[4] Ocular manifestations of shingles include many anterior and posterior segment eye disorders. Hutchinson's sign, while not required in the diagnosis of herpes zoster ophthalmicus (HZO), presents when herpetic lesions involve the side, tip, or root of the nose, and is a strong indication of ocular involvement.[2]

Less commonly, viral disorders like the herpes zoster virus have been noted to cause Bell's palsy.[5] This case describes a patient diagnosed with HZO and subsequent facial nerve palsy (Bell's palsy). The link between the two conditions as well as the importance of close follow-up for identifying potentially long-term complications is also discussed.

CASE REPORT:

Initial Visit:

A 73-year-old immunocompetent African American female presented with complaints of a red right eye with associated pain (rated 8 out of 10 on a pain scale) and tearing. She presented to the emergency room the previous day and was diagnosed with shingles. There, she was prescribed oral valacyclovir 1g three times per day for one-week, oral cephalexin 500 mg every 8 hours for one week, and oral acetaminophen-hydrocodone 325-5 mg every 6 hours or as needed for pain. She reported excellent compliance with all medications. The patient's medical history was remarkable for hypertension and hypercholesterolemia, and she was allergic to iodine. She was oriented to person, place, and time, and her mood was appropriate.

Entering distance visual acuities with correction were OD 20/40, no improvement with pinhole (NIPH), OS 20/20. Confrontation visual fields were full to finger counting in both eyes.

Extraocular muscle testing showed full range of motion without pain or diplopia in each eye. Pupils were equal, round, and reactive to light with no afferent pupillary defect noted. Slit lamp biomicroscopy of the right eye was remarkable for a unilateral vesicular rash along the V1 dermatome without the presence of Hutchinson's sign. Also noted was eyelid scurf, moderate upper eyelid edema, 3+ conjunctival injection of the right eye (on a scale of 1 to 4), and dense superficial punctate keratitis with pseudodendritic staining present inferiorly. The anterior chamber was void of any inflammatory reaction, and the iris was flat and normal. Evaluation of the left eye was unremarkable. Intraocular pressure measurements were OD 13 mm Hg, OS 15 mm Hg at 3:09 pm with a Tonopen (95% confidence interval). Dilated evaluation was deferred, but nuclear and inferior cortical cataracts were noted in both eyes.

The patient was diagnosed with herpes zoster keratoconjunctivitis in the right eye. Since the patient was started on medications for shingles by an outside provider, she was educated on the ocular findings and told to continue oral valacyclovir, cephalexin and acetaminophen-hydrocodone. Preservative-free artificial tears were added at four time a day (QID) dosing in the right eye with Celluvisc® at bedtime for symptom relief. She was told to return in one week for follow-up.

The differential diagnoses for this case included:

- **Herpes Zoster Ophthalmicus (Keratoconjunctivitis)**: because the patient came in with a confirmed diagnosis of shingles and presented with a characteristic pseudodendrite, herpes zoster ophthalmicus is the assumed diagnosis.
- **Herpes Simplex Keratitis (HSK)**: pseudodendrites can resemble HSK at first, but during slit lamp evaluation, this patient lacked the presence of terminal bulbs characteristic of HSK, so this diagnosis was ruled out.
- **Superficial Punctate Keratitis (SPK)**: coarse patches of SPK can orient linearly, resembling a branch-like dendrite. While this patient had a dense area of noted SPK at her initial visit, it was in addition to a pseudodendrite that was consistent with the diagnosis of shingles.

• **Ramsay-Hunt Syndrome**: this condition may be misdiagnosed as Bell's palsy. It presents as facial paralysis with accompanying herpes zoster oticus, and is associated with ear pain, changes in or loss of taste, and vertigo.[2] Further cranial nerve testing is warranted to rule this diagnosis out.

Follow-up Visit #1:

At the one-week follow-up appointment, the patient reported an overall improvement in symptoms, but pain was still rated a 6 out of 10. She reported vision was stable and that the vesicles were improving. She used all medications as prescribed in the emergency room for the full week and was no longer on any medications.

Entering visual acuity with correction was stable in both eyes as was entrance testing. While the adnexal edema and vesicular rash showed improvement, a new lagophthalmos and a mild ectropion of the right lower eyelid were present. The pseudodendrite was resolved but there remained 4+ diffuse superficial punctate keratitis (SPK) below the visual axis with diffuse corneal edema. The anterior chamber revealed trace cells in the right eye. An evaluation of the left eye was again unremarkable. Intraocular pressure measurement was OD 12 mm Hg, OS 14 mm Hg with a Tonopen at 2:06pm. Dilated fundus evaluation was remarkable for a posterior vitreous detachment and 1+ nuclear sclerotic cataract with 1+ inferior anterior cortical cataract in the right eye. The optic nerve, macula, vessels, and periphery were otherwise normal in both eyes. Cranial nerve testing was remarkable for an inability to move her forehead and drooping of her mouth and eyelid, revealing upper and lower CNVII weakness along the right side. She also exhibited an increased sensation in all dermatomes of CNV along the right side. Testing of CNIII-XII was otherwise intact and equal between two sides of her face. Specifically, the patient denied having ear pain, vertigo, or difficulty hearing.

The patient was educated that though the pseudodendrite had resolved, there was persistent superficial punctate keratitis. Additionally, a post-herpetic Bell's palsy with upper and lower motor neuron involvement was diagnosed and managed conservatively with preservative-free Celluvisc® every hour and lubricating ointment at bedtime to the right eye for symptom relief. Oral valacyclovir 1g was prescribed by the attending optometrist for use three times per day (TID) for one week to aid with pain management.

Follow-up Visit #2:

At the two-week follow-up appointment, the patient reported good compliance with oral valacyclovir. She visited her primary care physician two days prior who prescribed oral gabapentin 600 mg TID and her pain was much improved, rated a 4 out of 10. However, she reported persistent tearing and redness. Vision was stable in both eyes. Slit lamp biomicroscopy of the right eye was remarkable for a persistent, incomplete blink with lagophthalmos. Her eyelid vesicles were crusting over and improving, and her eyelid edema resolved. The diffuse superficial punctate keratitis (SPK) below the visual axis was slightly improved from the previous visit. The anterior chamber evaluation revealed resolution of inflammation without cell or flare. An evaluation of the left eye was unremarkable. Intraocular pressure measurements were OD 12 mm Hg, OS 13 mm Hg by applanation at 1:26pm.

With the patient's HZO resolving and right CNVII palsy with Bell's phenomenon stable, the patient was to complete the valacyclovir and gabapentin as prescribed. Preservative-free artificial tears (Celluvisc®) were reduced to QID, and lubricating ointment was continued to the right eye at bedtime.

Follow-up Visit #3:

The patient returned as directed; she completed all oral medications with excellent compliance and was using preservative-free Celluvisc® 3-4 times per day in both eyes. She reported her symptoms had improved since the last visit, and denied any ocular irritation, photophobia, pain, or redness. Her vision was stable from the previous visit in both eyes. An evaluation of the right eye revealed a persistent incomplete blink with lagophthalmos and diffuse SPK inferiorly. An evaluation of the left eye was again unremarkable. Intraocular pressure measurement was OD 10 mm Hg, OS 11 mm Hg by applanation.

Due to the incomplete blink likely causing decreased vision, the patient was told to continue use of Celluvisc®, and lid taping with lubricating ointment was recommended for the right eye at bedtime. She was told to return in one month for follow-up.

Follow-up Visits #4-6:

Due to the COVID-19 pandemic, three visits were performed by the provider via telehealth phone calls in a three-week period. The patient reported excellent compliance with Celluvisc® QID in the right eye with significant improvement in her vision. She had not used any ointment or performed lid taping as recommended at the last visit. She was educated to contact the urgent care department in the event her vision worsened, or symptoms increased.

Follow-up Visit #7:

The patient returned to the office three months after the initial presentation and reported that her eye comfort and vision had improved since the last in-person visit. She denied any redness, pain, or light sensitivity. She remained compliant with Celluvisc® three to four times per day in the right eye but did not use ointment or tape her lids at bedtime.

Entering visual acuities with correction were improved by one line at OD 20/30, PHNI, OS 20/20. Slit lamp biomicroscopy of the right eye was largely improved and remarkable for complete blink, mild orbicularis muscle weakness and trace SPK inferiorly. The evaluation of the left eye remained unremarkable.

The patient was educated that findings for both facial palsy and herpes zoster keratoconjunctivitis were improved. She was advised to continue use of Celluvisc® QID in the right eye and told to return in 2-3 months for follow-up.

Follow-up Visit #8:

The patient was lost to follow-up for eight months after which she returned for a comprehensive eye examination, reporting that her vision OD was stable and remained slightly blurry, but denied any redness, pain, or light sensitivity. She reported using Celluvisc® three to four times per day in each eye. The entering visual acuities with correction were OD 20/40-2, PHNI, OS 20/20.

Entrance testing was unremarkable in both eyes and her refractive error was unchanged from previous current spectacle correction: OD +0.25 -1.00 x 057 VA 20/40, OS +0.25 -0.50 x 108 VA 20/20, add +2.50. Slit lamp biomicroscopy of the right eye was remarkable for a complete blink but a weak orbicularis muscle was noted, along with 2-3+ diffuse SPK inferiorly. The anterior chamber was void of

any inflammatory reaction, and the iris was flat and normal. The left eye remained unremarkable. Intraocular pressure measurements were OD 13 mm Hg, OS 14 mm Hg by applanation. Following dilation, lens evaluation was remarkable for 2+ nuclear and peripheral anterior cortical changes, as well as 2+ posterior subcapsular cataract within the visual axis in the right eye, and 1+ nuclear and peripheral anterior cortical changes in the left eye. Super pinhole testing revealed potential acuity improvement of 20/25+ in the right eye. Fundus examination was otherwise remarkable for posterior vitreous detachment in both eyes.

The patient was educated that her decreased visual acuity in the right was due to a combination of the cataract and the residual keratitis secondary to a weak orbicularis muscle. While herpes zoster was a contributor, the corneal findings at this point were more likely due to exposure from the Bell's palsy and incomplete lid closure. She expressed interest in a surgical consultation for the cataract and was instructed to increase use of Celluvisc® to a minimum of QID.

DISCUSSION:

HERPES ZOSTER OPHTHALMICUS

Herpes zoster typically presents with a prodrome of malaise, headache, and fever 1-7 days prior to the onset of skin lesions.[2] Lesions are identified as red, unilateral, painful maculopapular eruptions of skin vesicles respecting the vertical midline. The incidence of herpes zoster in the United States is highest among the elderly and immunocompromised,[6] however most patients with herpes zoster have no underlying systemic conditions.[4] Age is the most common predisposing factor for herpes zoster due to changes in T cells and decreased varicella zoster virus antibody which allow for reactivation of the latent virus. Herpes zoster affects 20-30% of the population and occurs most commonly between the ages of 50 and 59.7 While others assert there is no predilection for any race or gender, a study by Insinga et al. found a slightly higher rate of herpes zoster in females regardless of age, suggesting differing immune responses to latent viruses between males and females.[6] Among individuals with the herpes zoster virus, the lifetime risk for developing HZO is 10-20%.[2]

Upon diagnosis of HZ, it is critical to perform a thorough eye examination and rule out vision-threatening ocular complications. Herpes zoster ophthalmicus

results from involvement of the ophthalmic branch of the trigeminal nerve (V1) which is divided into frontal, lacrimal, and nasociliary areas that innervate the eyelids. The other two main divisions of the trigeminal nerve are the maxillary (V2) and mandibular (V3) branches (Figure 1). The supraorbital and supratrochlear divisions of the frontal branch, innervates the forehead and upper lid.[2] The nasociliary nerve, the only subdivision of V1 that innervates the eye, also supplies the tip of the nose. When the nasociliary nerve's external nasal and infratrochlear branches are affected, Hutchinson's sign presents and signals a greater likelihood of sight-threatening ocular complications. Its absence still indicates a 34% chance of ocular involvement. Ocular sequelae can occur in 50-85% of such cases immediately, within a few weeks, or what seems idiopathically months to years following the initial vesicular eruption.[4]

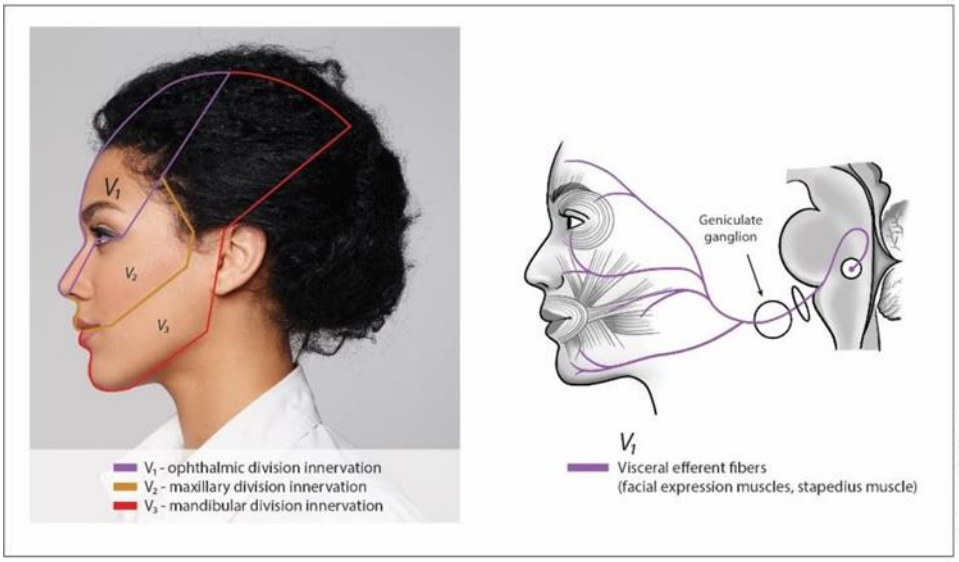

Figure 1. (left) The area affected by the ophthalmic branch of the trigeminal nerve (V1) stretches from the forehead to the tip of the nose and respects the vertical midline. (right) Herpes zoster infection can spread beyond the confines of the geniculate ganglion. Facial nerve inflammation may occur secondary to motor fiber involvement as it passes through the geniculate ganglion, possibly triggering Bell's palsy following herpes zoster. Try to move this closer to where it is referenced

Complications of lid involvement may result in lid ulceration, scarring, pitting, pigmentation, trichiasis, and lid retraction.[8] Corneal involvement can occur in up to 65% of patients, including punctate epithelial keratitis 1-2 days following initial presentation, pseudodendrites (mildly stained with Rose Bengal or sodium fluorescein) 4-6 days after, anterior stromal opacities, and neurotrophic keratitis. Corneas following HZO and those with chronic exposure are more at-risk development of neurotrophic keratitis. This patient is at particularly high risk given the combination of HZO and exposure with Bell's palsy. Herpes zoster dendrites, while self-limiting in nature, change daily. Herpes zoster can cause either a non- granulomatous or granulomatous chronic anterior uveitis with or without corneal disease. Fundus involvement can include acute retinal necrosis or peripheral outer retinal necrosis, both indications of more severe immunocompromised disease.[9] A recent study by Niederer et al. found that approximately one in 10 individuals with HZO may develop moderate or severe vision loss, primarily due to corneal scarring. Severe permanent vision loss was linked to uveitis, immunosuppression, and older age.[10] This patient, though older, showed good potential acuity following cataract extraction and fortunately did not show any permanent signs of decreased vision due to HZO.

BELL'S PALSY:

Facial nerve palsy involves paralysis and weakness of cranial nerve VII (CNVII) which provides sensory afferent and motor efferent fibers. Bell's palsy, or acute idiopathic unilateral facial nerve palsy, is the most common manifestation of CNVII palsy.[11] Typically, there is an acute onset of lower motor neuron facial weakness, often accompanied by loss of facial expression and oral droop. The incidence of Bell's palsy is between 11.5 to 53.3 per 100,000 and highest among the 15-to-45-year-old age demographic. Risk factors for Bell's palsy include pregnancy, obesity, hypertension, and diabetes.[12]

While there are reports of suspected viral cause, there is no clear etiology, and Bell's palsy remains a diagnosis of exclusion.[12] The facial nerve courses through the facial canal within the temporal bone and is susceptible to compression; nerve edema and inflammation in this tight space ultimately results in injury. Injury to the facial nerve in cases of Bell's palsy occurs at or near the geniculate ganglion.[12] Depending on the lesion's location, the degree of paralysis varies. Because the facial nerve supplies both motor and sensory neurons, facial expression, salivation, taste, and ocular tearing can be affected. Patients with

Bell's palsy may express dryness of the eye or mouth, loss of taste, difficulty hearing, and droopiness of their eye and/or mouth.[11] Facial weakness associated with Bell's palsy must be identified as due to an upper or lower motor neuron lesion.[12] Mobility of the forehead muscles indicate an upper motor neuron lesion, while lower motor neuron lesions are signaled by facial weakness on the entire side of the face.[11] This patient was asked to close her eyes tightly but was unable, a sign of Bells phenomenon. She was asked to "make a face like a blowfish" and pucker her lips, which showed weakness on her right side. Since she was unable to move her forehead due to a facial paralysis, she was determined to have both upper and lower motor neuron disorders. This is also known as a peripheral lesion, where the CNVII palsy inhibits movement of the forehead.

The varicella zoster virus can also present as vesicular eruptions with facial palsy and ear involvement, known as Ramsay Hunt Syndrome (RHS) or herpes zoster oticus. Likewise, it can also present as zoster sine herpete (ZSH), a condition where facial palsies caused by VZV present without vesicular eruptions.[5] It is important to rule out both RHS and ZSH when performing an ocular exam. In this case the patient presented with skin lesions, and later presented with a facial nerve palsy, which rules out ZSH. Her cranial nerve testing also showed that CNVIII was unaffected, ruling out RHS.

CONNECTING THE TWO:

Ocular involvement in HZO may be the result of direct viral invasion (keratitis), an inflammatory response (uveitis), nerve damage (CN involvement), or vasculitis (scleritis, iris atrophy, or retinal necrosis). It can be acute, chronic, or relapsing in nature.[10] The fact that this patient's facial nerve palsy presented after the onset of herpes zoster suggests a possible connection between the two conditions.

In a literature review, the association between HZO and facial paralysis is a relatively infrequent occurrence. Hewlett associates a higher frequency of facial and neck paralysis with herpes zoster and highlights the separation between motor nerves affected and sensory nerves. Among groups of zoster-related facial paralysis, he identified 12 cases in which facial paralysis occurred a few hours to a few weeks following episodes of herpes zoster affecting CNV, the majority of which were ipsilateral.[13] Paralysis is usually situated on the same side as the zoster, though some cases have been found to occur contralaterally.[14]

One possible etiology connecting HZO and Bell's palsy is that inflammation spreads from the affected ganglion along the trigeminal nerve, then goes on to affect the facial nerve which results in paralysis.[15] Another theory connecting the two conditions is that the inflammation spreads to the facial nerve by way of the geniculate ganglion and paralyzes muscles innervated by it. In 1948, Thomas Parkinson made the connection between the geniculate ganglion and the varicella zoster virus, stating that a facial palsy may occur secondary to involvement of the lower motor neurons as they transit through the geniculate ganglion.[15] His theory is consistent with this case where zoster infection preceded facial paralysis.

Another occurrence of ipsilateral Bell's palsy after HZO was discovered by Wakil et al., who found that simultaneous effects in both facial and trigeminal nerves within the same clinical course can be explained by proximity between the two cranial nerves as they are found to communicate at three different locations.[16] The vidian nerve branches off the facial nerve and communicates with the maxillary nerve by way of the sphenopalatine ganglion. The chorda tympani of the facial nerve join a branch of the mandibular nerve called the lingual nerve to innervate the anterior tongue. Lastly, the auriculotemporal branch of the mandibular nerve communicates with the facial nerve as it crosses the parotid gland.[16] Ambrose Earl Edgerton describes a clinical picture between HZO and Bell's palsy by drawing anatomic similarities between the trigeminal and facial nerves. He suggests that as the facial nerve's sensory root enters the geniculate ganglion, some fibers extend beyond and become involved with motor fibers.[14] The cause of paralysis that accompanies HZO may not be clear, but the common link is inflammatory in nature and involves the geniculate ganglion and its proximity to the trigeminal and facial nerves.

TREATMENT/MANAGEMENT:

Whether coincidence or viral in etiology, HZO associated with a subsequent Bell's palsy is uncommon, and treatment should be addressed for each separate diagnosis. Current guidelines recommend that patients start an oral antiviral within 72 hours of the onset of herpetic lesions to decrease the severity in

and duration of the disease.[17] This includes post-herpetic neuralgia (PHN) as well as other ocular complications through the inhibition of viral replication. Therapy includes acyclovir (800 mg orally 5 times daily for 7 days), famciclovir (500 mg orally TID for 7 days) or valacyclovir (1000 mg orally TID for 7 days).[9,18] These drugs reduce pain, decrease the spread of zoster and accelerate the clearance of the virus from vesicles. Zirgan® has been shown to accelerate pseudodendrite resolution. Aggarwal and associates found topical 0.015% ganciclovir gel to be an effective treatment for pseudodendrites and suggests its use in conjunction with other oral or topical antivirals.[19] Patients are considered infectious until skin lesions have dried and crusted over (usually 5-7 days after onset); this infectious period is likely shortened if the patient is taking a full course of oral antiviral medication. Initiation of treatment outside of the initial onset of symptoms can still yield successful outcomes but the probability of a complete recovery decreases with time.[18]

While all three medications are well tolerated and similar in their safety and efficacy profiles, the newer famciclovir and valacyclovir are more frequently prescribed than acyclovir due to patients' improved compliance with fewer doses throughout the day.[17] They also have improved activity against the virus and are available generically. While antivirals are typically administered for between 7 and 14 days, there is no guideline established for its duration.[12] When the patient returned for her first follow-up and still reported a 6 out of 10 pain, treatment with valacyclovir was continued for one more week. Topical antibiotic ointments can also be prescribed to prevent crusting and reduce inflammation at the lesion sites.

Punctate keratitis and pseudodendrites represent viral replication at the level of the corneal epithelium.[4] Pseudodendrites are elevated, fine branching lesions with tapered ends without terminal end bulbs and typically treated with topical antivirals like acyclovir; ophthalmic acyclovir is not available for use in the United States but has shown promise.[4,8] Pseudodendrites have a self-limited clinical course and the most appropriate management is to use lubricating agents and preservative-free artificial tears.[12] These lesions are transient and typically resolve within 1-2 weeks of the initial presentation, however debridement of pseudodendrites may minimize epithelial damage. Topical corticosteroids should be reserved for cases of inflammatory corneal involvement such as interstitial keratitis or immune-specific complications such as interstitial keratitis or immune-specific complications such as anterior stromal infiltrates.[4] The patient's corneal involvement was limited to the epithelium and thus managed conservatively with complete resolution. Her ocular involvement also included a

mild anterior chamber reaction during her first follow-up visit which could have been treated with topical steroids. However, her inflammation resolved without initiating steroid therapy.

Patients with Bell's palsy show recovery without intervention within a few weeks after the onset of symptoms, and 70% report complete recovery within six months.[12] Bell's palsy management is geared toward minimizing risk for exposure keratitis, corneal abrasions, and corneal ulcers. Palliative treatment of Bell's palsy associated with HZO, including the use of lubricating ointment and artificial tears, results in an overall favorable prognosis.[11,16] Taping the eyelid closed helps in cases of SPK resulting from paralytic ectropion and lagophthalmos. Additionally, topical steroid use has been shown to aid Bell's palsy in recovery of facial motor function and minimizing the risk for complications such as motor synkinesis and autonomic dysfunction.[16,20]

Oral treatment with prednisolone and antivirals has been recommended to treat Bell's palsy.[20] A study performed by Sullivan et al. compared early treatment of Bell's palsy with oral prednisolone alone, oral acyclovir alone, and oral prednisolone with acyclovir. They showed that patients recovered favorably without treatment, but early treatment with prednisolone (dosed 25 mg by mouth [po] BID) increased the rate of complete recovery. This suggests that, by inhibiting and decreasing the immune response, prednisolone sped up the rate of edema reduction surrounding the facial nerve within the facial canal.[20] Other treatments including lateral tarsorrhaphy, botulinum toxin (Botox) or use of gold weight lid loading can be effective options for patients with longstanding facial paralysis.[4,11]

Addressing pain is critical as PHN is a complication of herpes zoster that can be difficult to manage.[21] Post-herpetic neuralgia, defined as persistent pain beyond three months following skin lesion resolution, is one of the most common complications of HZ.[21] The risk of PHN increases with age and can occur in up to 20% of those 80 years or older. Pain results from sensory nerve damage, often interfering with day-to-day activities and associated with clinical depression.

Treatment of pain associated with herpes zoster includes the use of nonsteroidal anti-inflammatory drugs, opioids, capsaicin, tricyclic antidepressants, and topical lidocaine ; the level of treatment is dependent on the severity of the pain.[4] Gabapentin can also reduce pain associated with herpes zoster, however Cohen reported that opioids such as oxycodone were more effective at reducing pain than anticonvulsants like gabapentin.[9] Gabapentin works by decreasing the density of

calcium channels in pre-synaptic terminals which result in the decreased release of neurotransmitters that are responsible for pain progression.[27] Gabapentin, and acetaminophen-hydrocodone were prescribed to this patient for pain management by her primary care physician.

Careful follow-up is recommended for at least the first year after the onset of HZO, to identify exposure changes, measure corneal sensation, evaluate for uveitis, promptly identify the development of neurotrophic keratitis, and assess any PHN.[23] Chronic epithelial keratitis may arise as late as one year following cutaneous eruption, and PHN can present as debilitating pain so severe it results in depression and suicidal ideations.[24] Treatment with antidepressants, opioids, and anticonvulsants can remedy these symptoms but eye care providers must first identify the underlying cause with follow-up.

Vaccination for individuals ages 50 and older can prevent or reduce severity of herpes zoster and sequelae, including post-herpetic neuralgia, for at least five years following vaccination.[25] The U.S. Centers for Disease Control recommends the Shingrix vaccine for people 50 years or older. Separated by two to six months, the two-dose vaccine is more than 90% effective at preventing shingles and PHN, and protections remains high for at least four years following vaccination.[12] A recent study by Shekhawat et al. found that rates of HZO have been on the rise, likely due to the fact that individuals have less exposure to chicken pox. Immunity is aided by community re-exposure, so without exposure to chicken pox, individuals are at greater risk for HZO.[26] Patients should be advised if they have not already had chicken pox, they are highly susceptible to VZV.

CONCLUSION:

The VZV infection can present with manifestations that are potentially vision- and life-threatening. Identification of shingles and the importance of referring to an eye care provider to help manage the condition is critical to minimizing sequelae. Asking about a prior history of herpetic infection in any patient presenting with chronic inflammatory eye disease should be considered.

HZO and Bell’s palsy are thought to be linked by way of inflammation of the facial nerve as it traverses through the geniculate ganglion, and its proximity to the trigeminal nerve’s ophthalmic branch. Understanding the ocular complications

of herpes zoster and conducting a thorough case history are subsequently vital to management of HZO and related conditions such as Bell's palsy. The prognosis of HZO and Bell's palsy is dependent on how well symptoms are managed, including close observation beyond resolution. The complications of VZV will hopefully become less common as vaccination becomes more widespread.

Treatment for HZO and Bell's palsy can vary, and conservative treatment must be weighed against issues with polypharmacy and the severity of the disease. The patient's successful clinical course was, in large part, because she was closely monitored even during the COVID-19 pandemic.

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