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Case Report

"The Myriad of Postherpetic Anterior Uveitis in Herpes Zoster Viral Infection"

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Abstract

Herpes viridae is an infective family of viruses that is common worldwide. Additional 100 herpes viruses have been categorized, but humans may affect by only eight that are Herpes simplex virus (HSV), varicella-zoster virus (VZV), cytomegalovirus (CMV), Epstein-Barr virus (EBV), human herpes virus [1]. That broad spectrum of viruses can cause various ocular diseases with clinical presentations of keratoconjunctivitis, dendritic epithelial keratitis, Blepharitis, geographic or trophic herpetic corneal ulceration, stromal keratitis, and intraocular inflammation. Two significant entities may comprise ocular inflammation, anterior uveitis with or second with active corneal involvement as epithelial keratitis or interstitial keratitis, and viral retinopathy, seen in both immunocompetent immunocompromised patients. An estimated 90% of intraocular inflammation is associated with anterior uveitis, and 5% to 10% of uveitis cases have chances of herpetic anterior uveitis due to herpes simplex or varicella-zoster virus infection [2-4]. It is the common cause of anterior infectious uveitis. This case outlined an overview of post herpetic anterior uveitis, its occurrence in Herpes Zoster, and the effectiveness of medical therapy.

Keyword: *HZO (Herpes Zoster Ophthalmicus); OD (Ocular Dexterous); OS (Ocular Sinister); PHN (Post Herpetic Neuralgia); The Herpetic Eye Disease Study (HEDS); Cytomegalovirus (CMV); Epstein-Barr Virus (EBV)*

Introduction

Case Report

A 45-year-old Asian male presented with chief complaints of red eye, lacrimation, persistent, intense pain, photophobia, and blurred vision in the right eye for two days. He denied an ocular history of spectacles. His previous history showed systemic therapy antiviral acyclovir 800 mg tablet oral qid for eight d, prednisolone, 1% eye, drops for Herpes Zoster ophthalmicus before six months, and an as PHN history of severe pain after three months of infection HZ, and birth history showed measles. He denied any itching and rashes on examination day. Moreover, his cutaneous examination showed a previous scar on the right face due to pustules and cysts. He rejected any insect bites, hearing problems, and shortness of breath. Family ocular history was none. Social history was normal. He had no known drug allergies. He had fatigue and depression and was not oriented to time, place, and person.

Uncorrected distance visual acuity was counting finger 2 Feet OD and 20/40 OS. There was no previous habitual spectacle correction for distance and near. Near acuity showed Nil OD and N12 OS to N5 with +1.50 additions. Manifest refraction revealed no improvement due to incomplete open eyes opening left eye 20/25 with + 1.75 DS. Goldman applanation tonometry measured 17 mmHg OD, 12 mmHg OS at 10:03 am. The right eye showed blepharospasm with increased sensitivity to light. Pupils were equal, round, and reactive. Slit-lamp Biomicroscopy of right revealed normal adnexa with lid swelling with circumcorneal congestion. And left eye showed normal lid adnexa, lashes, and puncta. The right cornea findings revealed diffused corneal edema inflammation and endothelial decompensation. Epithelial keratitis was absent. Right eye anterior chambers appeared with evidence of cell or flare, and Kps, uveitis, and chamber angle estimation was 4/4 via the Von Herrick method. The contour of irides was flat and brown in the left eye, where sectoral atropine in the right was noted without posterior synechiae. When pupils were dilated with one drop, 1% Mydriacyl showed lenticular changes posterior subcapsular changes grade III. Marked vitritis was pointed out in the right eye. Detail fundus assessment of the left eye revealed normal optic nerves contour with a cup-to-disc ratio of 0.3 in both eyes; the right fundus couldn't be revealed due to hazy media and inflammation.

Diagnosed the disease

The diagnosis was relatively straightforward because of an accompanying skin scar, active interstitial keratitis, and corneal anesthesia. However, in the absence of preliminary evidence of current or past HSV infection, it requires clinical suspicion and observation. Anterior uveitis secondary to HSV or VZV is associated with active or inactive corneal involvement, but anterior uveitis without associated corneal changes also occurs as an isolated entity.

The diagnosed ocular case was anterior herpetic Uveitis with corneal edema, a rare non-ulcerating clinical case associated with Herpes Zoster Varicella HZV Virus. The determined depth of inflammation of stroma and other corneal manifestations required a comprehensive Slit-lamp biomicroscopy, patient History of HZO, and intraocular pressure guard. For stromal keratitis, which is the most visually significant problem for the cornea, a delicate balance of steroids is sometimes necessary, and even with it, patients can lose vision. Stromal keratitis can also be seen in the recurrent case of 20% of eyes with HZO.

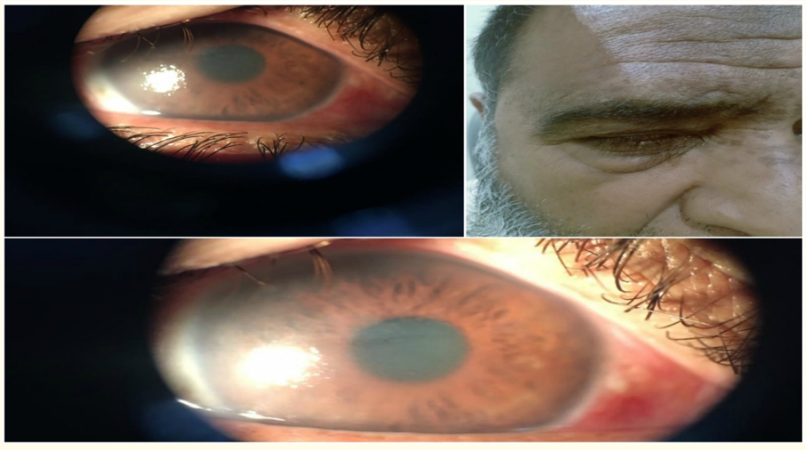

Figure 1: *(Photo courtesy of Dr. Rubina Shah National Eye Centre) NOTE: The biomicroscopy showed diffuse corneal edema and endothelial decompensation. Cutaneous scarring right side of the face revealed a Herpes zoster.*

Differential diagnosis

Increased awareness of the characteristic clinical features is essential for early diagnosis and appropriate treatment.

Fuchs' uveitis syndrome

Fuchs' uveitis syndrome [4] can be difficult to discriminate from herpetic anterior uveitis because of transillumination defects, elevated intraocular pressure, and iris atrophy. However, iris atrophy tends to be concentrated over the iris sphincter and stromal rather than full-thickness. The transillumination defects showa radial pattern rather than sectoral, and the cumulative damage to the trabecular meshwork rather than a transient trabeculitis are marked in raised IOP. It is thus unresponsive to topical corticosteroid and antiviral treatment. Additionally, the Fuchs' uveitis syndrome patient is not inclined to form posterior synechiae. In contrast, posterior synechiae were frequently included in patients with herpetic anterior uveitis and were noted in 58% of the patients from the Netherlands. It shows an abnormal uveal pigmentation with chronic low-grade inflammation and ultimately causes iris atrophy and secondary glaucoma. Manifest ocular symptoms have no pain, redness of the external eye, or miosis and may associate without any other systemic disease is present. Even though it typically affects unilaterally, 7.8% - 10% of affected patients have bilateral conditions [5]. It may be undiagnosed during a routine examination or present with raised IOP [6]. Treatment requires short-term topical corticosteroids for flares. However, it might need long-term therapy. Moreover, like other uveitides, a must be prescribed to break down the blood-aqueous barrier and inflammatory activity to eliminate cells from the anterior chamber.

Posner-schlossman syndrome

Posner-Schlossman syndrome is a glaucomatocyclitic condition of the eye that is self-limited and recurrent episodes of raised intraocular pressure (IOP) and anterior chamber inflammation. It is known as secondary inflammatory glaucoma. Posner and Schlossmann first diagnosed the features [7]. The glaucomatocycliticc crisis is characterized by a slight decrease in vision, elevated IOP, open anterior chamber angles, normal visual fields, and optic nerve appearance. In addition, IOP and outflow facility are normal between episodes [8]. It is bilateral and very rare simultaneous [9,10]. Currently, the literature supported cytomegalovirus (CMV) infection as the inflammatory precursor to the anterior uveitis and raised IOP [11]. The patient may report blurred vision or halo visionraisedaise IOP or if there is corneal edema-the history of past attacks of blurred vision lasting several days or monthly or yearly. The treatment revolves around complete medical care with a history of present illness, any drug allergies, eye examination, a careful explanation of the disorder, and an understanding of the disease with long-term follow-up [12].

Peripheral corneal endotheliitis

The primary focus of inflammation in this condition (peripheral corneal enendothelinsr PCE), initially thought to be autoimmune (Khodadoust., *et al.* 1982), appears to be the corneal endothelium, but significant anterior uveitis is usual. Patients present with the unilateral or bilateral blurring of vision. The central cornea is usuastraightforwardlear, but a variable area of the corneal periphery may be edematous-essential ally this a white in the filtration of the deep cornea which may be seen. The distinction of the border between apparent normal endothelium may be seen as a distinct line, similar to the Khodadoust line of corneal rejection after corneal transplantation. Generally, large KPs present at the abnormal area, and gonioscopy may reveal AC anterior chamber angle within the affected loci. Scar cases are associated with intermediate uveitis Khodadoust., *et al*. 1986). Herpetic inflammation is a common manifestation that causes disciform corneal endothelins and other viruses with mumps, varicella-zoster virus (VZV), human herpesvirus 7 (HHV7), and rhabdo-

virus can also affect the endothelium. Nevertheless, there is the concept of CMV as etiology of corneal endothelins(Chee., *et al*. 2007) and following corneal graft, with linear deposition of KPs and focal endothelial lesions features(Koizumi., *et al*. 2008).

Generally, Fuchs uveitis is associated with HSV and CMV [13-15]. The most helpful diagnosis of herpetic anterior uveitis is sectoral iris atrophy and raised IOP. However, various inflammatory and noninflammatory diseases include bilateral iris atrophy, Fuchs uveitis syndrome, Vogt-Koyanagi-Harada disease, pigment dispersion syndrome, acute angle-closure glaucoma, trauma, and Horner's syndrome. A recent study has shown diffuse depigmentation and bilateral geographical changes in the iris stroma [16]. The differential diagnosis of the acute rise of IOP can also occur in granulomatous anterior uveitis with the systemic association of sarcoidosis, toxoplasmosis, syphilis, and leprosy, and also in Posner-Schlossman syndrome [17]. The recurrency is typically noted temporality with acute onset of Herpetic anterior uveitis disease. Consequently, that challenges the course from chronic entities like Fuchs uveitis syndrome and Vogt-Koyanagi-Harada disease. The DDx differential diagnosis can also be judged from the distribution of KPs. Herpetic KPs are large, greyish in color, flat, and deposit on the central corneal endothelium. However, Fuchs uveitis syndrome diffuses medium-sized Kps, which is hard to differentiate [18]. While herpetic KPs usually vanish with treatment. The acute episode may present with focal edema that may cause irregular dilation. The clinical presentation of HZV and HSV cannot be judged from the appearance of iris atrophy. However, VZV uveitis may occur without skin involvement (herpes zoster sine herpete) [19]. A clinical diagnosis of VZV uveitis can only be diagnosed with past attacks of herpes zoster ophthalmicus cutaneous involvement. Van der Lelij., *et al* [20]. termed that aqueous humor analysis revealed HSV in 83% and VZV in 13% with sectoral iris atrophy. However, sectoral iris atrophy can also be seen in recurrent unilateral herpetic anterior uveitis affected by CMV [21]. The IOP elevation is associated with acute trabeculitis, and prompt response to anti-inflammatory treatment is noted [22].

Treatment

The mainstay therapy of herpetic anterior uveitis is topical corticosteroids and oral antiviral agents. The Herpetic Eye Disease Study [23] demonstrated that prophylactic oral acyclovir could hamper the number of recurrences of the disease for 12 months. In a retrospective study, miserocchi., *et al.* showed long-term prophylactic acyclovir's effectiveness beyond 12 months [24]. Miserocchi., *et al* [24]. reported the tender of lifelong treatment using oral acyclovir 800 mg daily for at least two years after herpetic uveitis. It showed that 13% of our patients could use oral acyclovir for at least 12 months. The high cost of long-term treatment restricted the use of prophylactic therapy.

The pathogenesis of herpetic iridocyclitis is believed to be involved in active viral replication and the host immune responses. Topical corticosteroids and antiviral agents are commonly used to treat ocular herpetic disease. Topical corticosteroids control the iridocyclitis and acutely decrease intraocular pressure owing to their anti-inflammatory effects on the trabecular meshwork. However, topical and oral antihypertensive agents may be necessary to control the ocular pressure.

Corticosteroids should be slowly tapered once the inflammation is regregressed and topical antiviral agents affect epithelial keratitis, but a much deeper ocular involvement pattern has not been recognized. It should be used with corticosteroid drops to prevent recurrent epithelial keratitis. Yet, another topical anti-herpetic agent of treatment of choice has become gancyclovir gel.

A detail concerning oral acyclovir, demonstrated by the HEDS (herpetic eye disease control group) results, suggests a benefit of oral acyclovir in treating herpetic iridocyclitis in patients receiving topical corticosteroids and topical antiviral prophylaxis. And, Oral acyclovir at a dosage of 400 mg 5 times per day for several weeks is usually used. Intravenous acyclovir (10 mg/kg/d) may be considered severe anterior uveitis and must be employed in all immunocompromised patients. Alternatively, one may use valacyclovir 1 g 3 times per day or famciclovir 500 mg 3 times daily. Given on a long-term basis, oral acyclovir, 600 to 800 mg/day, can diminish the recurrence of herpetic anterior uveitis.

Ocular medication

Ocular medication prescribed by ophthalmologist endothelial decompensation and uveitis was cyclopean eye drop qid, Eyebradex eye drop eight times a day, synigan eye drop twice a day, ophthypertonic saline solution 4x day.

The patient had a history of postherpetic neuralgia and was using PHN medication Deltracotril 30 mg analgesics, Tab tramal pain reliever in case of severe pain, Tab tegral (anticonvulsant) 200 mg twice a day. Patient had a history of Oral acyclovir 800mg during attach of HZ herpes Zoster. Ocular Medication used was Tobrex eye qid, Zaviraz ointment tid.

General treatment protocol PHN for the disease turns around reducing the neuronal membrane and beginning and transmission of the impulse. The tranquilizing effect and anticholinergic effect can be reduced by a group of tricyclic antidepressants such as amitriptyline(Evanil) may be helpful in severe neuronal pain. Second, the clinical course of antiviral has been concise for antantiviralsat inhibits complications and recurrences. Such as, famciclovir directly acts on the Viral DNA synthesis. Third, anticonvulsants can be used for severe muscle spasms and provide restfulness during pain compared with placebo treatment specific medication such as gabapentin products (i.e., Neurontin, Gralise, Horizant), describe statistical progress in pain scores (i.e., a decrease by at least 50% from baseline). Fourth, corticosteroids play a vital role as anti-inflammatory action, which modifies the body's immune response to various stimuli as dexamethasone is used for aller and inflammatory as well pred methylprednisone. Fifth, studies have shown that some natural chemicals derived from plants of the Solanaceae family may lessen skin and joints insensitive to pain. It is thought be acting as a chemomediator of pain transmission from the periphery to the CNS, such as capsaicin. Topical anesthetics such as lidocaine can stabilize neuronal membrane and its neural activity.

Follow up #1 (week)

On the exam date, the patient revealed a vision of the affected eye 20/60 with no improvement with refraction. Slightly regressed edema was noted. Mydriatic pupil and lenticular changes. Slightclearancee of vitritis. Fundi were unremarkable. Topical Eyevradex ointment during the night, eyebradex eye drops five times daily. Tears plus eye drop four times a day.

Follow up #2 (weeks)

On the examination date, the patient revealed edema of the cornea with vision 20/40 in The regressed flare cells. PThe patient was recovering from the herpetic stage. Corticosteroids were tapered, and artificial tears were given. Although variable follow-ups and the nature of the study did not allow calculation, recurrences seemed to be more frequent after discontinuation of treatment. We did not observe severe bounce attacks since we tapered topical steroids very slowly.

Follow up #3 (3 weeks)

Follow-up examination showed regressed symptoms with visual acuity of 20/30 in the right eye with a manifest refraction of -2.00DS and 20/40 to 20/25 in the left eye + 1.75DS. Near acuity showed N10 on the right and N5 on the left with + 1,50 addition for near. The patient was advised artificial tears and spectacle correction for distance and near. However, the cataract of grade III PSSC posterior subcapsular cataract was still noted, and the educated patient may experience a gradual decline in vision. Further, follow up was required after three month to rule out the recurrence.

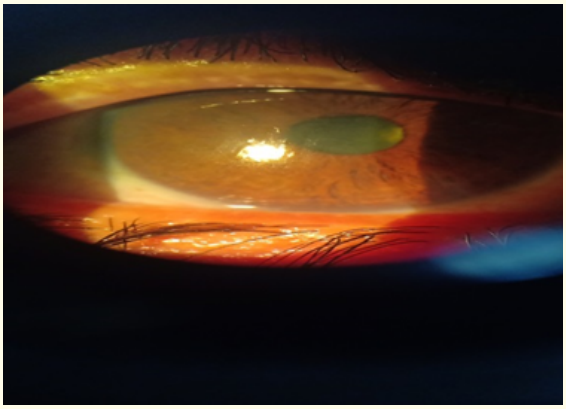

Figure 2: *The illustration of the cornea after regressed herpetic keratitis.*

Discussion

The major morbidity worldwide is Herpes viruses which have eight distinct viruses, with six of them affecting humans. Specifically, herpes simplex virus, herpes zoster virus and cytomegalovirus, and HSV2 and VZV3 are the most common ocular pathogens, with an approximate incidence of 10 to 20/per 100,000 person-years estimations [25-27].

The case was reviewed and managed for Herpetic anterior uveitis associated with a history of postherpetic neuralgia and herpes zoster infection. Clinical presentation may cover broad ocular manifestations such as Blepharitis, conjunctivitis, scleritis, keratitis, anterior uveitis, necrotizing retinitis, choroiditis, and optic neuritis of anterior uveitis was ruled out. The anterior chamber activities of inflammation occur in up to 10% of patients with HSV keratitis [28-30] and 50% of immunocompetent patients with herpes zoster ophthalmicus [31-33]. Although anterior herpetic uveitis diagnosis is usually straightforward due to dermatitis or dendritic keratitis, it can be tricky in the absence of these signs. Moreover, nonspecific symptoms like patients' complaints of blurred vision, pain, redness, and photophobia are usually seen. Localized corneal scars or edema decreased corneal sensation, geographically or diffusely distributed keratic precipitates, acutely raised intraocular pressure, and iris atrophy, which is frequently localized and produces both pupillary distortions the clue to diagnosing herpetic uveitis.

Additionally, it is almost always unilateral. Perhaps harder to discriminate anterior herpetic uveitis distinguishing features of HSV from VZV due to anterior chamber inflammation [29,34]. The history of herpes zoster ophthalmicus provides a supportive and striking way to diagnose VZV-induced iritis, but it's not necessary to notice the presence of cutaneous lesions. The reduction of corneal sensation can be challenging to quantify, but it also suggests the presence of VZV. Similarly, the company of pattern of dendritic keratitis may help distinguish HSV from VZV keratitis. The prominence of well-developed terminal end bulbs of HSV may stain with fluorescein. Still, the clinical features of VZV pseudo dendrites are usually slightly elevated, broader, and with less regular branching, few terminal dilatations or polymorphous that stain centrally with rose Bengal staining or edge pooling with fluorescein. Moreover, sectoral iris atrophy is highly suggestive of VZV-induced anterior uveitis and urges some investigation, such as serum anti-VZV antibodies that have led to iris sectoral

atrophy [29,34,35]. It remains likely to measure directly herpes virus deoxyribonucleic acid or antiherpes virus antibodies in the aqueous humor from patients associated with sector iris atrophy. The article by Van der Lelij and colleagues [36], which appeared in the June issue of Ophthalmology, addressed this importance where the authors performed anterior chamber paracentesis on a significant, seen unilateral anterior uveitis association with sectoral iris atrophy have no evidence of concurrent epithelial and stromal keratitis that was a clinical-based cohort of patients from the Netherlands. About 20% of the patients defined by Van der Lelij and associates were initially thought to have Posner Schlossman syndrome (glaucomatocyclitic crisis). The differential diagnosis of herpetic anterior uveitis [37] can also be difficult to distinguish from Fuch's endothelial uveitis because of trans illumination defects, elevated intraocular pressure, and iris atrophy. It is evident that herpetic anterior uveitis with being treated with antiviral drugs [33,34]. Oral acyclovir, 400 mg twice daily, has been shown to reduce the recurrence rate of HSV [33].

The Herpetic Eye Disease Study (HEDS), prospective, randomized, double-masked, placebo-controlled multicenter trial supported by the National Eye Institute, established further that the effect of oral acyclovir decreases the recurrence rate of herpetic stromal disease and its beneficial prophylactic effect depends on continued use of suppressive doses of an oral antiviral agent [38]. The HEDS collaborators also examined the effect of oral acyclovir on the duration and number of recurrences of anterior uveitis in patients with prior or concurrent herpetic keratitis [39]. Nevertheless, it failed to demonstrate statistical significance of the overall effect, even notifying the benefit trend of Oral acyclovir therapy. Likewise, there are various number antiviral agents are recently available to treat herpetic anterior uveitis. Oral acyclovir has been shown to reach the therapeutic level in both tears and aqueous humor [40], essentially eliminating the requirement of antiviral, even inactive corneal involvement.

Even controlled comparisons have yet to be considered in patients with eye disease. The more convenient dosage provided to benefit the patient is valacyclovir, 1 g three times daily, and famciclovir, 500 mg three times daily, which appear to be equivalent to oral acyclovir for the treatment. Furthermore, valacyclovir benefits have shown a prodrug of acyclovir that is absorbed much better from the gastrointestinal tract and tends to yield threefold to fourfold higher circulating drug levels than oral acyclovir. Another valacyclovir antiviral agent has been reported to cause thrombocytopenic purpura or hemolytic uremic syndrome in a small number of patients infected by the human immunodeficiency virus. However, it must probably be avoided in this population of patients. Alternatively, topical antiviral agents fail to reach therapeutic levels and absorb in the aqueous humor [41]. The side effects can involve the corneal and conjunctival epithelium for prolonged dosage, causing 5% to 10% allergic blepharoconjunctivitis or a corneal epitheliopathy [42,43]. The single exception is Acyclovir ointment which stabilizes adequate aqueous levels and is well tolerated for prolonged usage. It causes blurry vision but isn't readily available clinically. However, topical therapy with ointment does not directly affect viral recurrence in sensory ganglia. For these reasons, practioner prefer oral therapy of acyclovir over ointment after evaluating the condition of the anterior chamber and is considered safest for more extended period therapy in HSV and HZV involving uncontrolled eye problems eye. The active disease of the postherpetic uveitic eye also needs corticosteroid therapy and a cycloplegia/mydriatic agent to avoid any posterior synechiae formation and reduce pain and provide comfort. A core treatment regimen might include prednisolone acetate, 1%, and tropicamide, 1%, every four times daily. Reducing the course of corticosteroids can be challenging, and May requires months to achieve the ultimate effect. To control long-term inflammation, some patients may need low-dose corticosteroids indefinitely.

Exemplifying the available clinical data, physicians and ophthalmologists should recommend the best treatment systemically and ocular to prevent any further worsening of the disease. For PHAU Post herpetic anterior uveitis, current therapy for older adults age modifies administering the herpes zoster vaccination. Further, clinically a systemic trial medication associated with PHN postherpetic neuralgia or herpes zoster can also be guarded with the latest treatment, such as patching monotherapy before resorting to a systemic therapy must be considered in PHN patients. Alternatively, it may be administered in combination with a tricyclic antidepressant or gabapentinoids to potentiate analgesic response and reduce the dose requirement of systemic therapies.

Conclusion

Post Postherpetic uveitis is a specific condition caused by infection of the zoster virus. Herpes zoster (HZ) infection occurs when a varicella-zoster virus (VZV) reactivates. It remains dormant after the primary attack in the dorsal root of the ganglia. The characteristic raises a painful vesicular rash with the unilateral dermatomal distribution. The myriad of infections is approximately 1 million estimated in the USA, and one in three individuals gets infected from herpes zoster which has worldwide geographic distribution. Still, annual epidemics are more widespread in temperate climates, and over 90 percent of adults in the United States who have evidence of VZV infection are at risk for herpes zoster [44]. In Pakistan and certain other Asian countries, the overall prevalence of primary varicella infection is relatively low and late [45,46]. This change in climatic conditions may be responsible for a different seasonal and morphological pattern in herpes zoster. Uveitis is primarily either an ocular condition or related to various systemic disorders. The analyzed frequency of systemic diseases associated with uveitis varies from 18% to 43% [47,48-52]. It is crucial to managing the ocular involvement and better treatment protocol to manage the patient. The recurrence of the disease can be addressed to reduce and prevent vision-threatening conditions such as keratitis, glaucoma, and cataract in herpetic AU with an early and accurate diagnosis. The developing treatment available for ocular manifestation requires oral antiviral agents and topical steroids, which are current herpetic AU treatments. Prophylactic oral acyclovir is evidenced by a reduction in the number of recurrences of herpetic eye disease. Longer follow-up and long-term treatment can reduce the recurrence of anterior herpetic Uveitis.

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