

Association between Facial Nerve Palsy and Herpes Zoster infection in patients with immunocompromised state

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ABSTRACT:

Objective: To examine the relationship between patients' immunocompromised state and clinical severity and polycranial nerve involvement and recovery outcomes in groups of patients with Ramsay Hunt Syndrome (RHS) at tertiary hospitals of Sindh, Pakistan.

Methodology: This retrospective cohort study included 136 confirmed RHS cases during January to December 2024. Patients were classified as immunocompromised (n=41) and immunocompetent (n=95). Data included demographics, comorbidities, initial House-Brackmann facial nerve grade, polycranial nerve involvement, timing of initiation of antiviral therapy, and 6-month recovery. Statistical analysis consisted of Mann-Whitney U, chi-square, and odds ratios.

Results: Patients with immunocompromised RHS were found to be significantly older (mean age, 61.2 vs 48.6 years; $p<0.001$) and had a higher prevalence of diabetes (58.5% vs 14.7%; $p<0.001$) and HIV (22% vs 0%; $p<0.001$). Severe initial facial nerve palsy (House-Brackmann Grade V/VI) was more prevalent in immunocompromised patients compared to immunocompetent patients (68.3% vs 36.8%; $p<0.001$). Polycranial nerve involvement was encountered in 56.1% of immunocompromised patients, whereas polycranial nerve involvement was identified in only 27.4% of immunocompetent patients (OR 2.71; $p=0.001$). Initiating antiviral therapy early (≤ 72 hours) in immunocompromised patients significantly improved rates of complete recovery at six months (57.1% vs 25.0% with delayed therapy; $p=0.029$).

Conclusion: Immunocompromised RHS patients are older, have more severe facial palsy, have a greater risk of polycranial nerve involvement, and recover at a slower pace, particularly if treated late.

Key words: Ramsay Hunt Syndrome, Immunocompromised, facial palsy, Herpes Zoster.

Cite as: Shaikh SP, Mahar GS, Mushtaq AB, Ansari NA, Waheed A, Ashraf F. Association between Facial Nerve Palsy and Herpes Zoster infection in patients with immunocompromised state. J Muhammad Med Coll. 2025; 16 (1) pp-76-79

Introduction:

Facial nerve palsy (FNP) presents a substantial problem in the clinical setting, restricting functional capacities, inflicting esthetic damage to the individual, as well as significant psychological dissatisfaction with the cosmetic and functional results. Although Bell's palsy (idiopathic FNP) is the most common type of facial paralysis, a large proportion of FNP cases have identified causes, with herpes zoster infection being one of the most significant.¹ Ramsay Hunt Syndrome (RHS), usually defined as the trio of acute FNP, ipsilateral auricular vesicular rash, and otalgia, is linked to the reactivation of latent varicella-zoster virus (VZV) in the geniculate ganglion of the facial nerve.^{2,3} Herpes zoster (HZ), a clinical manifestation of reactivated varicella zoster virus (VZV), is well known to be associated

with reduced cell-mediated immunity, particularly in elderly people.⁴ However, immunocompromised states such as human immunodeficiency virus (HIV) infection, diabetes mellitus, hematological malignancies, solid organ transplantation (e.g., liver, kidney), autoimmune diseases requiring immunosuppression, and chronic corticosteroid use significantly increase the risk of HZ and alter its natural history.^{5,6} These patients had a larger viral incidence, more severe and longer cutaneous lesions, a higher risk of neurologic sequelae (including postherpetic neuralgia), and a higher risk of disseminated HZ than the immunocompetent population.⁷ RHS is a neurological consequence of HZ, thus one would predict a similar tendency in the immunocompromised host. Data suggest that immunocompromised HZ patients have a higher relative risk of developing cranial neuropathies (including FNP) than immunocompetent HZ patients.^{8,9} Furthermore, in immunocompromised hosts, the clinical presentation of RHS may be more severe, with higher frequencies of multiple cranial nerve involvement (polycranial neuritis), more severe initial facial paralysis (House-Brackmann grades), and possibly poorer recovery.^{10,11} The underlying pathophysiology is most likely caused by poor VZV-specific T-cell immunity, which allows for uncontrolled viral replication and increased damage to neuronal structures inside the facial nerve as well as surrounding structures.¹²

While the association between immunocompromise and HZ severity has been established, and RHS has been recognized within the immunocompromised population, there remains a relative lack of strong, population specific data describing the unique association, clinical spectrum, and prognostic factors of RHS in the immunocompromised population in our region.¹³ Most of the literature has focused on immunocompetent populations and HZ complications in general, with only a few studies directly comparing RHS

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Received: 4.7.2025 . Revised: 23.7.2025

Accepted: 21.08.2025 Published online 22.10.2025

features and outcomes in immunocompromised and immunocompetent patients.^{10,14} Further understanding the unique relationship between HZ related facial nerve palsy and underlying immunocompromise is important because it aids in the early diagnosis and recognition of RHS versus Bell's Palsy (which has implications for management), it informs prognostication, aids in the intensity and length of antiviral and adjunctive therapies (e.g. corticosteroids), and it defines preventive approaches such as vaccination for HZ in groups of immunocompromised patients.^{15,16}

The goal of this study is to explore the specific association of facial nerve palsy related to herpes zoster infection (Ramsay Hunt Syndrome) and distinct immunocompromised states. In this study researcher examined the associated parental immunocompromise in patients presenting with RHS, compare initial clinical severity and pattern of cranial nerve involvement, and compare recovery outcomes of facial nerve palsy between immunocompromised and immunocompetent patients diagnosed with RHS from our cohort.

Objective:

The primary goal of this study was to look into the prevalence of immunocompromised states (such as diabetes, HIV, immunosuppressive medicine, and cancer) in Ramsay Hunt Syndrome (RHS) patients at a tertiary care hospital. Secondary objectives includes determining the degree of early facial nerve paralysis (House-Brackmann grading system) in RHS patients with immunocompromised states, rate of multiple cranial nerve involvement and to investigate the link between delayed beginning of antiviral medication (>72 hours after the onset of symptoms) and recovery in immunocompromised individuals with RHS.

Methodology:

This retrospective cohort study was conducted from January 2024 to December 2024 at the Department of Neurology/ENT at Sir Syed Medical College Hospital, Karachi, and Muhammad Medical College Hospital, Mirpurkhas, that resulted patients both from urban and rural Sindh. Using non-probability consecutive sampling technique 136 confirmed patients enrolled. The sample size was justified to achieve an estimated statistical power of 80% and a level of significance (α) of .05 to be statistically powered to detect a difference between immunocompromised (IC) and immunocompetent (IC) patient populations. Adult patients of either gender, with complete medical record and with 6 months follow diagnosed as RHS (facial nerve palsy + ipsilateral herpes zoster rash in cranial nerve VII distribution) were included.

Neurology registries, electronic medical records, and clinic follow-up files from participating centers were among the many sources from which data was collected. The following variables were considered for each confirmed RHS case: the degree of facial nerve involvement, which was graded using the House-Brackmannings (Grades I-VI) at diagnosis, as advised by the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS, 2020); clinically determined evidence of polycranial nerve involvement, which was supported by MRI brain; and the recovery outcome at six months, which was also assessed using the House-Brackmann grading system, with full recovery being a final grade of I or II. The duration between the rash onset and the initiation of antiviral medication, as well as the type of immunocompromised state, were also variables of interest. These were ascertained through laboratory testing or clinical documentation. A comprehensive analysis of patient characteristics, clinical presentation, and treatment

outcomes is made possible by this method of data collection

Statistical analysis was performed using SPSS v28.0. Descriptive statistics and prevalence ratios were used to report the frequency of immunocompromised states. The Mann-Whitney U test was used to compare the severity of facial nerve versus optic nerve cranial nerve damage (median, IQR) between groups. Chi-square or Fisher's exact tests were used to evaluate associations between immunocompromised and polycranial nerve involvement, as well as odds ratios. To analyzed relationship between time to initiation of antiviral therapy and recovery outcome, Spearman's correlation was performed. For all analyses, a p-value of less than 0.05 was considered significant.

Results:

Among 136 cases of RHS, 41 (30.1%) were immunocompromised, while 95 (69.9%) were immunocompetent. The mean age of the cohort was 52.3 ± 14.7 years. Immunocompromised patients were significantly older (61.2 ± 10.4 years) when compared to immunocompetent patients (48.6 ± 14.1 years; $p < 0.001$). The distribution of gender between groups was not statistically significant, with males comprising 54.4% of the overall cohort. Diabetes mellitus was reported by 27.9% of all patients and was significantly more common in immunocompromised patients (58.5% vs. 14.7%; $p < 0.001$). HIV infection was only reported among immunocompromised patients (22.0%), and the use of immunosuppressive therapy was also more common in immunocompromised patients (24.4% vs. 7.4%; $p = 0.006$). The mean time from symptom onset to diagnosis was longer in immunocompromised patients (5.1 ± 2.3 days) than immunocompetent patients (3.8 ± 1.8 days; $p < 0.001$) as shown in table no 1.

Table No1: Demographic and clinical characteristics of RHS patients (N=136)

Characteristic	N=136	Immunocompromised (n=41)	Immunocompetent (n=95)	Statistical Test	p-value
Age (years), M $\pm$ SD	52.3 $\pm$ 14.7	61.2 $\pm$ 10.4	48.6 $\pm$ 14.1	t=5.41	<0.001
Gender, n (%)				$\chi^2=0.38$	0.538
Male	74 (54.4)	23 (56.1)	51 (53.7)		
Female	62 (45.6)	18 (43.9)	44 (46.3)		
Comorbidities, n (%)					
Diabetes mellitus	38 (27.9)	24 (58.5)	14 (14.7)	$\chi^2=26.91$	<0.001
HIV	9 (6.6)	9 (22.0)	0 (0)	Fisher's exact	<0.001
Immunosuppressive therapy	17 (12.5)	10 (24.4)	7 (7.4)	$\chi^2=7.65$	0.006
Symptom onset to diagnosis (days), M $\pm$ SD	4.2 $\pm$ 2.1	5.1 $\pm$ 2.3	3.8 $\pm$ 1.8	t=3.72	<0.001

Table No 2 Initial severity grades of Facial Nerve palsy

House-Brackmann Grade	Immunocompromised (n=41), n (%)	Immunocompetent (n=95), n (%)	Mann-Whitney U test	p value
Grade III	2 (4.9)	18 (18.9)	U=1,210	<0.001
Grade IV	11 (26.8)	42 (44.2)		
Grade V	23 (56.1)	31 (32.6)		
Grade VI	5 (12.2)	4 (4.2)		

Severity of facial nerve paralysis, shown in table no 2, was assessed as per House-Brackmann grading system. Initial severity was found more severe in immunocompromised and those who were immunocompetent. Among immunocompromised patients 56.1% were rated Grade V, and 12.2% were rated Grade VI. While among immunocompetent patients; 18.9% rated as Grade III and 44.2% as

Grade IV; 32.6% had Grade V, and just 4.2% had Grade VI facial paralysis. The Mann-Whitney U test identified significantly greater severity of initial facial nerve involvement in immunocompromised patients compared to immunocompetent patients ($U = 1,210$, $p < 0.001$). Polycranial nerve involvement was found in 56.1% (23/41; 95% CI: 40.5-70.7) of immunocompromised RHS patients and 27.4% (26/95; 95% CI: 19.1-37.5) of immunocompetent patients, with a statistically significant difference ($\chi^2 (1) = 10.24$, $p = 0.001$) suggesting additional cranial nerve involvement among immunocompromised patients. Additionally, immunocompromised patients had significantly higher odds of polycranial nerve involvement (OR: 2.71; 95% CI: 1.49-4.91) compared to immunocompetent patients as shown in table 3.

Table No 3: Polycranial Nerve Involvement in Ramsay Hunt Syndrome.

Group	n (%)	95% CI	Statistics
Immunocompromised	23 (56.1)	[40.5, 70.7]	$\chi^2 (1) = 10.24$, $p = .001$
Immunocompetent	26 (27.4)	[19.1, 37.5]	OR = 2.71 [1.49, 4.91]

As shown in table 4, the timing of initiation of antiviral therapy was statistically associated with recovery outcomes in immunocompromised RHS patients. Patients who were treated with antiviral therapy within 72 hours of symptom onset had a significantly greater rate of complete recovery, with 57.1% (12/21; 95% CI: 36.5-75.5; House-Brackmann grade I-II) at six months, compared to 25.0% (5/20; 95% CI: 11.2-46.9) of patients who were treated after 72 hours of symptom onset. Likewise, incomplete recovery was significantly more common in patients who were treated with antiviral therapy beyond 72 hours (75.0%; 15/20) compared to patients treated within 72 hours (42.9%; 9/21). The difference in recovery was statistically significant ($\chi^2 (1) = 4.76$, $p = 0.029$), and antiviral therapy administered beyond 72 hours was associated with significantly lower odds of complete recovery (OR = 0.26; 95% CI: 0.07-0.95).

Table 4 Association between Delayed Antiviral Therapy and Recovery Outcomes in Immunocompromised RHS Patients (n = 41)

Antiviral Therapy Timing	Complete Recovery n/(%)	Incomplete Recovery n/(%)	Total	Statistics
≤72 hours	12/21 (57.1) [95% CI: 36.5-75.5]	9/21 (42.9) [95% CI: 24.5-63.5]	21	$\chi^2 (1) = 4.76$ $p = .029$
>72 hours	5/20 (25.0) [95% CI: 11.2-46.9]	15/20 (75.0) [95% CI: 53.1-88.8]	20	OR = 0.26 [95% CI: 0.07-0.95]

Discussion:

The relationship between immunocompromised status and the clinical features, severity, and recovery from Ramsay Hunt Syndrome (RHS) is made clearer by this retrospective cohort study. Immunocompromised patients are more likely to experience severe illness, polycranial nerve involvement, and a less favorable recovery, especially when antiviral therapy is delayed, according to the research. The most prevalent underlying diseases in this study were HIV+, diabetic mellitus, and immunosuppressive medication, which combined to impair the immune systems of 30.1% of patients. This aligns with broader research that identifies immunosuppression, HIV infection, advanced age, and diabetes as known risk factors for herpes zoster

(HZ) and its neurological complications.^{3,6-9} In keeping with global studies demonstrating age-related declines in cell-mediated immunity and the likelihood of HZ reactivation and consequences, the immunocompromised group's mean age was significantly higher.^{3,6} Immunocompromised RHS patients have higher rates of diabetes (58.5%) and HIV status (22%), which is consistent with both local and global trends. This indicates that immunocompromised patients are more vulnerable to VZV reactivation and severe consequences.⁵⁻⁷ This data emphasizes the necessity of focused preventative measures, such immunization, for these vulnerable groups.^{15,16}

The study found that the two groups' initial facial nerve severity differed significantly. Severe facial paralysis (House-Brackmann Grade V or VI; 68.3%) was more common in immunocompromised patients than in immunocompetent patients (36.8%). This is in line with other research showing that immunocompromised condition is linked to worse neurological presentation and a greater degree of nerve damage in RHS.^{10,11} Furthermore, immunocompromised patients had a considerably higher risk of polycranial nerve involvement (56.1%) than immunocompetent patients (27.4%). This observation aligns with the established propensity of immunocompromised hosts to encounter more severe or widespread VZV infection and involvement of the cranial nerve.¹² The weakened VZV-specific T-cell response, which probably permits more extensive and stronger viral multiplication and extension from the facial nerve to other cranial nerves, is another factor contributing to more severe nerve involvement in immunocompromised patients.¹²

Initiating antiviral treatment early (<72 hours) was significantly related to improved recovery outcomes in immunocompromised RHS patients (57.1% complete recovery, 25.0% for delayed treatment, $p = 0.029$). This is consistent with guidelines and several meta-analyses highlighting the importance of timely initiation of antiviral therapy to maximize recovery and minimize the chance of irreversible nerve damage.^{14,15} The increase in the risk of poor outcomes associated with delayed treatment in this cohort of immunocompromised patients highlights the importance of a timely diagnosis and treatment, which is being emphasized in recent recommendations from international experts.¹³

A direct comparison of RHS between immunocompromised and immunocompetent patients has rarely been the focus of prior research. Our results corroborate those of NK Ghezta et al.¹⁰ who found that immunosuppression and hyperglycemia are separate risk factors for poor recovery in RHS. Goo B¹¹ additionally revealed initial severity and timing of treatments as key predictive outcomes for functional recovery from facial nerve palsy. Our study's findings that immunocompromised patients had much higher probabilities of polycranial involvement lend credence to the need for a more thorough evaluation of the cranial nerve in this population.

Clinical Implications:

- Prompt detection and antiviral therapy (within 72 hours) are essential, particularly for immunocompromised people who can experience a more severe progression of the illness.
- Because polycranial nerve involvement is common in this population, a comprehensive cranial nerve assessment (with imaging as necessary) ought to be standard procedure.
- More extensive rehabilitation and tighter monitoring may be necessary since immunocompromised individuals are

more likely to experience incomplete recovery and persistent consequences.

• According to international guidelines, vaccination against HZ should be seriously be considered for the suitable immunocompromised populations.

Limitation of the study:

The design of the current study and data collection procedure may be a limiting factor regarding generalizability. The definition and severity of immunocompromised may differ significantly from center to center. Future multicenter prospective studies may help to better understand prognostic factors, risk stratification and impact of early intervention and prophylactic vaccination for RHS particularly in immunocompromised patients.

Conclusion:

The immunocompromised patients with Ramsay Hunt Syndrome are older, have more severe facial nerve palsy, are at increased risk for polycranial involvement; generally, shows slow incomplete recovery particularly when antiviral therapy was delayed. This indicates importance of early recognition, prompt treatment, thorough assessment, and appropriate prevention measures, including vaccination for at-risk populations.

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Ghulam Shabir Mahar	Methodology Manuscript Draft
Allah Bux Mushtaq	Manuscript Draft
Noor Alam Ansari	Data Collection and Analysis Approval of final version
Abdul Waheed	Final Manuscript writing
Farhan Ashraf	Proof reading and Grammar