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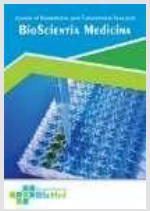

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Successful Management of Panuveitis and Associated Complications in a Patient with Clinical Stage III HIV: A Case Report

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A B S T R A C T

Background: Human immunodeficiency virus (HIV) infection can lead to a myriad of ocular complications, including panuveitis, an inflammatory condition affecting the entire uvea and surrounding structures. The management of panuveitis in HIV patients is complex, often requiring a multidisciplinary approach to address both the underlying immunodeficiency and the ocular inflammation. **Case presentation:** We present the case of a 37-year-old male patient with clinical stage III HIV who presented with panuveitis in the right eye (OD) secondary to herpes zoster ophthalmicus (HZO). The patient also had post-herpetic neuralgia, oral candidiasis, and malnutrition. He reported a history of unprotected sexual encounters with multiple partners. The patient's CD4 count was critically low at 15 cells/μL, indicating severe immunosuppression. He was initiated on antiretroviral therapy (ART), prophylactic medications to prevent opportunistic infections, and pain management for post-herpetic neuralgia. **Conclusion:** This case highlights the challenges in managing panuveitis and associated complications in patients with advanced HIV infection. Early diagnosis, prompt initiation of ART, and a multidisciplinary approach are crucial for successful management and improving patient outcomes.

1. Introduction

The human immunodeficiency virus (HIV) is a lentivirus that targets the CD4+ T cells, leading to progressive immunodeficiency and ultimately acquired immunodeficiency syndrome (AIDS) if left untreated. The global prevalence of HIV/AIDS remains a significant public health concern, with an estimated 38.4 million people living with HIV in 2021. While the advent of antiretroviral therapy (ART) has significantly improved the prognosis and quality of life for people living with HIV (PLWH), the virus continues to pose a formidable challenge due to its ability to evade the immune system and establish latent reservoirs. HIV infection can manifest with a wide range of clinical presentations, depending on the stage of the disease and the presence of opportunistic infections or malignancies. In the early stages, individuals may experience flu-like symptoms or remain asymptomatic. As the disease progresses and the CD4+ T-cell count declines, patients become increasingly susceptible to opportunistic infections, which can affect various organ systems, including the eyes.[1,2]

Ocular manifestations are common in HIV-infected individuals, with an estimated prevalence ranging from 30% to 70%. These manifestations can be broadly classified into two categories: those directly caused by HIV infection and those resulting from opportunistic infections or immune reconstitution inflammatory syndrome (IRIS). HIV-related ocular complications can affect any part of the eye, including the cornea, retina, optic nerve, and uvea. Uveitis, an

inflammation of the uvea, is one of the most frequent and potentially sight-threatening ocular complications in PLWH. The uvea comprises the iris, ciliary body, and choroid, which are collectively responsible for controlling the amount of light entering the eye, focusing the lens, and providing nourishment to the retina. Uveitis can present with a variety of symptoms, including eye pain, redness, blurred vision, and photophobia.[3,4]

The pathogenesis of HIV-associated uveitis is complex and multifactorial. HIV can directly infect ocular tissues, leading to inflammation and tissue damage. Additionally, the virus can disrupt the immune system, increasing the risk of opportunistic infections that can cause uveitis. CMV retinitis, toxoplasmosis chorioretinitis, and herpes zoster ophthalmicus (HZO) are among the most common opportunistic infections associated with uveitis in PLWH. The management of HIV-associated uveitis requires a comprehensive approach that addresses both the underlying HIV infection and the specific ocular manifestations. Early diagnosis and prompt initiation of ART are crucial in preventing vision loss and improving overall prognosis. In addition to ART, specific treatment for the underlying cause of uveitis, such as antivirals, antifungals, or corticosteroids, may be necessary.[5,6]

Panuveitis, a severe form of uveitis affecting the entire uvea, is a particularly challenging condition to manage in PLWH. It can lead to significant visual impairment and blindness if not treated promptly and effectively. The diagnosis of panuveitis requires a thorough ophthalmological examination, including visual acuity assessment, slit-lamp biomicroscopy, and funduscopy. Additional investigations, such as optical coherence tomography (OCT) and serological testing for infectious agents, may be necessary to identify the underlying cause. The treatment of panuveitis in PLWH often involves a combination of ART, systemic corticosteroids, and intraocular injections of corticosteroids or anti-vascular endothelial growth factor (VEGF) agents. The choice of treatment depends on the underlying cause, severity of inflammation, and the patient's overall health status. Close monitoring and follow-up are essential to assess treatment response and adjust therapy as needed.[7,8]

In addition to panuveitis, PLWH is also at risk for other ocular complications, such as retinitis, optic neuropathy, and keratitis. These complications can further contribute to visual impairment and negatively impact the patient's quality of life. Therefore, it is crucial to maintain a high index of suspicion for ocular manifestations in PLWH and initiate prompt evaluation and treatment to prevent vision loss.[9,10] This case report describes the successful management of a patient with clinical stage III HIV who presented with panuveitis and associated complications, including HZO, post-herpetic neuralgia (PHN), oral candidiasis, and malnutrition. The patient's clinical course highlights the challenges and complexities involved in managing ocular complications in advanced HIV disease. The prompt initiation of ART, along with appropriate prophylactic and symptomatic treatment, led to a significant improvement in the patient's ocular symptoms, visual acuity, and overall quality of life. This case underscores the importance of a multidisciplinary approach in managing complex cases of HIV-associated ocular complications and emphasizes the need for early diagnosis, prompt treatment, and close follow-up to prevent vision loss and improve patient outcomes.

2. Case Presentation

A 37-year-old male patient presented to the ophthalmology clinic with a chief complaint of progressively worsening right eye pain, accompanied by blurred vision and photophobia, over the preceding three days. This acute exacerbation was superimposed on a chronic, persistent pain in the right eye and the corresponding side of his face and head that had been ongoing for seven months. The patient described the chronic pain as a stabbing sensation. Further inquiry into his medical history revealed a concerning constellation of symptoms and risk factors. He reported the development of oral

candidiasis (thrush), manifested as white patches on his tongue, which had led to a significant decrease in appetite and an unintentional weight loss of approximately 12 kilograms. Additionally, he disclosed a history of engaging in unprotected sexual intercourse with multiple male partners over the past six years, a known risk factor for HIV acquisition.

The patient's past medical history was also significant for a recent episode of herpes zoster ophthalmicus (HZO) affecting the right side of his face, which had occurred seven months prior to his presentation. Although the acute cutaneous manifestations of HZO had resolved, he continued to experience persistent pain in the affected area, consistent with post-herpetic neuralgia (PHN). Prior to his presentation at the ophthalmology clinic, the patient had sought medical attention at a local health center (Puskesmas Pariaman) due to his ongoing symptoms. A rapid HIV test performed at the health center yielded a positive result, prompting his referral to our facility for further evaluation and management. Upon physical examination, the patient appeared visibly underweight, corroborating his reported weight loss. Oral examination confirmed the presence of oral candidiasis, with characteristic white plaques observed on his tongue. An ocular examination of the right eye revealed several abnormalities, including the presence of crusts and scars on the superior eyelid, conjunctival injection (redness), and corneal clouding. Visual acuity in the affected eye was markedly reduced to 1/~, indicating significant visual impairment. Funduscopic examination of the right eye was attempted but proved challenging due to the corneal opacity, hindering a detailed assessment of the posterior segment.

Laboratory investigations were conducted to confirm the diagnosis of HIV infection and assess the patient's immune status. Both Anti-HIV confirmation I and Anti-HIV confirmation II tests returned reactive results, definitively establishing the diagnosis of HIV infection. The patient's CD4+ T-cell count was critically low at 15.0 cells/μL, indicative of advanced HIV disease and severe immunosuppression. Further serological testing revealed positive IgG antibodies for CMV, Toxoplasma gondii, and HSV-1, suggesting prior exposure to these pathogens. A non-contrast brain CT scan was performed to rule out cerebral toxoplasmosis, a common opportunistic infection in individuals with advanced HIV, and the results were within normal limits. Optical coherence tomography (OCT) was utilized to evaluate the structural integrity of the eyes. While the right eye was difficult to assess due to the corneal opacity, the left eye showed no abnormalities on OCT. This finding further supported the suspicion that the patient's panuveitis was primarily attributable to HZO, rather than other potential causes such as CMV retinitis or toxoplasmosis chorioretinitis.

Table 1. Timeline of disease progression.

Time point	**Event**
6 years prior	Unprotected sexual encounters with multiple partners (Risk factor for HIV acquisition)
7 months prior	Herpes Zoster Ophthalmicus (HZO) affects the right side of the face
Subsequent to HZO	Development of post-herpetic neuralgia (PHN) on the right side of the face
Unspecified timeframe	Onset of oral candidiasis (thrush) and associated weight loss (approximately 12 kg)
3 days prior to present	Exacerbation of right eye pain, blurred vision, redness, and photophobia
Present	- Clinical presentation with panuveitis OD, PHN, oral candidiasis, and malnutrition. - Laboratory confirmation of HIV infection (CD4 count 15 cells/μL). - Serology positive for CMV, Toxoplasma, and HSV-1 IgG. - Initiation of ART, prophylactic medications, and pain management.
Subsequent Weeks	- Gradual improvement in ocular symptoms and visual acuity. - Resolution of oral candidiasis. - Reduction in PHN severity.

Table 2. Treatment timeline.

Time point	Intervention	Rationale
Initial presentation	- Antiretroviral Therapy (ART): Tenofovir, Lamivudine, and Dolutegravir (TLD)	Suppress HIV replication, improve immune function, and reduce the risk of opportunistic infections.
	- Prophylactic Medications: Cotrimoxazole, Isoniazid, Vitamin B6, Fluconazole	Prevent opportunistic infections in the context of advanced HIV disease.
	- Pain Management: Analgesics, Gabapentin	Address post-herpetic neuralgia and other sources of pain.
Subsequent weeks (follow-up)	- Monitoring for ART adverse effects and opportunistic infections. - Assessment of treatment response: Improvement in ocular symptoms and visual acuity. Resolution of oral candidiasis. Reduction in PHN severity.	Ensure treatment efficacy and safety, and adjust management as needed.

3. Discussion

The diagnosis of HIV clinical stage III, also referred to as AIDS, signifies a critical juncture in the progression of HIV infection, carrying profound implications for the patient's health and overall prognosis. This stage is characterized by a severely compromised immune system, a state tragically exemplified by our patient's critically low CD4 count of 15 cells/μL. This numerical value, far below the normal range, paints a stark picture of the immune system's devastation, leaving the patient perilously vulnerable to a wide array of opportunistic infections and malignancies that can infiltrate and wreak havoc on virtually any organ system. The hallmark of AIDS is profound immunosuppression, a state where the body's defenses are so weakened that they can no longer effectively fend off pathogens that would otherwise be easily controlled. This vulnerability stems from the relentless attack of HIV on CD4+ T cells, the orchestrators of the immune response. As HIV replicates and destroys these crucial cells, the immune system's ability to recognize and combat threats diminishes, opening the door for opportunistic infections to take hold. The severity of immunosuppression in AIDS is often classified based on the CD4 count. A CD4 count below 200 cells/μL is considered indicative of AIDS, and our patient's count of 15 cells/μL places him in the most severe category. This degree of immunosuppression not only increases the risk of opportunistic infections but also influences their severity and clinical course. Infections that might be mild or self-limiting in immunocompetent individuals can become life-threatening in those with AIDS. Opportunistic infections are a constant threat to individuals with AIDS, lurking in the shadows, ready to strike when the immune system falters. These infections are caused by microorganisms that are typically harmless to healthy individuals but can cause serious illness in those with weakened immune systems. The range of potential opportunistic infections is vast, encompassing bacteria, viruses, fungi, and parasites. In this case, the patient's advanced HIV disease manifested itself through the presence of oral candidiasis, commonly known as thrush. This fungal infection, caused by an overgrowth of *Candida albicans*, is a frequent harbinger of AIDS. The oral cavity, with its warm, moist environment, provides an ideal breeding ground for *Candida*. In immunocompromised individuals, the normal balance of microorganisms in the mouth is disrupted, allowing *Candida* to flourish unchecked. The clinical manifestations of oral candidiasis can range from mild to severe. In its mildest form, it may present as white patches on the tongue, inner cheeks, and roof of the mouth. However, in more severe cases, it can cause pain, difficulty swallowing, and even

extend into the esophagus, leading to esophagitis. In our patient, the presence of thrush likely contributed to his decreased appetite and subsequent weight loss, highlighting the interconnectedness of opportunistic infections and their impact on overall health. Beyond opportunistic infections, advanced HIV disease can also exert a systemic toll on the body. The chronic inflammation and immune dysregulation associated with HIV can lead to a cascade of metabolic and physiological disturbances, contributing to malnutrition, wasting, and other systemic manifestations. Malnutrition is a common complication of HIV/AIDS, often exacerbated by opportunistic infections, decreased appetite, and malabsorption. It can lead to a significant loss of body weight and muscle mass, impairing physical function and overall quality of life. In this case, the patient's weight loss of 12 kilograms is a testament to the profound impact of HIV on his nutritional status. Beyond malnutrition, advanced HIV disease can also manifest with a range of systemic symptoms, including fatigue, fever, night sweats, lymphadenopathy, and diarrhea. These symptoms can significantly impact the patient's daily life and contribute to a sense of debility and despair. This case serves as a poignant reminder of the interconnectedness of HIV complications. The patient's advanced HIV disease not only predisposed him to opportunistic infections like oral candidiasis but also contributed to his malnutrition and underweight status. The resulting immunosuppression and systemic inflammation likely played a role in the development and severity of his panuveitis and post-herpetic neuralgia. The complex interplay of these various complications creates a challenging clinical scenario, requiring a multidisciplinary approach to management. Addressing one complication without considering its impact on others can lead to suboptimal outcomes. For example, treating the patient's panuveitis with corticosteroids, while necessary to control inflammation, could potentially exacerbate his oral candidiasis or other opportunistic infections. Panuveitis, an inflammatory condition that engulfs the entire uvea and often extends to neighboring ocular structures, stands as a particularly formidable complication in the landscape of HIV/AIDS. The uvea, comprising the iris, ciliary body, and choroid, forms a critical part of the eye's middle layer, responsible for functions such as controlling light entry, focusing, and nourishing the retina. When inflammation ravages this intricate system, the consequences can be devastating, ranging from persistent pain and visual disturbances to irreversible vision loss and even blindness. The inflammatory cascade in panuveitis triggers a series of events that can profoundly disrupt ocular function. The iris, responsible for regulating the amount of light entering the eye, can become swollen and sluggish, leading to photophobia (sensitivity to light) and blurred vision. The ciliary body, which produces aqueous humor to nourish the lens and cornea, can become inflamed, potentially causing increased intraocular pressure and glaucoma. The choroid, a vascular layer that supplies oxygen and nutrients to the outer retina, can also be affected, leading to chorioretinal lesions and impaired retinal function. In the context of HIV/AIDS, the development of panuveitis is often linked to opportunistic infections, which exploit the weakened immune system to establish a foothold in the eye. Cytomegalovirus (CMV), toxoplasmosis, and herpes viruses are among the most common culprits. These pathogens can invade the uvea and trigger a robust inflammatory response, leading to the characteristic signs and symptoms of panuveitis. In this particular case, the patient's panuveitis was attributed to herpes zoster ophthalmicus (HZO), a reactivation of the varicella-zoster virus (VZV). This virus, responsible for chickenpox in childhood, remains dormant in sensory ganglia throughout life. However, in individuals with compromised immune systems, such as those with advanced HIV, VZV can reactivate and travel along nerve fibers to the skin and eye, causing a painful vesicular rash and ocular inflammation. HZO typically presents with a characteristic rash in the distribution of the ophthalmic division of the trigeminal nerve,

which supplies sensation to the forehead, scalp, eyelid, and cornea. The rash is often accompanied by ocular manifestations, which can range from mild conjunctivitis to severe panuveitis. In immunocompromised individuals, HZO tends to be more severe and is associated with a higher risk of complications. The patient's history of HZO, the presence of telltale scars on the right side of his face, and positive HSV-1 IgG serology provide compelling evidence for the diagnosis of HZO-related panuveitis. The severity of his ocular symptoms, including excruciating pain, blurred vision, and photophobia, underscores the profound impact of this condition on his visual function and overall quality of life. Panuveitis, regardless of its underlying cause, can have a devastating impact on visual function and quality of life. The inflammation can lead to a range of visual disturbances, including blurred vision, floaters, and decreased visual acuity. In severe cases, it can cause permanent vision loss or even blindness. Beyond its impact on vision, panuveitis can also cause significant pain and discomfort. The inflammation can irritate the nerves in the eye, leading to a constant or intermittent aching or burning sensation. This pain can be exacerbated by bright light, making it difficult for the patient to perform daily activities or even venture outdoors. The emotional and psychological toll of panuveitis should not be underestimated. The fear of vision loss, coupled with the physical discomfort and limitations imposed by the condition, can lead to anxiety, depression, and social isolation. In patients with HIV/AIDS, these challenges can be further compounded by the stigma and discrimination associated with the disease. The case of this 37-year-old man serves as a stark reminder of the importance of early recognition and intervention in cases of HIV-related panuveitis. Early diagnosis and prompt initiation of treatment can significantly improve visual outcomes and prevent irreversible vision loss.[11,12]

Beyond the acute onslaught of pain associated with herpes zoster ophthalmicus (HZO) and panuveitis, our patient grappled with the insidious specter of post-herpetic neuralgia (PHN), a chronic pain condition that can persist long after the visible manifestations of the HZO rash have subsided. This lingering torment, characterized by persistent or recurrent pain in the area once ravaged by the rash, often manifests as a burning, stabbing, or aching sensation, leaving an indelible mark on the patient's physical and emotional well-being. PHN arises from the complex interplay between the varicella-zoster virus (VZV) and the nervous system. During the acute phase of HZO, VZV reactivates from its dormant state in sensory ganglia and travels along nerve fibers, causing inflammation and damage. This neuronal injury can lead to persistent alterations in pain signaling pathways, resulting in chronic pain that outlasts the rash itself. The mechanisms underlying PHN are multifaceted and not fully understood. However, several factors are thought to contribute to its development. The inflammatory response triggered by VZV reactivation can damage nerve fibers, leading to abnormal nerve signaling and spontaneous pain generation. The persistent bombardment of pain signals from the affected area can lead to changes in the central nervous system, resulting in increased sensitivity to pain and a lowered pain threshold. The chronic pain of PHN can take a toll on the patient's mental health, leading to anxiety, depression, and sleep disturbances, which can further exacerbate the pain experience. In this case, the patient's PHN manifested as a continuous, stabbing pain in the right eye and face, radiating to the head. This relentless onslaught of pain, superimposed on the acute pain of panuveitis, created a symphony of suffering that significantly impacted his quality of life. The pain likely interfered with his ability to sleep, eat, and engage in daily activities, contributing to his decreased appetite and weight loss. The emotional and psychological burden of PHN should not be underestimated. Chronic pain can lead to a sense of hopelessness and despair, eroding the patient's resilience and coping mechanisms. In the context of HIV/AIDS, where patients already face numerous challenges, the added burden of PHN can be particularly overwhelming. The management of PHN is complex and often requires a

multimodal approach. Analgesics, such as acetaminophen or nonsteroidal anti-inflammatory drugs (NSAIDs), may provide some relief for mild to moderate pain. However, for more severe pain, stronger medications like opioids or anticonvulsants may be necessary. In this case, the patient was treated with gabapentin, an anticonvulsant medication that has proven effective in managing neuropathic pain, including PHN. Gabapentin works by modulating calcium channels and neurotransmitter release in the nervous system, thereby reducing pain signals. In addition to pharmacological interventions, non-pharmacological approaches can also play a role in managing PHN. These can include physical therapy, cognitive-behavioral therapy, and relaxation techniques. These approaches can help patients develop coping mechanisms, improve their quality of life, and reduce their reliance on medications. The experience of pain in HIV/AIDS is often complex and multifaceted. The physical pain of opportunistic infections and complications like PHN can be intertwined with the emotional and psychological pain of living with a chronic, stigmatized illness. Addressing the patient's pain requires not only pharmacological interventions but also a compassionate and holistic approach that recognizes the interplay of physical, emotional, and social factors.[13,14]

The case presented in this report serves as a poignant illustration of the multifaceted and devastating impact of HIV on human health, extending far beyond the well-known consequences of immunodeficiency. It paints a vivid picture of the physical, emotional, and social challenges faced by individuals living with advanced HIV disease, highlighting the intricate interplay between the virus, opportunistic infections, and the overall well-being of the patient. HIV's assault on the immune system sets off a cascade of complications that can affect virtually every organ system. Opportunistic infections, once held at bay by a robust immune response, seize the opportunity to invade and wreak havoc. In this case, the patient's oral candidiasis, a fungal infection commonly known as thrush, is a testament to this vulnerability. The white patches on his tongue, a hallmark of thrush, not only cause physical discomfort but also contribute to decreased appetite and subsequent weight loss. This malnutrition, in turn, further weakens the immune system, creating a vicious cycle of decline. Beyond opportunistic infections, HIV can also directly damage various organs and tissues. The virus can infiltrate the nervous system, leading to neurological complications such as HIV-associated dementia, peripheral neuropathy, and myelopathy. It can also affect the gastrointestinal tract, causing malabsorption and diarrhea, further contributing to malnutrition and weight loss. In this case, the patient's underweight status and history of weight loss likely reflect the combined impact of opportunistic infections and direct HIV-related damage to various organ systems. The physical toll of HIV is often accompanied by a heavy emotional and psychological burden. The diagnosis of HIV, particularly in its advanced stages, can trigger a range of emotions, including fear, anxiety, depression, and anger. The stigma associated with HIV can lead to social isolation, discrimination, and a loss of self-esteem. In this case, the patient's experience with panuveitis and post-herpetic neuralgia (PHN) likely added another layer of emotional distress. The chronic pain associated with these conditions can be debilitating, interfering with sleep, daily activities, and overall quality of life. The fear of vision loss, a potential consequence of panuveitis, can also create significant anxiety and uncertainty about the future. The cumulative impact of these physical and emotional challenges can be overwhelming, leading to a sense of hopelessness and despair. It is crucial for healthcare providers to recognize and address the psychological needs of patients with HIV/AIDS, providing support, counseling, and resources to help them cope with the emotional burden of their illness. HIV/AIDS is not only a medical condition but also a social phenomenon. The stigma and discrimination associated with HIV can have a profound impact on the patient's social well-being, relationships, and access to care. Fear of

rejection and judgment can lead to social isolation and withdrawal, further exacerbating the emotional challenges of living with HIV. In this case, the patient's history of unprotected sexual encounters with multiple partners highlights the complex social and behavioral factors that can contribute to HIV transmission. Addressing these factors is crucial for preventing new infections and reducing the spread of HIV. Healthcare providers play a vital role in combating HIV-related stigma and discrimination. By providing compassionate and non-judgmental care, educating patients about HIV prevention and treatment, and advocating for policies that protect the rights of people living with HIV, healthcare providers can help create a more supportive and inclusive environment for those affected by this disease. The multifaceted impact of HIV necessitates a holistic approach to management that addresses not only the physical manifestations of the disease but also the emotional, psychological, and social challenges faced by patients. This approach requires collaboration between various healthcare professionals, including infectious disease specialists, ophthalmologists, pain management specialists, mental health providers, and social workers. By providing comprehensive care that encompasses medical treatment, psychological support, and social services, healthcare providers can help patients with HIV/AIDS achieve optimal health outcomes and improve their overall quality of life. This holistic approach recognizes the interconnectedness of physical, emotional, and social well-being and strives to empower patients to live fulfilling lives despite the challenges of HIV.[15,16]

In the intricate tapestry of this patient's clinical presentation, the diagnosis of panuveitis, a severe and potentially sight-threatening inflammatory condition affecting the entire uvea and its surrounding structures, was inextricably linked to herpes zoster ophthalmicus (HZO). This connection was not merely a matter of conjecture but was firmly established through a convergence of compelling clinical evidence, including the patient's prior history of HZO, the presence of characteristic scars on the right side of his face, and the detection of positive HSV-1 IgG serology. Panuveitis, in essence, is an all-encompassing inflammation that engulfs the entire uvea, the middle layer of the eye composed of the iris, ciliary body, and choroid. This inflammatory maelstrom often extends its reach to adjacent ocular structures, such as the retina, vitreous, and optic nerve, creating a complex and potentially devastating clinical scenario. The uvea, a vital component of the eye's intricate machinery, plays a pivotal role in various functions, including controlling the amount of light entering the eye, focusing images on the retina, and providing nourishment to the surrounding tissues. When inflammation ravages this delicate system, the consequences can be far-reaching and profound. The iris, responsible for regulating the size of the pupil and controlling light entry, can become swollen and sluggish, leading to photophobia (an abnormal sensitivity to light) and blurred vision. The ciliary body, tasked with producing aqueous humor to nourish the lens and cornea, can become inflamed, potentially disrupting the delicate balance of fluid within the eye and leading to increased intraocular pressure and the dreaded complication of glaucoma. The choroid, a vascular layer that supplies oxygen and nutrients to the outer retina, can also fall victim to the inflammatory onslaught, resulting in chorioretinal lesions and compromised retinal function. In the context of HIV/AIDS, the development of panuveitis is often intertwined with opportunistic infections that exploit the weakened immune system. Cytomegalovirus (CMV), toxoplasmosis, and herpes viruses are among the most frequent offenders, their insidious invasion of the uvea triggering a cascade of inflammation that culminates in the hallmark signs and symptoms of panuveitis. In this particular case, the patient's panuveitis was unequivocally attributed to herpes zoster ophthalmicus (HZO), a reactivation of the varicella-zoster virus (VZV). This virus, infamous for causing chickenpox in childhood, establishes a lifelong presence within sensory ganglia, nerve cell clusters nestled near the spinal cord, where it lies dormant, awaiting an opportunity to strike anew. In

individuals with compromised immune systems, such as those battling advanced HIV, VZV can seize this opportunity, reactivating and embarking on a destructive journey along nerve fibers, ultimately reaching the skin and eye. This viral resurgence can manifest in two primary forms: herpes zoster, commonly referred to as shingles, characterized by a painful, blistering rash, and HZO, where the virus specifically targets the ophthalmic division of the trigeminal nerve, wreaking havoc on the eye and its surrounding structures. In individuals with robust immune systems, the reactivation of VZV is typically kept in check by a vigilant immune response. However, in the setting of advanced HIV infection, where the immune system is severely compromised, VZV can exploit this immunological chasm and unleash its full pathogenic potential. The resulting HZO can be particularly severe and is associated with a heightened risk of complications, including the dreaded specter of panuveitis. The ophthalmic division of the trigeminal nerve, the conduit for VZV's ocular invasion in HZO, innervates a vast network of ocular and periocular structures. This includes the cornea, the clear front surface of the eye the conjunctiva, the thin membrane covering the sclera (the white part of the eye) the sclera itself the iris, the colored part of the eye that controls the size of the pupil the ciliary body, responsible for producing aqueous humor and controlling lens shape the choroid, a vascular layer that nourishes the outer retina the retina, the light-sensitive layer at the back of the eye and the optic nerve, which transmits visual information from the retina to the brain. Given this extensive innervation, HZO can manifest with a diverse array of ocular findings, ranging from relatively mild keratitis (inflammation of the cornea) and conjunctivitis (inflammation of the conjunctiva) to the more severe and potentially sight-threatening complications of panuveitis and optic neuropathy. In this particular case, the patient's panuveitis presented with a constellation of symptoms that painted a vivid picture of widespread ocular inflammation. The severe pain, a relentless and stabbing sensation that radiated to the right side of his face and head, served as a constant reminder of the inflammatory inferno raging within his eye. The redness, a visible manifestation of dilated blood vessels in the conjunctiva and sclera, further underscored the intensity of the inflammatory response. Photophobia, an abnormal sensitivity to light, added another layer of discomfort to the patient's experience. This symptom arises from the inflammation of the iris, which controls the size of the pupil and regulates the amount of light entering the eye. In panuveitis, the iris can become swollen and sluggish, making it difficult to constrict the pupil in response to bright light, leading to a painful aversion to light. The blurred vision reported by the patient reflects the impact of the inflammation on the clarity of the ocular media and the function of the retina. The presence of a cloudy cornea, likely due to keratitis or corneal edema, further contributes to the visual impairment. The presence of characteristic scars on the right side of the patient's face served as a silent testament to his prior encounter with HZO. These scars, often described as dermatomal in distribution, trace the path of the affected nerve, providing a visual map of the virus's destructive journey. In this case, the scars on the right side of the face, corresponding to the distribution of the ophthalmic division of the trigeminal nerve, offered a crucial clue to the underlying etiology of the patient's panuveitis. The detection of positive HSV-1 IgG serology provided further evidence to support the diagnosis of HZO-related panuveitis. IgG antibodies, produced by the immune system in response to a prior infection, can persist for years, even after the infection has been cleared. The presence of HSV-1 IgG antibodies indicates a past exposure to the herpes simplex virus type 1 (HSV-1), a close relative of VZV. While HSV-1 is more commonly associated with oral and genital herpes, it can also cause ocular infections, including keratitis and uveitis. In this case, the positive HSV-1 IgG serology likely reflects the patient's prior HZO episode, as VZV and HSV-1 share antigenic similarities, leading to cross-reactivity in serological tests. The presence of these antibodies, in conjunction

with the clinical presentation and history of HZO, solidifies the diagnosis of HZO-related panuveitis.[17,18]

Herpes zoster ophthalmicus (HZO) stands as a formidable adversary, particularly in the context of immunocompromised individuals such as those battling advanced HIV infection. It represents a reactivation of the varicella-zoster virus (VZV), the same insidious agent responsible for the familiar childhood illness, chickenpox. Following the primary infection, typically characterized by an itchy, vesicular rash, VZV retreats into a state of latency, establishing a silent sanctuary within sensory ganglia, nerve cell clusters strategically positioned near the spinal cord. Within these neuronal havens, VZV lies dormant, its genetic material integrated into the host cell's DNA, evading detection by the immune system. This state of latency can persist for years, even decades, without causing any symptoms. However, in individuals with weakened immune systems, the delicate balance between the virus and the host can be disrupted, allowing VZV to reawaken and emerge from its slumber. This reactivation, often triggered by stress, illness, or medications that suppress the immune system, sets in motion a cascade of events that can have devastating consequences. The virus, once dormant, begins to replicate, traveling along nerve fibers to reach the skin and, in the case of HZO, the eye and surrounding structures. VZV reactivation can manifest in two primary ways: herpes zoster, commonly known as shingles, and HZO. Herpes zoster typically presents with a painful, blistering rash that follows the distribution of a single sensory nerve, often on one side of the body. The rash can be accompanied by fever, malaise, and other systemic symptoms. HZO, on the other hand, represents a more localized but potentially more severe manifestation of VZV reactivation. In HZO, the virus specifically targets the ophthalmic division of the trigeminal nerve, the nerve responsible for sensation in the forehead, scalp, eyelid, and cornea. This can lead to a range of ocular complications, from mild conjunctivitis to severe panuveitis and even vision-threatening conditions like optic neuropathy. In immunocompetent individuals, the immune system typically mounts a robust response to VZV reactivation, limiting the severity and duration of the illness. However, in individuals with compromised immune systems, such as those with advanced HIV infection, the immune response is blunted, allowing the virus to replicate unchecked and spread more extensively. The profound immunosuppression associated with advanced HIV infection creates a fertile ground for VZV reactivation. The virus can exploit the weakened immune defenses to establish a foothold in the eye, leading to a cascade of inflammation and tissue damage. The resulting HZO can be particularly severe and is associated with a higher risk of complications, including panuveitis, keratitis, scleritis, and optic neuropathy. The ophthalmic division of the trigeminal nerve, the conduit for VZV's ocular invasion in HZO, innervates a vast network of ocular and periocular structures. In its mildest form, it may present with conjunctivitis, characterized by redness, tearing, and a gritty sensation in the eye. However, in more severe cases, it can lead to keratitis, an inflammation of the cornea that can cause pain, photophobia, and blurred vision. Scleritis, an inflammation of the sclera, can also occur, causing severe pain and a bluish discoloration of the sclera. Perhaps the most feared complication of HZO is panuveitis, an inflammation that involves all three layers of the uvea - the iris, ciliary body, and choroid. Panuveitis can lead to a range of visual disturbances, including blurred vision, floaters, and decreased visual acuity. In severe cases, it can cause permanent vision loss or even blindness. Even after the acute phase of HZO has resolved, the patient may continue to experience pain in the affected area. This chronic pain condition, known as post-herpetic neuralgia (PHN), can persist for months or even years after the rash has healed. PHN is thought to result from damage to nerve fibers caused by the VZV reactivation. The pain can be severe and debilitating, significantly impacting the patient's quality of life.[19,20]

4. Conclusion

This case report underscores the critical importance of early HIV diagnosis and the prompt initiation of antiretroviral therapy (ART) in preventing the progression to AIDS and its associated complications, such as panuveitis and opportunistic infections. It also highlights the necessity of a multidisciplinary approach in managing complex cases involving multiple comorbidities. By addressing both the underlying HIV infection and its associated complications, healthcare professionals can significantly improve the quality of life and visual outcomes for patients living with HIV/AIDS.

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