CASE STUDY

Herpes Zoster Ophthalmicus in Two Virally Suppressed HIV-Positive Patients: Early Initiation of Therapy Improves Visual Outcome

Onyinyechi Anyaso¹*, George Ikpe², Anthonia Udeaja³, Olufunmi Otuka⁴, and Israel Popoola⁵

ABSTRACT

Herpes Zoster is a viral infection, which occurs with reactivation of the Varicella Zoster Virus that has lain dormant in the dorsal or cranial root ganglia. It is often a painful rash, but may also occur as pain along a dermatome, but without a rash. The involvement of the ophthalmic branch of the Trigeminal nerve gives rise to Herpes Zoster Ophthalmicus, with its potential risk of visual loss. Here we present a case of Herpes Zoster Ophthalmicus in two virally suppressed HIV positive men, each having a different outcome as a result of the intervention of the intensive clinical mentorship by the National AIDS, Hepatitis and Sexually Transmitted Infections Program, NASCP, which culminated in the adoption of best practices and improved clinical outcome and prevention of a catastrophic eye event.

Keywords: Herpes Zoster, HIV, Immunosuppression, Visual acuity.

1. Introduction

Herpes Zoster remains a cause of significant morbidity. The global incidence ranges from 1.2 to 3.4 per 1000 persons in the young and healthy population but rises to 3.9 to 11.8 per 1000 persons in persons older than 65, and as high as 50% in a cohort surviving to 85 years. It is thought to rise with increasing age owing to diminished effectiveness of cellular immunity [1], [2]. Risk factors include the elderly, female sex, and those who are immunosuppressed from diseases, for example, HIV, or Diabetes Mellitus or those receiving immunosuppressive therapy [3]. The disease may also occur in the immune-competent subject [4].

It carries a lifetime risk of 30%. Approximately 10% to 20% of patients who have Herpes Zoster will develop HZO [5], with its attendant potential for visual loss. However, if treatment is prompt and initiated within 72 hours of the eruption of vesicles, the prognosis is markedly improved [6]–[9]. The disease is regarded as an ophthalmologic emergency [10] because it is a vision-threatening disease, which may cause profound eye inflammation if left untreated.

Complications include facial paralysis, visual loss, post-herpetic neuralgia and an intense itch over the lesions [11], [12] and even stroke. It is believed that the virus can cause vascular inflammation and occlusion, as the trigeminal nerve fibers extend along the anterior, middle and inferior cerebellar arteries [13].

Typically, patients experience prodromal symptoms, neuropathic pain and vesicular eruptions in a dermatomal fashion, with complete resolution within 4 weeks in the immunocompetent. The presence of vesicles on the tip of the nose, Hutchinson’s sign, is a poor prognostic factor for HZO, usually signifying ocular involvement [2], (Figs. 1 and 2). However, the absence of Hutchinson’s Sign does not rule out ocular involvement [10].

Diagnosis is clinical, based on history and classic physical findings [6]–[9]. Other tests like PCR, and viral cultures are rarely required to establish a diagnosis. Occasionally, HZO may be mimicked by the lesions of herpes simplex virus [12].

Submitted: May 01, 2024
Published: June 13, 2024
10.24018/ejmed.2024.6.3.2114

¹Amachara General Hospital Umuahia, Nigeria.
²National AIDS and Sexually Transmitted Diseases and Hepatitis Control Program (NASCP), Nigeria.
³Ophthalmology Unit, Department of Surgery, Chukwu Emeka Odimegwu Ojukwu University, Nigeria.
⁴Ophthalmology Unit, Department of Surgery, Abia State University Teaching Hospital, Nigeria.
⁵World Health Organization, Switzerland.

*Corresponding Author:
e-mail: onyinyejaneanyaso@gmail.com
2. Case Report

We report Mr. A, a 37-year-old male who presented to the clinic with a 2-day history of scalp discomfort, malaise and unusual pain on the scalp and forehead, and a 1-day history of a painful rash on the same side of his face, associated with a red eye on the affected side, purulent eye discharge, and with the eye swollen almost shut (Fig. 1).

He had been diagnosed with HIV 2 years prior to presentation and had since been on anti-retroviral therapy with a 95% adherence measure. He did not commence any new medication around the time of eruptions, having been on Cotrimoxazole since initiating therapy with no adverse reaction in the form of mucocutaneous involvement.

General examination showed a well-nourished young man, he had no fever and was in moderate painful distress. Vesicles were extending from the left side of the scalp down to the eye, causing peri-orbital edema and redness, and involving the cheek, nose and nasal tip (Fig. 1). There was profound hyperesthesia. There were no signs of facial nerve palsy.

Ophthalmology review reports blepharitis, vesicles along the nerve distribution, profound periorbital swelling and ocular tenderness. In addition, there were conjunctival and episcleral hyperemia, corneal edema and photophobia. Intraocular pressure was 25 mmHg, in both eyes and there was decreased corneal sensation. Using Snellen chart, visual acuity on the affected eye was 20/60, but 20/20 on the unaffected eye which also showed nil abnormal findings. Samples drawn for tests revealed an HB of 14 g/dL and a CD4 cell count of 576 cc/microliter. His HIV viral load was <20 cp/ml, indicating viral suppression.

He commenced Oral Acyclovir 800 mg 5 hourly on the same day (within 72 hours of onset of symptoms) as per the National Guidelines, along with analgesics, amitriptyline, antibiotic ointments, antihistamines to relieve the itch and artificial tears. He had the lesions dressed with anesthetic creams.

A week after the presentation, the patient came for review and by then, had made significant progress. The ophthalmology review showed reduced orbital and periorbital inflammation. The vesicles were in different stages of healing. There were hypopigmented patches at the site of the healed vesicles (Fig. 2). By week 4, he was glad the pain had reduced, but, unfortunately, was replaced by an intense/severe itch over the region. His vision had returned to normal, with a visual acuity of 20/20 using the Snellen chart, although he reported a mild ocular itch (Fig. 3). He had minimal scarring.

Mr. B. was a 40-year-old HIV-positive patient who had been on antiretrovirals for over 5 years prior to presentation. He had been adherent to therapy. He presented to the clinic with a history of having suffered headaches and painful rashes on his face, forehead and nose a month prior to presentation. These were associated with eye pain, purulent eye discharge, with inflamed eyelids that eventually became difficult, and subsequently impossible to pry open. On examination, he was found to be a well-nourished young man.

He was afebrile, in no obvious distress, and showed no signs of facial nerve palsy. He had a linear scar, which extended from the middle of the forehead to the tip of the nose. His right eye was “frozen” shut. There was no associated eye pain or periorbital tenderness. During an ophthalmology review, the affected eye was found to have no perception of light (NPL) and had sunken deep into its sockets (Fig. 4).

3. Discussion

Herpes Zoster is a neurocutaneous infection characterized by painful, blistering skin rashes that occur along a dermatome, resulting from the re-activation of latent
Herpes Zoster Ophthalmicus in Two Virally Suppressed HIV-Positive Patients

Onyinyechi et al.

Fig. 3. The Hutchinson’s sign.

Fig. 4. The profound scars and Hutchinson’s sign.

Varicella zoster virus infection that has lain dormant in the dorsal root ganglia [5], [14]. Herpes Zoster Ophthalmicus occurs when the ophthalmic (V1) branch of the Trigeminal nerve is affected. It may occur with or without ocular involvement [15]. The presence of herpetic skin lesions at the tip of the nose, the Hutchinson’s Sign, is a predictor of ocular complications, as the tip of the nose and ocular structures are both innervated by the nasociliary nerve, a branch V1 [2]. It is now a vaccine-preventable disease, as administration of the Zoster vaccine, Shingrix, as 2 doses given two to six months apart, has a vaccine efficacy of 97.2% risk reduction in patients aged 50 years and older [3], [16].

Of those who acquire Herpes Zoster infection, approximately 10%–20% will develop herpes zoster ophthalmicus [17], with 50%–71% of these developing eye complications [4]. The global incidence of HZ is 2.9–19.5/1000 person-years, and of those who acquire it, 7.9% go on to develop Herpes Zoster Ophthalmicus [18].

Incidence increases with advancing age, with the attendant reduction in cell-mediated immunity the older one gets. Several other factors have been found to trigger this disease. These include a weakened immune system, being more than 50 years old, experiencing a high level of physical and emotional stress, and being on anti-cancer medications, especially when treating leukaemias and lymphomas [12]. In addition, there is an ever-growing list of risk factors. Traumatic brain injuries, a positive family history, depression and heart failure have all been implicated [4], including the use of statins [19].

HZO is similar to HZ in its disease process. It invades the respiratory tract through infected droplets, which replicate and invade the adjacent lymph nodes, from where it changes to a cutaneous infection, moving in an anterograde fashion towards the superficial tissues, and finally emerging as shingles [14]. The reactivated virus descends from the trigeminal root ganglion to the ophthalmic branch of the trigeminal nerve, which supplies the skin of the forehead, eyelids, eyeballs and nose [12].

Clinical features include fever, malaise, headache and eye pain before the emergence of rashes. The rashes initially appear to be maculopapular and gradually transition to vesicular or papular which then rupture and form scabs. There may be orbital swelling, ptosis, keratitis, uveitis, central opacification and corneal scarring [14].

Approved and recommended treatment is with Valacyclovir 1000 mg three times daily, Acyclovir 800 mg five times daily [6], or famciclovir 500 mg daily, all for 7–10 days, for the immunocompetent. However, for the immunocompromised, intravenous anti-virals should be used [7]. No additional benefit was achieved by using topical acyclovir, although a study revealed a better outcome with Interferon gel [9].

Anti-virals should be initiated immediately [6], as HZO is an ophthalmologic emergency [10]. Other measures include the use of steroids, antibiotics for superimposed bacterial infections, analgesics, and corneal debridement, as supportive care is tailored to the case.

4. Complications

HZO may come with an array of complications. It may lead to blepharitis, conjunctivitis, keratitis, uveitis, and cornea may become denervated leading to neuropathic keratitis [20]. Among neurological emergencies, post-herpetic neuralgia (PHN) is the most common [21], and more rarely, stroke [13]. Manifestations of PHN decrease with time, affecting 30% in 6 weeks, and 9% after one year [22]. Older age, immunosuppression, uveitis and poor visual acuity on presentation were found to be poor prognostic factors for loss of vision [11].
5. CONCLUSION

Herpes Zoster ophthalmicus is an ophthalmologic emergency. The community should be made to know that it is vaccine-preventable and all immunocompetent individuals >50 years old should receive the new Shingrix vaccine. If it does occur, treatment should be prompt and initiated within 72 hours of the emergence of symptoms in order to avoid bad ocular outcomes.

A public health approach via clinical mentorship is highly recommended as a way of communicating best practices to primary care physicians in order to encourage prompt diagnosis of HZO and timely initiation of treatment to prevent catastrophic visual outcomes, as can be evidenced by this report.

CONFLICT OF INTEREST

Authors declare that they do not have any conflict of interest.

REFERENCES


