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RELATIONSHIP BETWEEN HERPES SIMPLEX VIRUS INFECTION AND HEARING LOSS

Introduction:

Herpes simplex viruses (HSV-1 and HSV-2) belong to the alpha herpesvirus family and can establish lifelong infections in sensory neurons of the peripheral nervous system. Following primary infection in the epithelium or mucosa, the virus infects the axon endings of sensory nerves in the dorsal root ganglia (DRG) or trigeminal ganglia.[1] Globally, an estimated 3.8 billion people under age 50 (64.2%) are infected with HSV-1, primarily associated with oral herpes, while approximately 519.5 million individuals aged 15–49 (13.3%) have HSV-2, which primarily causes genital herpes.[2] Although hearing loss in adults can arise from various factors, the potential role of viral infections, particularly HSV, in this condition is often overlooked. The World Health Organization has indicated that exposure to HSV-1 and HSV-2 may increase the risk of hearing loss throughout life. Notably, neonatal infections with HSV-1 are linked to a higher likelihood of encephalitis and hearing impairment compared to infections caused by HSV-2.[3] Despite these observations, large-scale studies exploring the association between HSV infection and hearing loss in adults are scarce. So, this study aimed to investigate the relationship between HSV infections and auditory impairment in American adults aged 20 to 49.

Methods:

This cross-sectional study utilized data from the National Health and Nutrition Examination Survey (NHANES) conducted during the 2011–2012 and 2015–2016 cycles, focusing on individuals aged 20–49 years. The final sample consisted of 4,608 participants excluding those outside this age range and those with incomplete audiogram or HSV test data.

Audiometric testing was performed in a controlled environment, measuring hearing levels at various frequencies using an audiometer. Hearing impairment was defined as a pure-tone average (PTA) of 20 dB or higher in at least one ear. HSV serologic testing involved blood samples analyzed for antibodies against HSV-1 and HSV-2, with results categorized as positive or negative.

To enhance the validity of findings, several covariates were adjusted for, including sociodemographic factors (age, race/ethnicity, education), health metrics (BMI, noise exposure, use of hearing aids), and medical history related to conditions affecting hearing. Using weighted multivariate regression, the association between HSV (types 1 and 2) infection and hearing loss was analysed.

Results:

The NHANES analysis included 4,608 adults aged 20–49 years, with a balanced gender distribution (50.4% male) and diverse racial representation (18.6% Hispanic, 60.1% non-Hispanic White, 11.8% non-Hispanic Black). Missing data were noted for some variables, such

as hypertension (n=252) and BMI (n=17), and significant associations were found between herpes simplex virus (HSV) infections and hearing impairment. Individuals with HSV-1 infection had a higher prevalence of hearing impairment (OR 1.4, 95% CI 1.1 to 1.9) and greater grades of hearing loss (OR 1.4, 95% CI 1.1 to 1.8). Those coinfected with HSV-1 and HSV-2 showed even higher risks (OR 1.6, 95% CI 1.1 to 2.3). Pure tone averages (PTA) also increased, with HSV-1 infected individuals showing a β of 1.04 (95% CI 0.33 to 1.75) and coinfected individuals of about β of 1.88 (95% CI 1.04 to 2.71).

The relationship between HSV-2 and hearing loss was significant for grades of hearing loss (OR 1.4, 95% CI 1.0 to 2.0) and PTA (β 1.17, 95% CI 0.45 to 1.89), although this diminished with additional confounders (Table 1). Notably, the association was stronger in younger adults (ages 20–34: OR 2.1, 95% CI 1.4 to 3.3) and those with a higher BMI (OR 1.8, 95% CI 1.1 to 2.8). These results highlight the substantial impact of HSV infections on hearing health, particularly in specific demographic groups.

Table 1: Association between Hearing Impairment and HSV Infections

	Model 1	Model 2	Model 3
Hearing impairment			
HSV-1	1.4 (1.1 to 1.9)	1.4 (1.0 to 1.8)	1.4 (1.0 to 1.9)
HSV-2	1.4 (1.0 to 2.0)	1.4 (1.0 to 2.0)	1.3 (0.9 to 2.0)
Coinfected	1.6 (1.1 to 2.3)	1.6 (1.1 to 2.2)	1.4 (0.9 to 2.1)
Grade			
HSV-1	1.4 (1.1 to 1.8)	1.3 (1.0 to 1.8)	1.5 (1.1 to 2.0)
HSV-2	1.4 (1.0 to 2.0)	1.4 (1.0 to 1.9)	1.4 (1.0 to 2.0)
Coinfected	1.6 (1.2 to 2.2)	1.5 (1.1 to 2.1)	1.5 (1.0 to 2.1)
Pure-tone average			
HSV-1	1.04 (0.33 to 1.75)	0.91 (0.22 to 1.61)	0.84 (0.18 to 1.50)
HSV-2	1.17 (0.45 to 1.89)	1.06 (0.35 to 1.78)	0.97 (0.35 to 1.59)
Coinfected	1.88 (1.04 to 2.71)	1.73 (0.90 to 2.55)	1.47 (0.66 to 2.27)

Model 1 adjusted for age, sex, and race/ethnicity
Model 2 included education in addition to age, sex, and race/ethnicity
Model 3 further adjusted for BMI, concurrent conditions (cardiovascular diseases, hypertension, respiratory diseases, diabetes, cancer)

Discussion:

This study found a significant inverse relationship between hearing ability and HSV infection, particularly with HSV-1, even after adjusting for confounders like age, sex, race/ethnicity, BMI, and comorbidities. The association was stronger in younger individuals and those with lower BMI. These findings align with a previous study showing an increased hearing loss in children with HSV-1.[3] In this study, it was found that 80% of ears affected by HSV-1 in the pediatric population had mild hearing loss,contrasting with a previous study that reported a 50% prevalence of moderate hearing loss.[3]

Conclusion:

This study demonstrated an association between HSV-1 infection and coinfections of HSV-1 and HSV-2 with a higher prevalence of hearing impairment compared to those without infections individuals. Notably, this association was stronger in younger individuals and those with lower BMI.

HSV-1 and HSV-2 coinfections are associated with increased hearing impairment, particularly in younger individuals and those with lower BMI.

Reference:

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ASSESSMENT OF RISK FACTORS AND CHARACTERISTICS OF SCARRING IN PATIENTS WITH MONKEYPOX INFECTION

Introduction:

The monkeypox virus (MPOX) is a zoonotic, double-stranded DNA virus belonging to the Orthopox virus genus, presenting skin manifestations like smallpox but typically less severe and self-limiting, with lower case-fatality rates. Prior to May 2022, MPOX was primarily a zoonotic and traveler-associated infection, with limited cases outside endemic regions. However, since May 2022, over 79,000 cases have emerged across 103 non-endemic countries. Human-to-human transmission occurs primarily through direct contact with skin lesions and respiratory droplets, with fomite transmission being less common. The incubation period ranges from 5 to 24 days,[1] followed by a prodromal phase of 2 to 4 days, during which patients may experience systemic symptoms such as fever, malaise, and lymphadenopathy. The characteristic lesions progress from whitish papules on an erythematous base to umbilicated pustules. Despite the WHO's declaration of a public health emergency in response to the outbreak from 2022 to 2023, cicatricial sequelae—such as hypertrophic, keloid, atrophic scars, and pigmentary changes—have been reported anecdotally in both the current outbreak and endemic cases. The risk of scarring following lesion resolution remains poorly defined, with limited objective data available due to insufficient long-term follow-up.[2] So, this study aimed to investigate the risk factors associated with scarring in MPOX patients and to characterize

the nature of scarring in individuals treated at the sexually transmitted infection (STI) unit of a tertiary hospital.

Methods:

This prospective cohort study included confirmed MPOX cases from a tertiary referral hospital, diagnosed via PCR testing of skin, oropharyngeal, urethral, and anal samples using the VIASURE Monkeypox Virus reverse transcription (RT)-PCR kit. Data on epidemiological, clinical, and microbiological factors were collected from medical records and follow-up consultations at 12–15 months post-diagnosis to assess scarring and its impact on quality of life, using a 0 to 10 scale. Statistical analysis using SPSS version 28 involved the Shapiro–Wilk test for normality, the student's t-test or Mann–Whitney U test for numerical variables, and Pearson's chi-squared or Fisher's exact tests for categorical variables, with significance set at $P < 0.05$.

Results:

Among 72 individuals with confirmed MPOX diagnoses, 40 patients were analyzed after excluding 32 due to lack of follow-up. Of these, 19 (47.5%) developed scarring, while 21 (52.5%) did not. The mean age was significantly higher in the scar-free group (42.5 years) compared to the scarred group (36.2 years), and all scarred patients were male, whereas most non-scarred patients were female (95%) (Table 1). The study found that 28.6% of scar-free patients had HIV, compared to 21.1% in the scarred group. There was no significant difference in vaccination status. Systemic symptoms were observed in 67% of the non-scarred and 74% of the scarred patients, with significant differences in initial presentation, 57% of non-scarred patients had systemic symptoms initially, while 47% of scarred patients presented with skin lesions first. Scar characteristics included a mean of 19.7 scars per patient, with the genital area being the most affected. Bacterial superinfection occurred in 15.7% of scarred patients, and severe pain was reported in 21%. The quality-of-life impairment for scarred patients averaged 5.2 out of 10, with 36.8% reporting substantial impairment. Overall, the study highlights significant differences in demographic, clinical, and quality of life outcomes related to scarring after MPOX infection.

Patient information	Patients without scars	Patients with scars
Age (range)	42.5 (22–69)	36.2 (23–49)
Male	20 (95)	19 (100)
Female	1 (5)	0 (0)
HIV positive	6 (28.6)	4 (21.1)
HIV negative	15 (71.4)	15 (78.9)
Vaccination	8 (38.1)	2 (10.5)
Non-vaccination	13 (61.9)	17 (89.5)
Arthromyalgia	8 (38)	8 (42)
Systemic symptoms	14 (66.7)	14 (74)
Fever	8 (38)	12 (63)
Asthenia	9 (43)	11 (58)
Headache	6 (29)	6 (32)
Adenopathies	12 (57)	13 (68)
Pharyngitis	6 (29)	4 (21)

Urethritis	4 (19)	2 (11)
Proctitis	5 (24)	3 (16)

Table 1. Epidemiological and clinical characteristics of patients with and without scars

Discussion:

The current study highlighted an intriguing finding regarding smallpox vaccination and monkeypox outcomes there was a lower proportion of vaccinated patients who developed scarring, although it was not statistically significant. This contrasts with previous research suggesting that vaccination correlates with milder disease and reduced scarring risk.[3,4] Additionally, the study found that HIV infection did not significantly increase the risk of scarring in monkeypox patients, diverging from earlier literature that associated HIV with a higher likelihood of extensive necrotic lesions and subsequent scarring.[5]

Conclusion:

This prospective study, the longest follow-up on MPOX patients to date, reveals that scarring is a more frequent complication than previously reported, significantly affecting quality of life and psychological well-being. Young patients with risk factors are particularly susceptible to severe sequelae, underscoring the need for vaccination as primary prevention. Close monitoring and early intervention for central facial and genital lesions, especially when they present initially, are essential to reduce long-term complications.

Reference:

1. Prasad S et al. A dermatologic assessment of 101 mpox (monkeypox) cases from 13 countries during the 2022 outbreak: Skin lesion morphology, clinical course, and scarring. J Am Acad Dermatol. 2023 May;88(5):1066-1073.
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MPOX scarring is more common than previously recognized, especially in young patients, highlighting the critical need for vaccination and early intervention.

ASSOCIATIONS BETWEEN CLINICAL CHARACTERISTICS AND BIOLOGIC THERAPY SWITCHING IN PSORIASIS TREATMENT

Introduction:

Psoriasis is a chronic, non-communicable, immune-mediated disorder characterized by painful, disfiguring, and disabling symptoms, primarily affecting the skin and nails with a papulosquamous morphology. The complex genetic architecture of psoriasis contributes to its variability in presentation and severity.[1] While global prevalence was estimated at 2-3%, epidemiological data vary, with rates from 0.91% to 8.5% in adults and 0.0% to 2.1% in children.[2] Although there were several medications available, biologics have greatly improved treatment outcomes for many psoriasis patients, allowing for remission or low disease activity. However, some may need to switch therapies due to ineffectiveness or side effects, and the risk factors for these changes are not well understood. Comorbidities like arthritis and metabolic syndrome can complicate treatment, and biomarkers such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and neutrophil-to-lymphocyte ratio (NLR) are important for assessing disease activity. Understanding the factors behind therapy changes is essential for optimizing patient care.[3] So, this study aimed to investigate the association between clinical characteristics and laboratory data that may predict the necessity for switching biologic therapies in psoriasis management.

Methods:

This retrospective study reviewed patients with psoriasis vulgaris (PsV), psoriatic arthritis (PsA), or generalized pustular psoriasis (GPP) treated with biologics. Eligible patients initiated first-line biologic therapy and were observed for over 24 weeks, categorized by the number of biologic switches: none, one, two, or more. Baseline data included age, sex, body mass index (BMI), and disease duration. Laboratory parameters, including CRP, ESR, NLR, MLR, and PLR, were collected within 3 months before assessment. Comorbidities were evaluated, and disease severity was assessed using Psoriasis Area Severity Index (PASI) scores. Descriptive statistics summarized continuous variables as median (IQR) whereas categorical variables as counts (%). The Kruskal-Wallis and Fisher exact tests were used for analysis, with drug-specific survival rates calculated via the Kaplan–Meier method, considering $p < 0.05$ as significant.

Results:

In this study of 285 patients, including 135 with psoriasis vulgaris (PsV), 137 with psoriatic arthritis (PsA), and 13 with generalized pustular psoriasis (GPP), 125 patients (43.9%) maintained their initial biologic therapy, while 56.1% switched at least once, primarily from Tumor Necrosis Factor inhibitors (TNFi) to other biologics. Those who switched more frequently had significantly higher PASI scores at therapy initiation (12.3 vs. 6.6, p=0.016) and a higher prevalence of concurrent arthritis (64.1% vs. 43.2%, p=0.012). Dyslipidaemia was less common in frequent switchers (12.8% vs. 25.6%, p=0.036). Patients with two or more switches also had a higher NLR of 2.65 compared to 2.30 in those with no switches (p=0.011). CRP and ESR levels tended to be higher in patients with two or more switches (Table 1). Regarding drug survival, median durations were significantly longer for IL-17 inhibitors (54 months) and IL-23 inhibitors (55 months) compared to TNFi (25 months, p=0.007), highlighting the greater long-term efficacy of IL-17i and IL-23i in these patients.

Number of switches	None	Once	Two or more	p-value
Patients, No.	125	82	78	
Concurrent arthritis	54 (43.2%)	39 (47.6%)	50 (64.1%)	0.012
Comorbidities, Dyslipidaemia	32 (25.6%)	23 (28.0%)	10 (12.8%)	0.036
NLR, median	2.3 (1.60–3.27)	2.55 (1.91–3.45)	2.65 (2.03–3.97)	0.011
CRP, median	0.12 (0.05–0.42)	0.09 (0.05–0.31)	0.18 (0.05–0.57)	0.517
ESR, median	13.5 (7–25)	16 (7–29)	24 (8.25–37)	0.169
MLR, median	0.23 (0.18–0.30)	0.25 (0.19–0.36)	0.25 (0.09–0.31)	0.109
PLR, median	14.18 (10.98–19)	16.1 (12.14–20.94)	15.77 (12.42–21.79)	0.137

Table 1: Clinical Characteristics and Baseline Biomarkers by Number of Switches

Discussion:

The current study found that a higher NLR was significantly associated with increased rates of biologic switching, underscoring the impact of systemic inflammation on treatment outcomes. In contrast, previous studies identified demographic factors like age and sex as contributors to switching rates, suggesting a shift in focus toward inflammatory markers in treatment adherence.[4] Patients on IL-17 and IL-23 inhibitors had longer drug survival than those on TNF inhibitors, underscoring the greater efficacy and safety of newer biologics. This supports existing literature and emphasizes the need for personalized treatment strategies based on the patient's inflammatory profile.[5]

Conclusion:

This study demonstrated a correlation between elevated PASI scores, NLR, and comorbid arthritis with the frequency of biological treatment switching. A treatment strategy that integrates clinical presentation and inflammatory biomarkers may be crucial for optimizing patient management in psoriasis.

Elevated PASI scores, NLR, and arthritis correlate with increased biologic treatment switching, emphasizing the need for integrated psoriasis management.

Reference:

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CASE REPORT ON ICHTHYOSIS PREMATURITY SYNDROME

Introduction:

Ichthyosis prematurity syndrome (IPS) is a rare autosomal recessive disorder characterized by a clinical trial of premature birth, ichthyosis, and neonatal asphyxia. The condition was attributed to mutations in the SLC27A4 gene (solute carrier family 27 member 4), which encodes fatty acid transport protein 4 (FATP4).[1] Histopathological examinations of the skin in IPS typically reveal hyperkeratosis, parakeratosis, and a thickened stratum corneum. These dermatological changes, in conjunction with respiratory distress, serve as key indicators for early diagnosis. Mutations in the SLC27A4 gene disrupt lipid metabolism, impair keratinocyte differentiation, and compromise the skin barrier function.[2] This report presented a case of a female neonate diagnosed with IPS, highlighting the importance of early recognition and comprehensive management in improving patient outcomes.

Case Presentation:

A female neonate was born prematurely at 30 weeks of gestation, weighing 1400 grams, measuring 42 cm in length, and having a head circumference of 30 cm. Upon delivery, she exhibited respiratory distress, including tachypnea, grunting, and retractions, which necessitated immediate resuscitation and mechanical ventilation. The infant's skin was notably thick, white, and desquamating, resembling vernix caseosa, particularly on her trunk and extremities (Figure 1). There were no significant findings in her cardiovascular, gastrointestinal, or neurological assessments, but the family history indicated consanguinity with no known cases of ichthyosis.

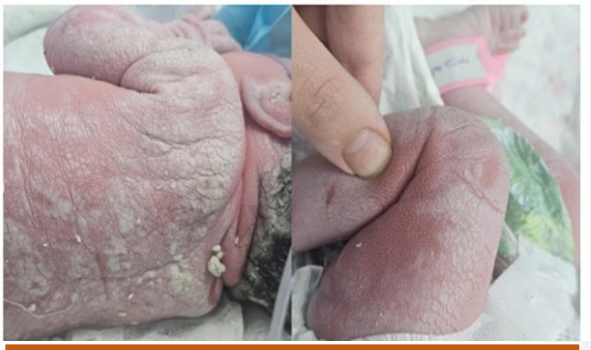

Figure 1: Patient displaying thick, white, caseous scales on various body areas

Initial evaluations included a chest X-ray that showed bilateral diffuse infiltrates consistent with respiratory distress syndrome (RDS) (Figure 2), alongside blood tests revealing normal complete blood count and corrected electrolyte levels. A skin biopsy showed hyperkeratosis, parakeratosis, and a thickened stratum corneum, which were key for diagnosing Ichthyosis Prematurity Syndrome (IPS). Genetic testing confirmed mutations in the SLC27A4 gene, and genetic counseling discussed the implications of the autosomal recessive inheritance pattern. A multidisciplinary team managed her care, and employed continuous positive airway pressure (CPAP), surfactant therapy, regular emollient applications, gentle skin care, and enteral feeding. Over a period of two weeks, she gradually reduced her reliance on respiratory support, with notable improvement in her skin condition. After 4 weeks in the NICU, she was discharged and continued follow-up care with dermatology and genetics. At one year of age, she demonstrated normal growth and development but required ongoing antihistamines and topical treatments for severe dry skin, significant itching, and recurrent skin infections.

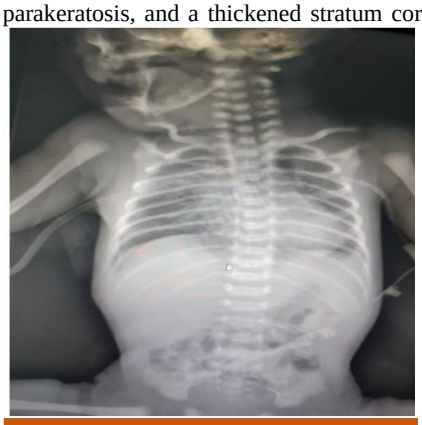

Figure 2: X-ray (AP view) demonstrating bilateral diffuse infiltrates consistent with respiratory distress syndrome (RDS)

Discussion:

This study found that the neonate's birth weight was 1400 grams, which was below the average for ichthyosis with a predilection for skin IPS. The reported median birth weight for this condition was 2060 grams, with a range of 1760–2480 grams. Family history indicated consanguinity, but there was no known history of ichthyosis, suggesting an autosomal recessive inheritance pattern consistent with previous research.[3] This case highlighted the significance of considering IPS in neonates with premature birth and distinctive skin findings. Early identification and genetic testing are crucial for accurate diagnosis and effective management. Furthermore, with a 25% risk of IPS in future pregnancies, genetic counseling is essential for affected families to help them understand potential risks and make informed decisions regarding prenatal care.[4]

Conclusion:

Ichthyosis Prematurity Syndrome (IPS) is a rare genetic disorder that necessitates prompt diagnosis and a multidisciplinary management approach for optimal outcomes. This case highlighted the importance of considering IPS in premature neonates with distinctive skin features and respiratory distress. Comprehensive care, including genetic testing and supportive therapies, was essential for the effective management and prevention of complications.

Early recognition and multidisciplinary management of Ichthyosis Prematurity Syndrome (IPS) in premature neonates are crucial for preventing complications and optimizing outcomes.

Reference:

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