Update on Medical Management of Acute Peripheral Facial Palsy

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Acute facial nerve palsy, particularly Bell's palsy, is a common neurological disorder with an annual incidence of 20-30 cases per 100,000 individuals. It is characterized by sudden or gradual facial muscle palsy and is caused by viral reactivation, inflammation, or ischemia of the facial nerve. Prognosis varies widely, depending on the severity of nerve damage and timeliness of the intervention. Steroid therapy remains the cornerstone of Bell's palsy treatment because it reduces inflammation and facilitates recovery. Early administration, preferably within 72 hours of symptom onset, considerably improves outcomes. However, the efficacy of combination therapy remains controversial. Current guidelines recommend oral steroids as the primary treatment for Bell's palsy and suggest the selective use of antiviral agents in severe cases or when viral involvement is strongly suspected. For severe facial palsy, such as Ramsay Hunt syndrome or varicella-zoster virus-induced cases, combination therapy may improve outcomes and reduce sequelae; however, high-quality evidence is limited. Steroid therapy is the main treatment of Bell's palsy and antiviral therapy can be added in severe cases to improve prognosis. Additional research is required to develop standardized guidelines, concerning the use of antiviral therapies in conjunction with steroids. J Audiol Otol 2025;29(1):1-7

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Introduction

Acute facial nerve palsy is a condition in which the facial muscles suddenly or gradually become paralyzed. It can be caused by various factors; however, the symptoms primarily manifest as facial muscle palsy, necessitating careful observation for an accurate diagnosis. The most common cause of facial palsy is Bell's palsy, accounting for approximately 51% of all cases. Other causes include head trauma (22%), herpes zoster oticus (7%), tumors (6%), infections (4%), congenital and birth-related disorders (3.5%), hemifacial spasms (2%), central nervous system lesions (1%), and atypical Bell's palsy (0.5%). Additionally, toxic, metabolic, and iatrogenic factors are contributing factors [1]. Acute facial nerve palsy must be appropriately treated to ensure recovery without sequelae, thereby improving the emotional and social quality of life of

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the patients [2]. Timely intervention considering the pathological and physiological characteristics of the nerve is critical in the treatment of acute facial nerve palsy. Acute facial palsy, commonly known as Bell's palsy, presents with facial muscle palsy and can also involve other symptoms such as taste disturbances, reduced lacrimation, or hyperacusis which makes patients fear serious central nervous system conditions, such as cerebral infarction or hemorrhage. Bell's palsy results primarily from reactivation of the herpes simplex virus latent in the geniculate ganglion within the temporal bone and can only be diagnosed after ruling out other causes [3].

Most cases of virus-induced facial nerve palsy recover naturally, with approximately 71% of patients achieving complete recovery and approximately 84% showing near-normal recovery even without treatment. However, approximately 15%-20% of patients experience incomplete recovery, highlighting the need for close attention to these cases [4]. Considering the pathophysiological characteristics of the facial nerve, prognosis is largely determined at the moment of nerve injury [5]. Similar to other peripheral nerves, facial nerve damage can be

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classified based on the severity of the injury, which progresses through the degeneration phase, followed by the regeneration phase. When the facial nerve is damaged, including its endometrium, abnormal regeneration, such as aberrant regeneration or ephaptic transmission, may occur during the recovery process, resulting in abnormal movement or contraction of the facial muscles [6]. Predicting the condition of the nerve fascicles after facial nerve injury is particularly challenging because of the varying degrees of damage across different fascicles. Electrophysiological studies can distinguish between neuropraxia and axonotmesis, which involves Wallerian degeneration. However, accurately differentiating axonotmesis from neurotmesis is more difficult. If a notable proportion of nerve fibers within the facial nerve exhibit neurotmesis. a sharp increase in the degeneration ratio on electrophysiological tests can indicate this condition. These findings are typically associated with House-Brackmann (H-B) grades V and VI, suggesting a high likelihood of incomplete recovery [7]. Ultimately, the most critical aspect of treating Bell's palsy is minimizing the number of nerve fibers undergoing Wallerian degeneration, which occurs between approximately 2 and 14 days after the onset of facial muscle palsy, thereby facilitating complete recovery of the facial nerve.

The treatment of facial nerve palsy aims to restore facial nerve function, particularly the functional recovery of the facial muscles, prevent the progression from partial to complete paralysis, and reduce the occurrence of synkinesis and muscle contracture. Early steroid therapy prevents the worsening of nerve edema, thereby minimizing further nerve damage and reducing the incidence of permanent facial nerve palsy [8]. However, the efficacy of combination therapy with antiviral agents and steroids is debatable. In this context, timely oral steroid treatment initiated shortly after the onset of facial muscle palsy increases the complete recovery rate from approximately 71% to 88%, which is an important finding [9,10]. An improvement of approximately 17% in the complete recovery rate is particularly noteworthy when considering that around 90,000 cases of facial palsy occur annually in South Korea [11].

This suggests that appropriate steroid treatment could provide an additional 9,000–10,000 patients with the opportunity for complete recovery, underscoring the importance of early pharmacological intervention. A literature search of articles relevant to pharmacological treatment options for acute facial palsy was conducted using the PubMed and Cochrane databases, with "acute facial palsy," "Bell' palsy" and/or "Ramsay Hunt syndrome" as search terms. Articles published over the last 10 years were preferred and references from the main review papers were also used to detect other relevant articles. A narrative condensed review format was employed with the

goal of providing a brief and concise tool for readers searching for an updated summary regarding the state-of-the-art drugs used in the management of acute facial palsy.

Steroid Therapy

The primary goal of the pharmacological treatment of Bell's palsy is to expedite recovery and promptly restore facial nerve function. Inflammation and edema of the facial nerve, believed to result from nerve compression within the facial nerve canal, are the main causes of Bell's palsy [11]. The anti-inflammatory effects of steroids are presumed to reduce nerve edema, thereby facilitating functional recovery. Steroids also play a role in minimizing sequelae such as synkinesis [12]. The recommended steroid regimen typically involves initiating highdose oral steroids such as prednisolone at 60 mg daily for 5 days, followed by a 10 mg reduction each day for the remaining 5 days, or prednisolone 50 mg daily for 10 days. Steroids are associated with a range of side effects, including gastrointestinal dysfunction, ulcer exacerbation, impaired glycemic control, elevated blood pressure, edema, and mental disturbances, and their short-term use (10 days) as a standard treatment for Bell's palsy is generally considered safe [13]. To prevent dyspepsia and gastric ulcers during steroid therapy, H2 receptor blockers should be co-administered [14].

Steroids must be prescribed cautiously to patients with diabetes, gastric or duodenal ulcers, uncontrolled hypertension, renal or liver disease, glaucoma, pregnancy, recent head trauma, or psychiatric disorders. A rare but serious side effect of steroid use is the avascular necrosis of the femoral head, which requires careful attention [15]. In cases where steroid-related side effects are a concern, such as in patients with diabetes or pregnant women, the treatment strategy should be tailored based on the condition of the patient [10].

A study (2007) evaluated recovery rates over 3 and 9 months using the H-B grading system among groups treated with steroids and aciclovir, placebo and aciclovir, steroids and placebo, and placebo alone. At 3 months, the steroid group showed significantly higher recovery rates than the placebo group (83% vs. 63.6%); at 9 months, the recovery rates were 94.4% and 81.6%, respectively. Another study published in 2008 evaluated various treatment combinations, including steroids and valaciclovir, for over 12 months. The steroid-treated group demonstrated higher recovery rates (72% vs. 57%) and tended to achieve complete recovery more quickly than the non-steroid-treated group [4,16]. Including previous two studies, the Cochrane review analyzed in 2016 review confirmed that steroids effectively reduced the number of patients with incomplete recovery at 6 months compared to placebo (risk ratio

[RR], 0.63; 95% confidence interval [CI]=0.5-0.8). This finding is based on high-level evidence derived from seven randomized controlled trials (RCTs) involving 895 patients with Bell's palsy of varying severity. Additionally, the results from three studies involving 485 participants showed that patients treated with steroids experienced fewer incidences of synkinesis and crocodile tears (tearing while eating or drinking) than those who received only a placebo [16-18].

In cases of facial palsy caused by the varicella-zoster virus (VZV), such as Ramsay Hunt syndrome, steroid treatment may be considered for patients with localized pain, multiple neuritis, nerve compression-related peripheral nerve damage, or evidence of central nervous system involvement [10,19]. Recent meta-analyses suggest that, beyond standard oral steroid therapy, high-dose steroid therapy exceeding 120 mg during the early phase or combining standard steroid therapy with intratympanic steroid injections may improve recovery rates in patients with facial palsy, although the evidence level is low because there was no RCT comparing high versus standard dose corticosteroids [20]. Another meta-analysis of five studies examined the combination of standard oral steroid therapy and intratympanic steroid injections. Subgroup analysis based on the severity of paralysis showed that in patients with severe Bell's palsy and Ramsay Hunt syndrome, the group receiving combined therapy demonstrated a significant reduction in incomplete recovery rates compared to those receiving oral steroids alone. However, the evidence was considered to be of low quality due to fundamental limitations in the design of the included studies [21-25].

The use of steroids in pediatric Bell's palsy remains controversial. Pediatric patients with untreated Bell's palsy generally have a favorable prognosis, with higher recovery rates than adults, raising questions about the necessity of steroid treatment [26,27]. Recent studies have suggested that initiating treatment within three days of symptom onset improves recovery potential, shortens recovery time, and reduces the likelihood of synkinesis (involuntary muscle movement) [28]. Some authors recommend starting steroid therapy within seven days of symptom onset [29,30]. Currently, no established optimal steroid dose is available for pediatric Bell's palsy. Reported doses vary from 0.5 mg/kg to a maximum of 2 mg/kg across studies. Generally, a dose of 1 mg/kg/day prednisolone for 7-10 days, followed by tapering, is recommended, which is similar to the approach used in adult [31]. In conclusion, given the similarities in pathophysiology between adults and children with Bell's palsy and the benefit-to-harm ratio, oral steroid use should be considered in pediatric patients with Bell's palsy.

Bell's palsy occurs 3.3 times more frequently during preg-

nancy than during the non-pregnant period. However, when averaged over the entire pregnancy period, the incidence rates were similar [32]. The increased prevalence during pregnancy is thought to be because of hormonal and fluid changes, with cases occurring primarily in the late stages of pregnancy or postpartum period. Pregnant women with preeclampsia have a sixfold higher risk of facial nerve palsy than those without preeclampsia. However, Bell's palsy in pregnancy is not associated with preterm birth, low birth weight, or congenital abnormalities. Despite this, the recovery rate for Bell's palsy during pregnancy is reported to be 52%, which is lower than the 77%-88% recovery rate in non-pregnant individuals. This poor prognosis is not fully explained by the lower rate of steroid use during pregnancy, suggesting the need for further research on the unfavorable outcomes of Bell's palsy during pregnancy [33].

When prescribing oral steroids during pregnancy, the risks to both the mother and fetus must be carefully considered. The first trimester carries particular concerns regarding potential fetal risks. According to Phillips, et al. [33], pregnant patients with Bell's palsy were significantly less likely to be prescribed steroids than non-pregnant patients (60.8% vs. 86.2%). This caution arises from earlier data prior to 2,000 indicating an increased risk of cleft palate, preterm birth, and low birth weight associated with steroid use [34]. However, recent studies have shown little evidence to support these associations, reporting no independent link between systemic steroid use during pregnancy and risks such as cleft palate, preterm birth, or low birth weight. Maternal risks associated with steroid use, such as hyperglycemia, hypertension, osteoporosis, and infections, are similar to those in non-pregnant individuals [35]. While short treatment durations with moderate steroid doses may slightly improve facial palsy recovery rates, pregnant women with comorbidities such as diabetes or hypertension should have steroid prescriptions carefully discussed with their obstetricians. Close monitoring of blood glucose levels, blood pressure, weight, infections, and gastrointestinal symptoms is essential during treatment [35].

Antiviral Monotherapy

Antiviral agents are the most effective when administered before viral activation. In cases such as Bell's palsy, the use of antivirals after symptom onset may reduce their efficacy because viral replication is already underway. The longer the delay after symptom onset, the more the host immune response facilitates viral replication, emphasizing the importance of early antiviral administration for effective treatment. Antiviral agents have been shown to reduce the progression of herpes zoster infection, decrease the duration of viral shedding and

the formation of new lesions, and expedite rash resolution and pain relief in immunocompetent adults [36]. However, antivirals do not prevent acute or chronic pain associated with infection, which occurs in 20% of patients aged >50 years.

Reactivation of latent viruses in the geniculate ganglion owing to stress, upper respiratory infections, high fever, tooth extraction, or cold exposure causes facial nerve inflammation and secondary ischemia [37]. Based on the hypothesis that viruses influence the pathophysiology of Bell's palsy, antiviral agents may be considered as an adjunctive therapy for Bell's palsy. The common antiviral agents include aciclovir, valaciclovir, and famciclovir. These nucleoside analogs inhibit viral DNA polymerase, thereby preventing viral replication. However, these drugs are effective in halting further viral replication only when administered within three days of the onset of facial palsy; they do not eradicate the virus. Valaciclovir is a prodrug of aciclovir (L-valine ester), which undergoes rapid and extensive first-pass metabolism after oral administration and is converted into aciclovir. Famciclovir is a prodrug of penciclovir that is activated in the gastrointestinal tract and liver. Both drugs have bioavailability approximately 3–5 times higher than that of aciclovir. Antiviral agents should be used with caution in patients with liver or kidney dysfunction during pregnancy or in immunocompromised states [38-40].

Currently, valaciclovir and famciclovir are preferred owing to their convenience and high bioavailability. Studies included in the Cochrane review investigated various dosing regimens: aciclovir (1.6–3.0 g per day for 5–7 days), valaciclovir (1.0–3.2 g per day for 5–7 days), and famciclovir. Generally, administration of 1 g valaciclovir three times a day for 7 days or 1 g famciclovir daily for 5 days is recommended (Table 1). Common side effects of antiviral agents include gastrointestinal disturbances such as nausea, vomiting, and diarrhea, as well as urticaria, bronchospasm, angioedema, and liver or kidney dysfunction [41].

Engström, et al. [16] reported that there was no difference in time to recovery between the 413 patients treated with valaciclovir and the 416 patients who did not receive valaciclovir. Meta-analyses of the 2015 reviews similarly reported no

Table 1. Dosages for antiviral agents in acute facial palsy

	Adult	Pediatric		
Valaciclovir	1,000 mg, three times/	-		
	day (20-30 mg/kg)			
Aciclovir	800 mg,	<12 yr: 40-80 mg/kg, thrice		
	five times/day	≥ 12 yr: 200 mg or 400 mg,		
	five times			
Brivudine	125 mg, once/day	-		
1 9 1 1				

^{-,} not available

significant differences between the antiviral monotherapy and placebo groups. Moreover, steroid monotherapy results in higher recovery rates than antiviral monotherapy. Despite the variability in antiviral type, dosage, and timing, the analyses consistently concluded that antiviral monotherapy was ineffective in treating Bell's palsy [41,42]. Therefore, antiviral monotherapy is currently considered ineffective and not recommended for Bell's palsy treatment because of its lack of efficacy, unnecessary costs, and potential side effects.

Combination of Steroid and Antiviral Therapy

Steroids are often used concurrently to reduce neural inflammation and the associated pain [10]. Previous studies have reported that combining steroids and antivirals within 72 hours of Bell's palsy onset did not show a significant difference in recovery rates compared with steroid monotherapy [9,16]. However, some small-scale, low-quality studies comparing long-term facial nerve recovery found that combination therapy yielded higher recovery rates than steroids alone [42,43]. A meta-analysis of studies reported that combination therapy reduced the rate of incomplete recovery by 25% [38,44].

A study (2007) conducted in Japan showed that the recovery rate at six months was higher in patients treated with a combination of steroids and valaciclovir than in those treated with steroids alone [9]. A recent study by Lee, et al. [42] demonstrated that combination therapy was approximately 2.6 times more effective than steroid monotherapy, particularly in patients with severe initial facial palsy without diabetes or hypertension. The meta-analysis by Turgeon, et al. [45] found no significant difference between the two groups in terms of complete recovery, but noted a statistically significant improvement in satisfactory recovery with combination therapy. The American Academy of Neurology concluded that adding antivirals to steroids increases the likelihood of facial nerve function recovery by less than 7%, indicating that the evidence was insufficient for combination therapy [46]. A Cochrane review (2015) initially suggested that combination therapy improved recovery rates during a 3-12 month follow-up in patients with Bell's palsy, supporting the clinical rationale for antiviral use [41]. The 2015 review analyzed 11 RCTs, while the 2019 review expanded to include 14 RCTs. Additionally, the 2019 review conducted a more thorough assessment of bias risk, performing sensitivity analyses that excluded studies with high or uncertain bias, thereby enhancing the reliability of the results. A high-quality Cochrane review (2019), which included three trials with 766 patients and follow-up periods of 3-12 months, found no significant benefit of combination therapy

Table 2. Summary of the clinical practice guidelines on pharmacologic treatment of Bell's palsy by American Academy of Otolaryngoloqy-Head and Neck Surgery and Canadian Society of Otolaryngology-Head and Neck Surgery

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20	013 Guideline by the American A	Academy of Otolaryngology-Head and Neck Surg	ery	
Statement	Action		Strength	
Oral steroids	Clinicians should prescribe oral steroids within 72 hours of symptom onset		Strong recommendation	
	for Bell's palsy patients 16 y	ears and older.		
Antiviral	Clinicians should not prescribe oral antiviral therapy alone for patients		Strong recommendation	
monotherapy	with new-onset Bell's palsy.		(against)	
Combination antiviral	Clinicians may offer oral antiviral therapy in addition to oral steroids		Option	
therapy	within 72 hours of symptom onset for patients with Bell's palsy.			
2014 Guidelines by the Canadian Society of Otolaryngology-Head and Neck Surgery				
Treatment	Severity	Recommendation	Strength	
Corticosteroids	Any severity	We recommend the use of corticosteroids	Strong	
		for all patients with Bell palsy.		
Antivirals	Any severity	We recommend against antiviral treatment	Strong	
		alone.		
Corticosteroids+antivirals	Mild to moderate paresis	We suggest against the addition of antivirals	Weak	
		to corticosteroids for patients with mild to		
		moderate severity.		
	Severe to complete paresis	We suggest the combined use of antivirals	Weak	
		and corticosteroids in patients with severe		
		to complete paresis.		

Based on Baugh et al. Otolaryngol Head Neck Surg 2013;149:S1-27 [19] and de Almeida et al. CMAJ 2014;186:917-22 [48]

over steroids alone for incomplete recovery (RR=0.81, 95% CI=0.38-1.74). The results were deemed uncertain, with a low level of evidence leading to retraction of the 2015 findings. Nevertheless, two clinical trials involving 469 patients reported that combination therapy was more effective than steroid monotherapy in reducing long-term complications, such as crocodile tears and synkinesis, with moderate-quality evidence based on the GRADE assessment (RR=0.56, 95% CI=0.36-0.87). Among the global clinical practice guidelines for Bell's palsy treatment, those from the United States and Canada are considered the most methodologically robust [47]. Guidelines from various countries mention combination therapy with steroids and antiviral agents for Bell's palsy. Owing to the low risk of adverse effects of antiviral treatment, the American Academy of Otolaryngology recommends the selective addition of antiviral therapy to steroids within 72 hours of symptom onset. Similarly, the American Academy of Neurology suggested the addition of antiviral agents to steroid treatment for newly diagnosed Bell's palsy, although the level of evidence is low. The Canadian guidelines (2014) recommend antiviral therapy only for cases of moderate-to-severe paralysis, with a weak recommendation strength and moderate confidence in the effectiveness of this approach (Table 2) [48]. Aciclovir and valaciclovir are classified as FDA Pregnancy Category B and are approved for treating genital herpes during pregnancy, including early pregnancy. However, studies on combination

therapy for Bell's palsy during pregnancy are limited. A thorough discussion with the patient is necessary, and combination therapy should be considered in cases of complete facial palsy suspected of having a viral cause [49].

Zoster sine herpes or Ramsay Hunt syndrome generally has a worse prognosis than Bell's palsy and is often associated with more severe paralysis [50]. Up to 30% of facial palsy cases caused by the VZV may present without vesicles. While no RCTs have evaluated combination therapy for Ramsay Hunt syndrome, a Cochrane review (2008) found no evidence supporting the use of antivirals commonly used for other conditions, to treat Ramsay Hunt syndrome [51]. However, VZV may be the primary cause of Bell's palsy in patients with severe initial paralysis and accompanying pain [52]. In such cases, combination therapy with steroids and antivirals can improve the prognosis and reduce complications. Therefore, for severe cases of early Bell's palsy, the possibility of VZV-induced facial palsy should be considered, and concurrent antiviral and steroid therapies should be initiated. Based on current evidence, oral steroids should be prescribed to patients aged 16 years or older within 72 hours of the onset of facial palsy. Oral antiviral agents should not be prescribed as sole therapy for patients with Bell's palsy. However, antiviral agents may be added to oral steroids within 72 hours for patients with Bell's palsy, particularly in those with severe paralysis, where combination therapy is preferable.

Conclusion

Steroid therapy should be initiated immediately upon the onset of Bell's palsy symptoms as it plays a crucial role in reducing inflammation and promoting recovery. Recent studies suggested that the addition of antiviral agents to steroid therapy may further enhance recovery and increase the likelihood of complete functional restoration. This combination therapy appears to be particularly effective when a viral infection is suspected to be the underlying cause of facial palsy. However, the appropriateness of antiviral use for all patients requires expert evaluation, considering factors such as the underlying conditions of the patient, severity of paralysis, and presence of comorbidities. Thus, a thorough medical evaluation is essential for diagnosing and managing facial palsy. Personalized treatment approaches tailored to the specific characteristics of each patient are required to optimize recovery and minimize long-term complications. Adopting individualized strategies can improve patient outcomes and reduce the risk of chronic sequelae.

Conflicts of Interest

The authors have no financial conflicts of interest.

Author Contributions

Conceptualization: Sang Hoon Kim. Data curation: Min Young Kwak. Formal analysis: Sang Hoon Kim. Funding acquisition: Sang Hoon Kim. Investigation: Sang Hoon Kim. Methodology: Sang Hoon Kim. Project administration: Sang Hoon Kim. Resources: Min Young Kwak. Software: Min Young Kwak. Supervision: Min Young Kwak. Validation: Sang Hoon Kim. Visualization: Sang Hoon Kim. Writing—original draft: Sang Hoon Kim. Writing—review & editing: Sang Hoon Kim. Approval of final manuscript: all authors.

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