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The Role of Herpes Simplex Virus in the Etiology of Bell's Palsy

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ABSTRACT

Background: Bell's palsy, the most common cause of acute peripheral facial paralysis, is strongly associated with reactivation of latent herpes simplex virus type 1 (HSV-1) in the geniculate ganglion. Through PRISMA-guided review we evaluated the molecular and clinical evidence for this association.

Methods: A systematic search of major electronic databases (PubMed, ScienceDirect, Google Scholar) was conducted for studies published up to December 2025. The PICO framework guided inclusion criteria, focusing on studies assessing HSV infection in Bell's palsy patients compared to controls, and reporting on HSV-1 DNA detection or antiviral efficacy. Molecular methods including PCR and serological assays were acceptable. The quality was assessed using the Newcastle-Ottawa Scale and the Cochrane Risk of Bias tool.

Results: Twenty-three studies met the inclusion criteria. Molecular analyses consistently detected HSV-1 DNA in the geniculate ganglia and peripheral fluids of patients, with detection rates up to 79% in endoneurial fluid. Epidemiological data confirmed a strong association (odds ratio: 6.49; 95% CI: 5.96-7.05). However, evidence regarding the efficacy of adjuvant antiviral therapy combined with corticosteroids remained inconsistent. Major systematic reviews demonstrated no significant clinical benefit for the general population, with combination therapy increasing recovery by a maximum of 7%.

Conclusion: Strong molecular and epidemiological evidence supports HSV-1 reactivation as a major etiological factor in Bell's palsy. However, the routine clinical benefit of concomitant antiviral therapy remains unproven for the overall population. Future management should prioritize diagnostic stratification using viral PCR testing and severity classification to identify virus-induced cases benefiting most from targeted antivirals.

Keywords: Bell's Palsy, Herpes Simplex Virus, HSV-1, Etiology, Systematic Review

Introduction

Bell's palsy (BP) is defined as an acute, unilateral weakness or paralysis of the peripheral facial nerve of unknown cause, making it the most common form of acute cranial nerve mononeuropathy (1). The condition has an annual incidence rate of approximately 20 to 30 cases

per 100,000 population (2). The hallmark of the disorder is a rapid decline in facial muscle strength, a change that often results in considerable physical limitations and emotional strain. While the label “idiopathic” suggests the cause is unknown, the prevailing medical understanding now recognizes that the pathogenesis primarily involves inflammation and swelling of the seventh cranial nerve within the narrow bony Fallopian canal (3).

The clinical presentation of Bell's palsy is distinctive and often alarming for the patient. Symptoms typically worsen rapidly, reaching their peak within 48 to 72 hours of onset. The three most common clinical signs are as follows:

1) Inability to close the eye on the affected side (lagophthalmos), 2) drooping of the mouth corner, and 3) flattening or disappearance of the nasolabial fold (2). Beyond these physical manifestations, the sudden alteration in the facial appearance frequently carries a significant psychological burden, potentially leading to social withdrawal, anxiety, or depression. This psychological impact is particularly pronounced in younger patients or those whose professions rely heavily on facial expression and communication (2). Clinicians typically assess the degree of paralysis using the House-Brackmann grading scale, which categorizes function from Grade I (normal) to Grade VI (total paralysis), providing a standardized framework for monitoring recovery and evaluating treatment efficacy.

An understanding of the anatomical vulnerability of the facial nerve is essential to comprehending the pathophysiology of Bell's palsy. The facial nerve traverses the Fallopian canal, a narrow and tortuous bony passage within the temporal bone. This canal is the longest and most constricted bony pathway traversed by any nerve in the human body. Critically, at its narrowest point, the meatal segment, the nerve occupies approximately 90% of the available space within the canal (3,4). This anatomical constraint means that even minimal inflammation or edema in this confined space can produce significant compression, compromise blood supply, and result in axonal damage. This mechanical vulnerability is the central anatomical factor that transforms a mild inflammatory event into a clinically significant neuropathy.

Currently, the most compelling etiological theory implicates the reactivation of herpes simplex virus type 1 (HSV-1) as the primary trigger for this inflammatory process (5, 6). It is well established that HSV-1 establishes lifelong latency in sensory ganglia, particularly the trigeminal ganglion; however, strong evidence indicates that it also reactivates within the geniculate ganglion, which houses the cell bodies of the sensory components of the facial nerve (7,8). The biological mechanism underlying viral latency and reactivation is complex. Following primary infection, which frequently occurs during childhood, the virus migrates via retrograde axonal transport to the sensory ganglia and enters a latent phase. Various triggers, including emotional stress, physical trauma, immunosuppression, or cold exposure, can reactivate the virus. Once reactivated, HSV-1 replicates and travels anterograde along the nerve, producing local inflammation, edema, and the compressive neuropathy characteristic of Bell's palsy (4).

The hypothesis linking HSV-1 to Bell's palsy was first proposed by McCormick in 1972, who observed that the clinical features of the condition were consistent with a herpetic infection of the facial nerve (4). Since that seminal observation, numerous molecular and clinical investigations have sought to confirm this association. The detection of HSV-1 DNA in the endoneurial fluid and posterior auricular muscle of patients with Bell's palsy constitutes the most direct molecular evidence to date (7). Nevertheless, the potential contribution of other viruses, particularly varicella-zoster virus (VZV), which causes Ramsay Hunt Syndrome, must be acknowledged. While the clinical presentations

may overlap, Ramsay Hunt Syndrome is typically distinguished by more severe otalgia and the presence of vesicles in the external auditory canal (8,9).

This systematic review was designed to critically evaluate the available evidence on the association between HSV and Bell's palsy, providing a clear and comprehensive analysis conducted in accordance with the PRISMA 2020 guidelines. The PICO framework was used to structure the review, with the central research question: "Is there evidence of HSV infection (I) in patients with Bell's palsy (P) compared with healthy individuals or other populations (C), and what impact does this have on clinical outcomes (O)?" By synthesizing findings from a broad range of studies, we aimed to provide a clear, evidence-based understanding of the role of HSV in the etiology of Bell's palsy, with important implications for the diagnosis and management of this condition. Additionally, it identifies gaps in the current literature and suggests directions for future research, particularly in the context of personalized treatment strategies based on viral diagnostic markers.

Methods

Protocol and Registration

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guidelines. The review protocol was not formally registered in a public registry such as PROSPERO.

Eligibility Criteria

The PICO framework guided the development of the research question and eligibility criteria as follows:

Population (P): Patients diagnosed with Bell's palsy, defined as idiopathic acute peripheral facial paralysis.

Intervention/Exposure (I): Evidence of HSV infection or reactivation, as detected through molecular methods including PCR, serological assays (e.g., IgG/IgM antibody titers), or clinical indicators of active herpetic infection.

Comparison (C): Control groups comprising healthy individuals, patients with facial paralysis of established causes (such as Ramsay Hunt Syndrome), or different therapeutic approaches (i.e., combination of antivirals and corticosteroids versus corticosteroids alone).

Outcome (O): The strength of association between HSV and Bell's palsy, the prevalence of detectable HSV-1 DNA in affected individuals, the therapeutic efficacy of antiviral agents, and the rates of clinical recovery following treatment.

We included original research articles, systematic reviews, and meta-analyses published in peer-reviewed, open-access journals. Studies were limited to those involving human subjects and published in English. Case reports, editorials, letters to the editor, and conference abstracts were excluded.

Search Strategy

A systematic search was performed across three major electronic databases: PubMed, ScienceDirect, and Google Scholar. The search covered articles published up to December 2025, with no earlier date restriction. The search strategy combined Medical Subject Headings (MeSH) terms and free-text keywords related to "Bell's palsy," "herpes simplex virus," "HSV-1," "idiopathic facial paralysis," and "etiology."

Additionally, the reference lists of included articles and relevant systematic reviews were manually screened for additional eligible studies (snowballing method) to ensure comprehensive literature coverage.

Study Selection

All identified records were entered into a reference management software, and duplicates were removed. Two independent reviewers (university lecturers) screened the titles and ab-

stracts of articles against the eligibility criteria. The full texts of articles deemed potentially relevant were retrieved and assessed for final inclusion decisions. Disagreements were resolved through discussion and consensus. The study selection process is illustrated using the PRISMA 2020 flow diagram (Figure 1).

Data Extraction and Synthesis

Data were extracted from the included studies using a standardized form, capturing: study characteristics (author, year, design, sample size), patient demographics, method of HSV detection (e.g., PCR target, sample type, serological assay), prevalence of HSV-1 detection, and key findings regarding the association with Bell's palsy or treatment efficacy.

Given the anticipated heterogeneity in study designs and outcome measures, the analysis of findings was conducted as a narrative synthesis, with tables and figures used to summarize key data. Where possible, quantitative data on the prevalence of HSV-1 detection and treatment outcomes were also reported.

Quality Assessment

The methodological quality and risk of bias of the included studies were assessed using validated and widely recognized tools. The Newcastle-Ottawa Scale (NOS) was used for observational studies (cohort and case-control studies), evaluating selection, comparability, and outcome domains. The Cochrane Risk of Bias tool was applied to randomized controlled trials (RCTs), assessing randomization, blinding, attrition, and reporting biases. Studies were categorized as having low, moderate, or high risk of bias, and this assessment was considered during the synthesis of evidence.

Results

Study Selection

The systematic search identified a total of 1,250 records. After the removal of duplicates, 890 records were screened by title and abstract. Of these, 55 full-text articles were assessed for eligibility, resulting in the inclusion of 23 open-access studies, comprising original research, systematic reviews, and meta-analyses, that met the predefined PICO criteria. The detailed selection process is illustrated in the PRISMA 2020 flow diagram (Figure 1).

Evidence of HSV-1 DNA Detection

Molecular studies utilizing PCR techniques provide the most direct and compelling evidence for the HSV-BP link, consistently demonstrating viral reactivation in affected patients:

Geniculate Ganglion and Endoneurial Fluid

The landmark study by Murakami et al. (7) detected HSV-1 genomes in the endoneurial fluid of the facial nerve in 79% of Bell's palsy patients (11 of 14 patients), a finding that strongly implicated HSV-1 as a major etiological agent. Subsequent post-mortem studies, notably by Arbusow et al. (10), confirmed the presence of HSV-1 DNA in the geniculate ganglia, the established site of viral latency for the facial nerve.

Peripheral Fluids (Saliva)

The detection of HSV-1 DNA in peripheral fluids, such as saliva, serves as a non-invasive marker for active viral shedding and reactivation. Furuta et al. (6) reported a significantly higher prevalence of HSV-1 shedding in the saliva of Bell's palsy patients (50%) compared to healthy controls (19%). Lazarini et al. (11) similarly found a 29% positive PCR rate for HSV-1 in the saliva of acute Bell's palsy patients. These findings collectively support the occurrence of active viral replication during the acute phase of the disease.

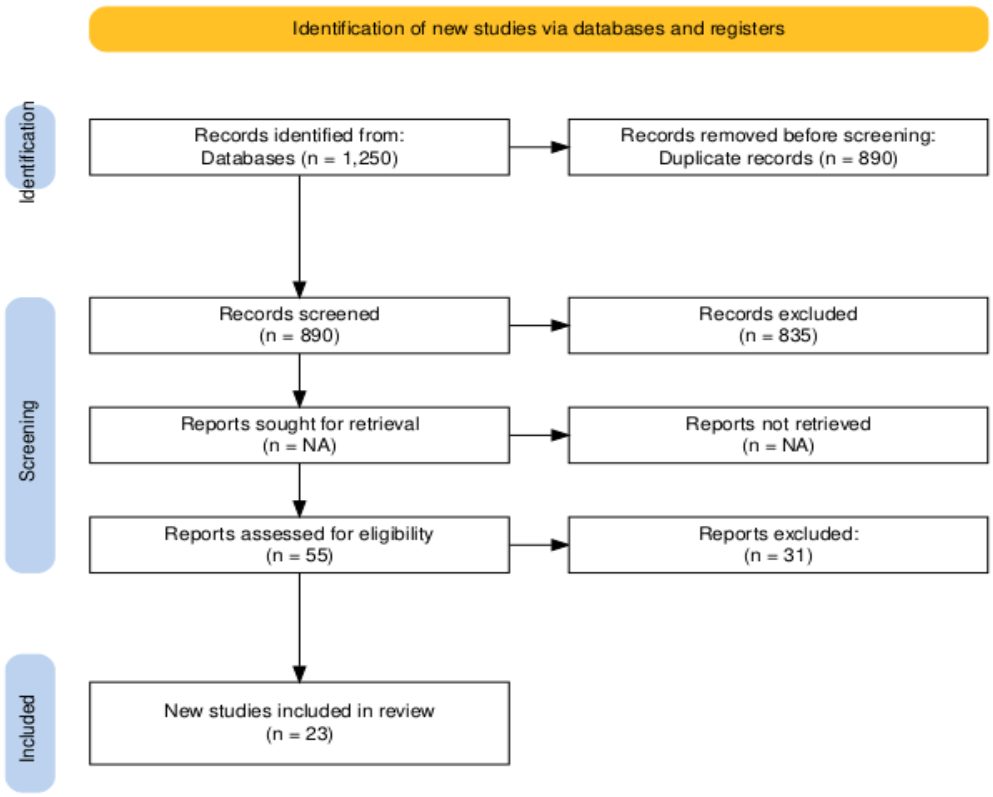

Figure 1: PRISMA 2020 flow diagram

Association and Risk

Epidemiological data further substantiate the causal role of HSV. A recent analysis based on real-world data by Neckel et al. (12) demonstrated that HSV had the strongest association with Bell's palsy compared with other risk factors, reporting an odds ratio of 6.49 (95% confidence interval: 5.96–7.05; $P < 0.001$), indicating that individuals with evidence of HSV infection are approximately 6.5 times more likely to develop Bell's palsy than those without. Furthermore, approximately one-third of all Bell's palsy cases are attributable to HSV-1 reactivation (13).

Efficacy of Antiviral Treatment

The clinical debate centers on the efficacy of adding antiviral agents (e.g., valacyclovir, acyclovir) to standard corticosteroid therapy for Bell's palsy.

Inconclusive Evidence: Authoritative systematic reviews, including the Cochrane review by Gagyor et al. (14), have generally concluded that there was no clear evidence of significant benefit from adding antivirals to corticosteroids in terms of complete recovery rates in the overall Bell's palsy patient population. Similarly, Gronseth et al. (15) found that combining antivirals with corticosteroids did not increase the likelihood of facial nerve function recovery by more than 7% compared to corticosteroids alone.

Subgroup Benefit: In contrast, some meta-analyses have suggested a possible additive benefit of combination therapy, particularly in patients with more severe paralysis or those treated very early in the course of the disease (16, 17). Turgeon et al. (18) tested this hypothesis by stratifying patients according to initial paralysis severity (House-Brackmann Grade IV–VI) and suggested that this subgroup of pa-

tients with more severe paralysis may benefit from the addition of antiviral drugs to corticosteroid treatment. This finding indicates that the efficacy of antiviral therapy may be dependent on disease severity and the timing of treatment initiation. A comprehensive summary of the key findings from the included studies is presented in Table 1.

Table 1: Summary of key findings on HSV-1 and Bell's Palsy

Study (yr)	*Focus*	*Method/Sample*	*Key Finding*	*Citation*
Murakami et al. (1996)	Etiology/Detection	PCR/Endoneurial Fluid	HSV-1 genomes detected in 79% of Bell's palsy patients.	(7)
Furuta et al. (1998)	Reactivation	PCR/Saliva	Shed HSV-1 in 50% of Bell's palsy vs. 19% in controls.	(6)
Arbusow et al. (2000)	Latency	PCR/Ganglia	Confirmed HSV-1 presence in geniculate ganglia.	(10)
Goudakos et al. (2009)	Treatment Efficacy	Systematic Review/RCTs	Suggested a possible benefit of adding antivirals to corticosteroids.	(16)
Gronseth et al. (2012)	Treatment Efficacy	Systematic Review/RCTs	Antivirals plus steroids did not increase recovery by >7%.	(15)
Lazarini et al. (2015)	Reactivation	PCR/Saliva	29% of acute Bell's palsy patients positive for HSV-1 in saliva.	(11)
Gagyor et al. (2019)	Treatment Efficacy	Cochrane Review/RCTs	No clear evidence of benefit from adding antivirals for complete recovery.	(14)
Boukhvalova et al. (2022)	Attribution	Review/Synthesis	Estimated about one-third of cases attributable to HSV-1.	(13)
Neckel et al. (2025)	Risk Factors	Real-World Data Analysis	HSV showed the strongest association (OR: 6.49; 95% CI: 5.96–7.05).	(12)
Zhu et al. (2024)	Treatment Efficacy	Systematic Review/RCTs	Ongoing debate on efficacy of combined therapy.	(19)

Discussion

The findings of this systematic review demonstrate a strong and biologically plausible link between the reactivation of HSV-1 and the etiology of Bell's palsy. Molecular evidence, particularly the frequent detection of HSV-1 DNA in the geniculate ganglion and endoneurial fluid of affected patients (7,10), provides a compelling biological mechanism. This evidence supports the concept that Bell's palsy, although still classified as “idiopathic,” is highly likely to represent herpetic mononeuritis in a significant proportion of cases (9). The landmark study by Murakami et al. (7) was pivotal in establishing this understanding, as it directly identified the presence of the virus in the region of neurological dysfunction. This finding has been corrobo-

rated by subsequent studies that detected viral DNA in saliva and tears, demonstrating that viral shedding occurs during the acute phase of the disease (6,9,11).

However, there is a significant gap between strong etiological evidence and inconclusive clinical data regarding the efficacy of antiviral therapy (14,15). This discrepancy has been termed the “antiviral paradox” of Bell's palsy: despite convincing molecular evidence pointing to a viral etiology, large-scale clinical trials and systematic reviews, such as the Cochrane review by Gagyor et al. (14), have often failed to demonstrate a significant benefit from adding antiviral agents to standard corticosteroid therapy.

Several factors may explain this paradox. First, the timing of treatment initiation is likely critical. The “golden window” for antiviral efficacy is typically within the first 72 hours after symptom onset; beyond this period, viral replication may have already peaked, and subsequent nerve damage is primarily driven by the inflammatory response and edema rather than ongoing viral replication (18,20). Second, the true proportion of Bell's palsy cases caused by HSV-1 is still under investigation. Some studies suggest detection rates as high as 79% (7), while others have reported considerably lower rates (21,22). This heterogeneity in the study populations means that a substantial proportion of patients enrolled in antiviral trials may not have a viral etiology, thereby diluting the observed treatment effect.

The severity of the initial paralysis also appears to be a critical factor influencing treatment response. Meta-analyses by Turgeon et al. (18) and De Almeida et al. (17) have shown that patients with more severe paralysis (House-Brackmann Grade IV–VI) may benefit more from combination therapy. In these cases, the viral load and the resulting inflammatory response may be more severe, making the addition of an antiviral agent clinically more meaningful. This observation underscores the importance of patient stratification in both clinical practice and future research trials.

The role of corticosteroids in the treatment of Bell's palsy is well established, as they effectively reduce edema and inflammation in the narrowed Fallopian canal, preventing further nerve compression and ischemia (1,2). The addition of antiviral drugs such as acyclovir or valacyclovir aims to inhibit replication of the virus responsible for initiating this inflammatory cascade. However, if viral replication has already ceased by the time the patient seeks treatment, the effect of the antiviral drug may be minimal or nonexistent. This highlights the need for more accurate and rapid diagnostic tools to identify the subgroup of patients with active viral shedding (11,23). Point-of-care PCR tests for the detection of HSV-1 in saliva or tears could transform the management of Bell's palsy by enabling clinicians to personalize treatment based on the presence of the virus (9).

Epidemiological data further support the causal role of HSV. The analysis by Neckel et al. (12), based on large-scale real-world data, showed that HSV had the strongest association with Bell's palsy compared to other risk factors, with an odds ratio of 6.49 (95% CI: 5.96–7.05), indicating that approximately one-third of all Bell's palsy cases are attributable to HSV-1 reactivation (13). The strength of this association, derived from real-world data, provides a different and complementary perspective to the more controlled environment of clinical trials and reinforces the significance of HSV-1 in the etiology of this condition.

Comparisons between HSV-1 and other viruses such as varicella-zoster virus (VZV) are also noteworthy. Reactivation of VZV in the geniculate ganglion causes Ramsay Hunt Syndrome, which is characterized by facial paralysis, severe otalgia, and vesicles in the external auditory canal. Although the clinical manifestations of Ramsay Hunt Syndrome are generally more severe and carry a worse prognosis than Bell's

palsy, the underlying mechanism of viral reactivation and nerve compression is similar (8,22). A small percentage of cases diagnosed as Bell's palsy may actually represent “zoster sine herpete,” where VZV reactivation occurs without the characteristic vesicular rash (9). This further complicates the diagnostic landscape and underscores the importance of comprehensive viral testing.

In terms of clinical outcomes, current evidences from the published literatures suggest that corticosteroids should remain the cornerstone of treatment for all patients with Bell's palsy, but the addition of antiviral drugs should be evaluated on a case-by-case basis, particularly for patients with severe palsy (House-Brackmann Grade IV–VI) or those presenting within 72 hours of symptom onset. The potential benefits of combination therapy should be weighed against the cost and risk of side effects, although modern antiviral drugs such as valacyclovir are generally well tolerated. The ongoing debate and conflicting results from different studies highlight the complexity of the disease and the need for a more nuanced, individualized approach to its management.

Limitations and Future Directions

The principal limitation of the current literature is the substantial heterogeneity among clinical trials evaluating the efficacy of antiviral drugs. Differences in study design, patient populations, drug dosages, treatment timing relative to symptom onset, and outcome measures make it difficult to draw definitive conclusions. Many studies also lacked the statistical power to detect a small but clinically meaningful benefit from antiviral therapy. Furthermore, the absence of a standard diagnostic test for HSV-1 reactivation in routine clinical practice results in the inclusion of patients without viral etiology in antiviral drug trials, thereby attenuating the observed treatment effect.

Future research should prioritize well-designed, large-scale, randomized, placebo-controlled trials in which patients are stratified according to two key criteria: 1) objective evidence of HSV-1 reactivation, as demonstrated by a positive PCR test result from saliva, tears, or other specimens, and 2) the severity of facial paralysis at presentation, as graded by the House-Brackmann scale. This targeted stratification approach is essential to definitively determine the clinical benefit of antiviral drugs in the management of Bell's palsy. Additionally, research on the long-term psychological and functional outcomes of patients treated with different regimens will provide a more comprehensive understanding of the disease and its treatment impact. The development and validation of rapid, point-of-care diagnostic markers for HSV-1 reactivation is also a high priority, as this will enable the implementation of personalized medicine in the acute care setting, ensuring that antiviral therapy is directed to those patients most likely to benefit.

Conclusion

This systematic review confirms a strong and biologically plausible link between HSV-1 reactivation and the etiology of Bell's palsy. Molecular evidence, including a 79% HSV-1 DNA detection rate in endoneurial fluid and a high odds ratio of 6.49 (95% CI: 5.96–7.05) from epidemiological data, strongly supports the concept of Bell's palsy as herpetic mononeuritis in a significant proportion of cases. However, clinical evidence of a universal benefit from adding antivirals to corticosteroids for the overall population of Bell's palsy patients remains inconclusive, as major systematic reviews have not demonstrated significant improvement in recovery rates. Consequently, future clinical guidelines should incorporate viral diagnostic markers (e.g., point-of-care PCR testing) and severity-based classification (House-Brackmann grading) to personalize treatment

decisions, thereby optimizing the likelihood of complete facial nerve recovery through targeted antiviral therapy in patients most likely to benefit.

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Conflict of Interest

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