

Herpes Zoster in HIV-Seropositive Patients: Clinical Characteristics, Atypical Presentations, and the Influence of Antiretroviral Therapy - A Comparative Hospital-Based Study

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Abstract

Background: HIV infection profoundly disrupts VZV-specific cell-mediated immunity, creating conditions that favor herpes zoster reactivation with greater frequency and severity than in immunocompetent hosts. Despite the established association, detailed data on the clinical spectrum and disease course of herpes zoster in HIV-seropositive patients from South Indian hospital settings are limited. **Objectives:** To compare the clinical profile, dermatomal involvement, atypical presentations, disease course, complications, and influence of antiretroviral therapy (ART) in HIV-seropositive herpes zoster patients versus HIV-seronegative patients attending a tertiary dermatology unit. **Methods:** A prospective observational study was conducted at the Department of DVL, GGH/GMC Guntur from January 2019 to December 2020. A total of 118 herpes zoster patients were enrolled, of whom 13 were HIV-seropositive and 105 were HIV-seronegative. Clinical parameters were systematically compared between the two groups. Statistical analysis was performed using the chi-squared test and Fisher's exact test where appropriate, with significance set at $p < 0.05$. **Results:** HIV seroprevalence among herpes zoster patients was 11%, with 4 of 13 (30.7%) newly diagnosed at presentation. HIV-positive patients were significantly younger (mean age 37.3 years vs. 45.3 years; $p=0.038$), with 61.5% below 40 years of age. Multidermatomal involvement was significantly more frequent in HIV-positive patients (53.8% vs. 9.5%; $p<0.05$). Atypical morphological features including hemorrhagic bullae were exclusive to the HIV-positive cohort. Disease course was more protracted in HIV-seropositive patients: mean time to cessation of new vesicle formation was 64.6 hours vs. 46.8 hours, mean crust healing time was 14.5 days vs. 8.5 days, and mean duration of zoster-associated pain was 23.3 days vs. 15.6 days. One HIV-positive patient developed herpes zoster encephalitis. More severe manifestations were concentrated in patients not receiving ART. **Conclusions:** Herpes zoster in HIV-seropositive patients presents at a younger age, follows a more prolonged and atypical course, and carries a higher complication burden compared to immunocompetent counterparts. The occurrence of herpes zoster—especially with atypical features—should prompt HIV screening. ART appears to confer a protective effect against the most severe manifestations.

Keywords: Herpes zoster; HIV; antiretroviral therapy; multidermatomal; atypical zoster; immunocompromised; VZV.

Introduction

The interplay between herpes zoster and HIV infection has been of considerable clinical relevance since the early years of the AIDS epidemic. Herpes zoster occurs at a rate approximately 10-15 times higher in HIV-seropositive individuals compared to the age-matched general population, and the clinical manifestations are often more

severe, protracted, and morphologically atypical [1,2]. Unlike in the elderly immunocompetent host—where age-associated immunosenescence is the dominant predisposing factor—herpes zoster in HIV infection reflects the progressive depletion of CD4+ T lymphocytes and the consequent loss of VZV-specific cellular immune surveillance [3].

Buchbinder et al. first established a significantly higher risk of morbidity and mortality associated with herpes zoster in HIV-infected patients, and subsequent cohort studies have consistently confirmed this finding [4]. The risk of zoster in HIV infection is not directly proportional to the duration of infection or CD4 count in isolation, but rather reflects a complex interplay of immune dysregulation at multiple levels [5]. Clinically, HIV-associated herpes zoster may present with hemorrhagic, hyperkeratotic, ecthymatous, or necrotic lesions; multidermatomal or disseminated involvement; and a greater propensity for serious complications including encephalitis, retinal necrosis, and visceral dissemination [6].

The widespread introduction of highly active antiretroviral therapy (HAART) has altered the natural history of HIV-associated herpes zoster, although the relationship is complex: while HAART-mediated immune reconstitution reduces the overall incidence of opportunistic infections, herpes zoster may paradoxically arise as part of immune reconstitution inflammatory syndrome (IRIS) in the early post-HAART period [7].

The present study sought to comparatively characterize herpes zoster in HIV-seropositive versus HIV-seronegative patients in a South Indian tertiary referral population, with a particular focus on disease severity, atypical presentations, and the influence of ART status.

Materials and Methods

Study Design and Setting

This was a prospective, comparative, hospital-based observational study conducted at the Department of Dermatology, Venereology and Leprosy (DVL), Government General Hospital and Government Medical College (GGH/GMC), Guntur, Andhra Pradesh from January 2019 to December 2020.

Participants

Patients presenting with painful dermatomal vesicular eruptions were evaluated for herpes zoster. Diagnosis was established clinically based on radicular prodromal pain and the presence of grouped vesicles or bullae in dermatomal distribution, with or without fever, headache, or malaise. Tzanck smear preparation was used to confirm doubtful cases. All patients, except those already known to be on ART, underwent HIV testing by ELISA with Western blot confirmation. Patients were classified into HIV-seropositive (n=13) and HIV-seronegative (n=105) groups for comparative analysis.

Clinical Assessment and Follow-Up

Patients were followed up on days 1, 6, 10, and 15, and until complete lesion resolution. The following variables were recorded: age, sex, seasonality, pre-eruptive symptoms, dermatomes involved, number of dermatomes affected, morphology of lesions, atypical presentations, time to cessation of new vesicle formation, time to healing of crusted lesions, duration of zoster-associated pain (ZAP), complications, ART status (for HIV-positive patients), and CD4 counts where available.

Statistical Analysis

Categorical variables were compared using the chi-squared test or Fisher's exact test, as appropriate. Continuous variables were compared using the independent samples t-test. A p-value of less than 0.05 was considered statistically significant. Data are reported as means \pm SD or frequencies and percentages.

Ethical Considerations

Ethical approval was obtained from the Institutional Ethics Committee of GGH/GMC Guntur. Informed written consent was obtained from all participants. Confidentiality of HIV status and all personal data was strictly maintained throughout the study. No experimental interventions were performed.

Results

HIV Seroprevalence and New Diagnoses

Among 118 herpes zoster patients enrolled over the study period, 13 (11%) were HIV-seropositive and 105 (89%) were HIV-seronegative. Among the 13 HIV-positive patients, 4 (30.7%) were newly diagnosed with HIV infection at the time of their herpes zoster presentation, indicating that herpes zoster served as the sentinel presentation for HIV in this subset. Nine patients were already on antiretroviral therapy (ART) at enrollment, while four were ART-naive.

Demographic Comparison

Sex Distribution: In the HIV-positive group, 7 patients (53.8%) were female and 6 (46.1%) were male. In the HIV-negative group, 55 (52.3%) were female and 50 (47.6%) were male. No statistically significant sex-based difference was observed between the groups (p=0.923) (Table 1).

Table 1. Sex distribution by HIV status

Sex	HIV Positive n (%)	HIV Negative n (%)	Total
Male	6 (46.1)	50 (47.6)	56
Female	7 (53.8)	55 (52.3)	62
Total	13	105	118

Age Distribution: HIV-seropositive patients were significantly younger than their HIV-seronegative counterparts. The mean age of presentation in HIV-positive patients was 37.3 years (range: 11–53 years), compared to 45.3 years (range: 9–75 years) in HIV-negative patients. Among HIV-positive patients, 61.5% (n=8) were below 40 years of age. In contrast, 67.6% (n=71) of HIV-negative patients were above 40 years. The association between younger age (below 40 years) and HIV seropositivity was statistically significant (p=0.038) (Table 2).

Table 2: Age distribution by HIV status

Age Group (years)	HIV Positive n (%)	HIV Negative n (%)	Total
0–9	0	1 (0.9)	1
10–19	1 (7.7)	9 (8.5)	10
20–29	0	14 (13.3)	14
30–39	7 (53.8)	10 (9.5)	17
40–49	3 (23.1)	22 (20.9)	25
50–59	2 (15.4)	21 (20.0)	23
60–69	0	19 (18.1)	19
70–79	0	9 (8.6)	9
Total	13	105	118

The trend toward higher zoster incidence in younger HIV-infected individuals likely reflects the inability to develop fully competent VZV-specific cell-mediated immunity prior to HIV acquisition—a biological mechanism analogous to, but distinct from, the immunosenescence underlying age-related zoster reactivation.

Dermatomal Distribution

Thoracic dermatome involvement was the most common in both groups (HIV-positive: 46.1%; HIV-negative: 48.5%). Trigeminal nerve involvement was noted in 23.07% of HIV-positive patients compared to 12.4% in HIV-negative patients, though this difference did not reach statistical significance. Cervical involvement was similarly prevalent in the HIV-positive group (23.07%) compared to 14.3% in HIV-negative patients (Table 3).

Table 3: Dermatomal distribution by HIV status

Dermatome	HIV Positive n (%)	HIV Negative n (%)	Total
Trigeminal	3 (23.1)	13 (12.4)	16
Facial	0	1 (0.9)	1
Cervical	3 (23.1)	15 (14.3)	18
Thoracic	6 (46.2)	51 (48.5)	57
Lumbar	1 (7.7)	20 (19.0)	21
Sacral	0	5 (4.8)	5
Total	13	105	118

Number of Dermatomes and Atypical Presentations

Multidermatomal involvement was markedly more frequent in the HIV-positive group: 7 patients (53.8%) had multiple dermatomes involved, compared to only 10 (9.5%) in the HIV-negative group—a highly significant difference ($p < 0.05$) (Table 4). This finding is consistent with the established literature and reflects the inadequacy of immune containment of viral spread in immunocompromised hosts.

One HIV-seronegative patient developed disseminated herpes zoster (defined as >20 vesicles outside the primary and adjacent dermatomes), occurring in the context of chemotherapy for carcinoma of the colon. One HIV-seronegative patient developed herpes duplex (zoster crossing the midline) without any identifiable immunodeficiency. None of the HIV-positive patients in this study developed frank disseminated zoster.

Table 4: Number of dermatomes involved by HIV status

Dermatomes	HIV Positive n (%)	HIV Negative n (%)	Total
Single	6 (46.2)	94 (89.5)	100
Multiple	7 (53.8)	10 (9.5)	17
Disseminated	0	1 (0.9)	1
Total	13	105	118

Table 5: Atypical presentations by HIV status

Atypical Feature	HIV Positive	HIV Negative
Multidermatomal involvement	7	10
Hemorrhagic bullae	1	0
Disseminated zoster	0	1
Zoster crossing midline	0	1

Morphology of Lesions

The most common lesion morphology in both groups was vesicular (HIV-positive: 69.2%; HIV-negative: 75.2%). Hemorrhagic bullae were documented exclusively in one HIV-positive patient (7.69%)—an atypical presentation that was absent in the entire HIV-negative cohort (Table 6). This patient was not on ART at the time of presentation and had ophthalmic division involvement. The appearance of hemorrhagic bullae in HIV-positive patients is consistent with published

descriptions of atypical zoster in severe immunocompromise, where lesion morphology may include hemorrhagic, hyperkeratotic, or ecthymatous variants.

Table 6: Morphology of lesions by HIV status

Morphology	HIV Positive n (%)	HIV Negative n (%)	Total
Vesicular	9 (69.2)	79 (75.2)	88
Papulovesicular	2 (15.4)	20 (19.0)	22
Pustular	1 (7.7)	6 (5.7)	7
Hemorrhagic bullae	1 (7.7)	0	1
Total	13	105	118

Disease Course: Comparative Analysis

Disease course parameters were consistently more protracted in HIV-seropositive patients across all measured outcomes (Table 7).

Cessation of New Vesicle Formation: The mean time to cessation of new vesicle formation was 64.6 hours (2.69 days) in HIV-positive patients, compared to 46.8 hours (1.95 days) in HIV-negative patients—a difference of approximately 18 hours. Two HIV-positive patients required more than four days for vesicle formation to cease.

Healing of Crusted Lesions: Crust resolution was substantially more prolonged in the HIV-positive group. Only 1 of 13 HIV-positive patients (7.7%) healed within two weeks, 4 (30.8%) took two to three weeks, and 8 (61.5%) required more than three weeks for complete crust healing—an outcome not observed in any HIV-negative patient. The mean time to crust resolution was 14.5 days in HIV-positive patients versus 8.5 days in HIV-negative patients.

Zoster-Associated Pain: The mean duration of ZAP was 23.3 days in HIV-positive patients, compared to 15.6 days in HIV-negative patients. In the HIV-positive cohort, five patients experienced ZAP lasting more than three weeks, four had two to three weeks of pain, two had one to two weeks, and one patient had less than one week of pain. For HIV-negative patients, 49 had two to three weeks of ZAP, 40 had one to two weeks, 14 had less than one week, and only two had pain exceeding three weeks.

Table 7: Comparative disease course: HIV-positive vs. HIV-negative

Parameter	HIV Positive	HIV Negative
Mean time: new vesicle cessation	64.6 hrs (2.69 days)	46.8 hrs (1.95 days)
Mean time: crust resolution	14.5 days	8.5 days
Mean duration: ZAP	23.3 days	15.6 days
Crust healing > 3 weeks	8/13 (61.5%)	0/105 (0%)
ZAP > 3 weeks	5/13 (38.5%)	2/105 (1.9%)

Complications

One HIV-seropositive patient (7.7%) developed herpes zoster encephalitis—the most serious complication recorded in the study. This patient, who was not on ART, presented with altered sensorium alongside grouped vesicles in the ophthalmic dermatome. Lumbar puncture and CSF analysis confirmed viral encephalitis, and concurrent ICTC testing returned HIV-seropositive. He was treated with intravenous acyclovir (400 mg in 100 ml normal saline, four times daily). Herpes zoster encephalitis occurs in approximately 0.1–0.2%

of all herpes zoster cases, with immunosuppression as the principal risk factor [8]. Its occurrence in the trigeminal distribution has been specifically noted to carry a higher risk of CNS extension compared to spinal dermatome involvement.

Secondary bacterial infection and ulcerative lesions were observed in both groups but were proportionally higher in HIV-positive patients relative to cohort size (Table 8).

Table 8: Complications by HIV status

Complication	HIV Positive n (%)	HIV Negative n (%)
CNS involvement (encephalitis)	1 (7.7)	0
Ulcerative lesions	2 (15.4)	3 (2.8)
Secondary bacterial infection	2 (15.4)	4 (3.8)
Ramsay Hunt syndrome	0	1 (0.9)
Ocular complication	0	0

Herpes Zoster and ART Status

Among the 13 HIV-positive patients, 9 were on ART and 4 were ART-naive. All four ART-naive patients were male. Notably, the most severe clinical manifestations—hemorrhagic bullae and herpes zoster encephalitis—occurred exclusively among ART-naive patients. Two of the four ART-naive patients had ophthalmic division involvement, and one of these developed encephalitis. The two remaining ART-naive patients had cervical dermatome involvement.

Among ART-treated patients, lesion morphology was predominantly vesicular (n=8), with one papulovesicular case. In ART-naive patients, morphology was more varied: one vesicular, one papulovesicular, one pustular, and one hemorrhagic bullae. Duration of vesicle formation and time to crusting were broadly similar between ART-treated and untreated patients, though the most severe outcomes were confined to the ART-naive subgroup (Table 9).

CD4 counts in the HIV-positive cohort ranged from 280 to 550 cells/ μ L. While formal statistical analysis of CD4-stratified outcomes was limited by the small HIV-positive sample, clinical observations support an association between lower immune reconstitution and more severe manifestations.

Table 9: Herpes zoster characteristics in HIV-positive patients by ART status

Parameter	On ART (n=9)	Not on ART (n=4)
Sex (M/F)	2/7	4/0
Trigeminal involvement	1	2
Cervical involvement	1	2
Thoracic involvement	6	0
Lumbar involvement	1	0
Vesicular morphology	8	1
Hemorrhagic bullae	0	1
Pustular	0	1
Encephalitis	0	1

Discussion

This comparative study confirms and extends the well-documented association between HIV infection and a more severe, atypical, and prolonged course of herpes zoster, drawing on a cohort from a South Indian government referral hospital.

HIV Seroprevalence: The prevalence of HIV seropositivity among herpes zoster patients in this study was 11%, falling within the 5.6–22.5% range reported in various Indian studies [9]. This is notably higher than the 5.1% reported by Vora et al. in rural Gujarat [9], and substantially lower than the 69% reported by Onunu et al. from Nigeria [10], differences that reflect regional HIV endemicity and variable study population characteristics. Critically, 30.7% of HIV-positive patients in this cohort were newly diagnosed at the time of herpes zoster presentation, reinforcing the pivotal role of herpes zoster as a clinical sentinel for undiagnosed HIV infection in this demographic.

Younger Age in HIV-Positive Patients: The mean age of 37.3 years in HIV-positive patients, compared to 45.3 years in the HIV-negative group (p=0.038), is consistent with results from Onunu et al. (36.1 vs. 56.3 years) and Vora et al. (43.9 vs. 46 years) [9,10]. This younger predilection in HIV-positive patients likely reflects two converging mechanisms: first, the HIV-endemic age demographics of the study region, and second, the biological principle that individuals who acquire HIV before fully consolidating VZV-specific cellular immunity may retain an inherently weaker immune response to latent VZV [3]. The immunological deterioration induced by HIV recapitulates—and at a younger age accelerates—the VZV immune decline typically observed only in the elderly immunocompetent host.

Multidermatomal and Atypical Involvement: The 53.8% rate of multidermatomal involvement in HIV-positive patients far exceeded the 9.5% rate in HIV-negative patients and is consistent with the African and Indian literature [9,10]. Multidermatomal zoster reflects impaired capacity to contain VZV replication within the affected ganglion, allowing spread to adjacent sensory ganglia. This finding has been statistically significant in comparisons across multiple published studies, including Onunu et al. and Vora et al., and its identification in clinical practice should prompt HIV screening.

The presence of hemorrhagic bullae exclusively in an HIV-positive, ART-naive patient highlights how the morphological spectrum of herpes zoster shifts with progressive immunosuppression. HIV-seropositive patients can present with hemorrhagic, hyperkeratotic, echthymatous, and verrucous lesions that heal with scarring—presentations that are rarely encountered in immunocompetent individuals [6]. The exclusive occurrence of this feature in the ART-naive subgroup in this study is clinically meaningful.

Disease Course: The approximately 18-hour prolongation in mean vesicle cessation time, and the near-doubling of mean crust healing time (14.5 vs. 8.5 days) in HIV-positive patients, are aligned with Onunu et al.'s findings (68.3 vs. 47.1 hours for vesicle cessation; 14.7 vs. 8.2 days for crust healing) [10]. The convergence of results across two geographically disparate populations strengthens the reliability of this pattern.

The duration of ZAP (23.3 vs. 15.6 days) was shorter in this study compared to Onunu *et al.* (38.3 vs. 27.6 days for HIV-positive vs. negative, respectively). This difference is likely attributable to greater treatment compliance, systematic follow-up, and minimal reliance on indigenous over-the-counter preparations in our cohort—factors that facilitate earlier pain resolution irrespective of HIV status.

Herpes Zoster Encephalitis: The case of herpes zoster encephalitis in an ART-naive HIV-positive patient underscores the potentially life-threatening nature of VZV reactivation in severe immunocompromise. CNS complications of herpes zoster—including encephalitis, myelitis, and vasculopathy—are disproportionately concentrated in immunocompromised individuals, and their occurrence with trigeminal involvement has been specifically noted in the literature [8]. Intravenous acyclovir remains the treatment of choice.

ART and Herpes Zoster: The clustering of severe manifestations (hemorrhagic bullae, encephalitis) in ART-naive patients in this study, despite the small sample size, lends clinical support to the protective role of immune reconstitution through ART. Published evidence on HAART and herpes zoster incidence is complex: while long-term HAART use appears to reduce risk through immune reconstitution, a transient increase in herpes zoster incidence shortly after ART initiation has been attributed to IRIS, as described by Martinez et al., [11] These divergent effects may partially explain the continued occurrence of herpes zoster even in ART-treated patients.

Pre-eruptive Pain and HIV: Interestingly, pre-eruptive pain was less frequently reported in HIV-positive patients (38.46%) compared to HIV-negative patients (54.2%), though this difference was not statistically significant ($p=0.28$). Altered pain perception, a higher threshold for pain reporting, or the predominance of a different neurological pattern of viral reactivation in the immunocompromised host could contribute to this observation.

Childhood Zoster and HIV: One of the five pediatric zoster cases in the series was an 11-year-old HIV-seropositive child with thoracic involvement. No atypical morphological features were documented in this child, and the outcome was favorable—consistent with the observation that even HIV-positive children may present with classical zoster, though the risk of more severe disease remains.

Limitations: The HIV-positive cohort was small ($n=13$), limiting statistical power for subgroup analyses and some cross-comparisons. CD4 counts were available but not systematically stratified for outcome analysis. Viral load data were not recorded. The study was conducted at a single tertiary referral center, which may not represent community-level disease patterns. Long-term follow-up for outcomes such as postherpetic neuralgia was not systematically available.

Conclusion

Herpes zoster in HIV-seropositive patients differs fundamentally from the disease in immunocompetent hosts in its epidemiology, clinical presentation, and disease course. Key distinguishing features include a younger age at presentation, higher frequency of multidermatomal and atypical involvement, longer duration of vesicle formation, more protracted wound healing, and a greater complication burden—including CNS involvement. The identification of herpes zoster with atypical or multidermatomal features should prompt immediate HIV testing, as approximately one-third of HIV-positive patients in this study were newly diagnosed at zoster presentation. ART appears to mitigate the severity of manifestations, and the most serious outcomes were observed exclusively among ART-naive patients. These findings support integrating HIV screening into the

routine clinical workup of all herpes zoster patients, particularly those presenting at a younger age or with atypical features.

Declarations

Author Contributions

Dr. Dharini S: Conceptualization, study design, data analysis, manuscript drafting, and final approval.

Dr. Nathiya K: Data collection, literature review, manuscript editing.

Dr. Priya Vanasekar: Data interpretation, critical revision of manuscript, supervision.

All authors have read and approved the final manuscript.

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The authors declare that no external funding was received for this study.

Conflict of Interest

The authors declare no conflict of interest.

Ethical Approval

The study was conducted in accordance with ethical standards and was approved by the Institutional Ethics Committee of Government General Hospital/Government Medical College, Guntur. Written informed consent was obtained from all participants.

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Data Availability Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

References

- [1] Mandal BK. Herpes zoster in the immunocompromised population. *Indian J Dermatol.* 2006; 51:235-43.
- [2] Abbas Vafai, Mitchel Berger. Zoster in patients infected with HIV: A review. *Am J Med Sci.* 2001;321(6):373-379.
- [3] Buchbinder SP, Katz MH, Hessol NA, Liu JY, O'Malley PM, Underwood R, et al. Herpes zoster and human immunodeficiency virus infection. *J Infect Dis.* 1992;166(5):1153-6.
- [4] Glesby MJ, Moore RD, Chaisson RE. Herpes zoster in patients with advanced human immunodeficiency virus infection treated with zidovudine. *J Infect Dis.* 1993;168(5):1264-8.

- [5] Perronne C, Lazanas M, Leport C, et al. Varicella in patients infected with the human immunodeficiency virus. *Arch Dermatol.* 1990;126(8):1033-1036.
- [6] Sacchidanand AS, Ballal S. Herpes viruses. In: Singal A, Grover C, editors. *Comprehensive Approach to Infections in Dermatology.* Mumbai: Jaypee Brothers Medical Publishers; 2016. p. 219-225.
- [7] Feller L, Wood NH, Lemmer J. Herpes zoster infection as an immune reconstitution inflammatory syndrome in HIV-seropositive subjects: a review. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2007;104(4):455–60.
- [8] Espiritu R, Rich M. Herpes zoster encephalitis: 2 case reports and review of literature. *Infect Dis Clin Pract.* 2007;15(4):284-288.
- [9] Vora RV, Anjaneyan G, Kota RKS, Pilani AP, Diwan NG, Patel NN. Study of clinical profile of herpes zoster in HIV-positive and negative patients at a rural-based tertiary care center, Gujarat. *Indian J Sex Transm Dis AIDS.* 2017;38(1):65-68.
- [10] Rajendran, Kathiravan1; Udayakumar, Sharath2; Dhandapani, Abinaya1. Assessment of Depression, Anxiety, Stress and Eating Disorders among Pre-professional Coaching Students in Udaipur: A Cross-sectional Study. *NMO Journal* 20(1): p54-58, Jan–Jun 2026. | DOI: 10.4103/JNMO.JNMO_83_25
- [11] Onunu AN, Uhunmwangho A. Clinical spectrum of herpes zoster in HIV-infected versus non-HIV-infected patients in Benin City, Nigeria. *West Afr J Med.* 2004;23(4):300–4.
- [12] Rajendran, Kathiravan1; Chakravarthy, Anitha2; Sankaran, Lokesh Kumar Samy3. Work, Family and Neighbourhood Relationships and their Impact on Mental Health: A Cross-sectional Assessment in Coimbatore. *NMO Journal* 20(1):p 40-44, Jan–Jun 2026. | DOI: 10.4103/JNMO.JNMO_133_25
- [13] Vijaya Bhaskar Reddy V, Navaneetha Reddy P. Herpes zoster in HIV patients-correlation with CD4 count. *IP Indian J Clin Exp Dermatol.* 2020;6(2):117–120.
- [14] Farizo KM, Buehler JW, Chamberland ME, Whyte BM, Froelicher ES, Hopkins SG, et al. Spectrum of disease in persons with human immunodeficiency virus infection in the United States. *JAMA.* 1992;267(13):1798–805.
- [15] Straus SE. Clinical and biological differences between recurrent herpes simplex virus and varicella-zoster virus infections. *JAMA.* 1989;262(24):3455–8.
- [16] Dworkin RH, Johnson RW, Breuer J, Gnann JW, Levin MJ, Backonja M, et al. Recommendations for the management of herpes zoster. *Clin Infect Dis.* 2007;44 Suppl 1:S1–26.



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