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Facial Paralysis After Dental Implant Surgery and Tooth Extraction: A Case Series

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ABSTRACT

Facial paralysis is a rare but clinically significant complication that may occur following dental procedures. In this study, three cases of facial paralysis are presented: two developed after dental implant surgery and tooth extraction, and one occurred spontaneously. In all patients, facial nerve dysfunction was assessed as Grade VI according to the House–Brackmann scale. Early systemic corticosteroid therapy was initiated in every case, combined with ocular protection and physiotherapy support. During follow-up, all patients demonstrated marked clinical improvement, with significant functional recovery achieved between the 5th and 7th months.

The findings suggest that anesthetic complications and mechanical factors are the primary etiological mechanisms in acute cases of facial paralysis associated with dental procedures, whereas viral reactivation represents the most plausible hypothesis in delayed-onset cases. Early diagnosis, a multidisciplinary management approach, and corticosteroid therapy initiated within the first 72 hours appear to be the most critical determinants of prognosis. Reporting this rarely documented clinical presentation through the current case series provides valuable insights into the underlying mechanisms and underscores the importance of timely intervention strategies.

Keywords: *Facial Paralysis, Implant Surgery, Tooth Extraction, Delayed Facial Paralysis, Corticosteroid Therapy*

Introduction

Bell's palsy is characterized by paresis or paralysis of the facial muscles due to involvement of the facial nerve. Peripheral facial nerve paralysis is the most common type of motor cranial neuropathy. The most frequent cause is idiopathic facial nerve paralysis; however, viral agents, trauma, vascular ischemia, autoimmune inflammatory syndromes, intracranial lesions, or infections may also contribute to its development (Al-Muharraqi and O'Sullivan, 2010). Although rare, Bell's palsy has been associated with dental procedures. The onset of Bell's palsy following dental interventions may be immediate or delayed. Immediate-onset Bell's palsy is usually associated with rapid recovery and may result from local anesthetic

complications, hematoma formation, or procedure-related trauma (Jenyon *et al*., 2020). Its exact etiology remains unclear (Mavrikakis, 2008).

The reported incidence of Bell's palsy ranges from 11.5 to 40.2 cases per 100,000 individuals. It occurs equally in both sexes and predominantly affects middle-aged and older adults (Eviston *et al*., 2015; Reich, 2017). A history of previous paralysis is observed in approximately 9% of patients, while bilateral involvement is seen in 0.3–2% of cases (Stahl and Ferit, 1989).

The diagnostic criteria for Bell's palsy were first described by Taverner and remain valid today (Taverner *et al*., 1967). These criteria include paralysis of all muscle groups on the affected side of the face, sudden onset of paralysis, absence of central nervous system signs or pathologies, and no evidence of a cerebellopontine angle or otogenic disease. In addition, symptoms such as taste disturbances, pain radiating to the postauricular region, and facial numbness may also accompany the condition (Peitersen, 2002).

The House–Brackmann grading system (HB) is the most widely used method for staging facial paralysis. Scores range from 1 to 6, with grade 1 indicating normal anatomical and physiological integrity of the facial nerve, and grade 6 indicating complete facial paralysis (Kang *et al*., 2002)

Delayed-onset facial paralysis after dental procedures is rare, and its underlying etiology is not fully explained. This condition may raise concerns of malpractice among clinicians (Lydiatt, 2003). In this article, we present three cases of delayed-onset facial paralysis following dental procedures and discuss them in light of the current literature.

Case Report 1

A 30-year-old male patient was referred to our clinic with facial paralysis that developed one day after the surgical extraction of the right maxillary first molar at another center. His medical history was unremarkable for systemic disease.

The patient reported sudden-onset muscle weakness on the right side of the face, marked difficulty in eye closure, and reduced taste sensation. Extraoral examination revealed evident facial asymmetry, complete inability to close the right eye, deviation of the oral commissure, and total obliteration of the right nasolabial fold. Taste sensation was diminished, but auditory function was normal. No pathological findings were detected at the surgical site. Radiographic evaluation revealed no additional pathology. No herpetic lesions were observed, and there was no prior history of facial paralysis following dental interventions.

According to the House–Brackmann (HB) scale, Grade VI (complete paralysis) facial nerve dysfunction was diagnosed. The patient was referred for further evaluation by neurology and otorhinolaryngology specialists. Magnetic resonance imaging confirmed the diagnosis and demonstrated no abnormalities in stapedius muscle function. There was no evidence of middle ear infection or other secondary pathologies (Fig. 1).

Figure 1: Case 1: (a–c) Findings include facial asymmetry on the right side, limited eyelid closure, inability to wrinkle the forehead, inability to smile, and inability to whistle. (d) At the six-month follow-up, normal functions were restored, except for mild tension in the mimic muscles.

For treatment, corticosteroid therapy was initiated with prednisolone 60 mg daily for the first 5 days, followed by a tapered dose over the next 5 days (Table 1). To prevent ocular dryness and corneal damage, he was referred to the Ophthalmology Department, where lubricating eye drops and a nighttime ophthalmic ointment were prescribed. To promote motor recovery, the patient was referred to the Physical Medicine and Rehabilitation Department, where physiotherapy and facial muscle exercises were initiated to stimulate the facial nerve.

No significant clinical improvement was observed in the initial weeks. However, retroauricular pain subsided in the following weeks after treatment initiation. At the three-month follow-up, marked improvement was noted, with near normalization of facial muscle movements.

At the six-month follow-up, the patient demonstrated substantial recovery, with only mild residual tightness of the facial muscles. Facial nerve function was evaluated as Grade I on the HB scale.

Table 1: Prednisolone tapering schedule in the Scandinavian Bell's Palsy protocol.

Day	Prednisolone Dose	Description
1–5	60 mg/day	Full dose, administered daily
6	50 mg	Tapering initiated (–10 mg)
7	40 mg	
8	30 mg	
9	20 mg	
10	10 mg	Final day, then discontinued

Case Report 2

A 45-year-old male patient presented to our clinic with facial paralysis that developed one day after the placement of a dental implant in the right mandible. His medical history was unremarkable for systemic diseases. The patient reported sudden-onset muscle weakness on the right side of the face, inability to close the right eyelid, and facial asymmetry that began within hours following the implant procedure.

Extraoral examination revealed marked facial asymmetry on the right side, loss of forehead wrinkling, obliteration of the right nasolabial fold, and deviation of the oral commissure. The patient was unable to close his right eye, and Bell's phenomenon was observed during attempted closure. Additionally, there was evident impairment of oral movements during speech and discomfort while eating. No significant loss of taste or hearing function was detected. The surgical site appeared clinically normal, with no signs of infection or trauma.

Facial nerve dysfunction was classified as Grade VI according to the HB scale. Neurology and otorhinolaryngology consultations were obtained to exclude other potential causes. Magnetic resonance imaging and further clinical evaluations revealed no additional pathology.

For treatment, corticosteroid therapy was initiated with prednisolone 60 mg daily for the first 5 days, followed by a tapered dose over the next 5 days (Table 1). To prevent ocular complications, the patient was referred to the Ophthalmology Department, where artificial tears and protective ophthalmic ointment were prescribed. He was also referred to the Physical Medicine and Rehabilitation Department, where facial nerve stimulation and facial muscle exercises were recommended to support recovery.

In the initial weeks, the clinical findings remained stable. However, gradual improvement was observed in the following months. At the five-month follow-up, significant functional recovery of the facial muscles was noted; the patient was able to close his eye, and the nasolabial fold reappeared. Only mild residual muscle tightness persisted. At this stage, facial nerve function was assessed as Grade I on the HB scale.

Case Report 3

A 32-year-old male patient presented to our clinic with complaints of spontaneously developed facial paralysis. The patient's history revealed tinnitus, impaired taste sensation, facial numbness, and postauricular pain approximately three days prior to the onset of paralysis. These symptoms were followed by sudden-onset muscle weakness and asymmetry on the right side of the face.

Extraoral examination showed loss of forehead wrinkling, incomplete eyelid closure, obliteration of the right nasolabial fold, and deviation of the oral commissure. During the eye closure test, significant restriction was observed, along with Bell's phenomenon. Taste sensation was reduced, while no significant hearing loss was detected. The patient had no history of systemic disease.

Clinical evaluation revealed Grade VI facial nerve dysfunction according to the HB scale (Fig. 2). To exclude infectious or other pathological causes, neurology and otorhinolaryngology consultations were requested. Imaging and further examinations did not reveal any underlying pathology.

Figure 2: Case 3: (a) Findings include inability to wrinkle the forehead, close the eyes, smile, and whistle. (b) At the seven-month follow-up, complete recovery was observed, except for mild tension in the mimic muscles.

Treatment was initiated with prednisolone 60 mg daily for the first 5 days, followed by a taper of 10 mg per day over the subsequent 5 days (Table 1). Artificial tears and nighttime ophthalmic ointment were prescribed to prevent ocular complications. In addition, the patient was referred to the Physical Medicine and Rehabilitation Department, where facial exercises and nerve stimulation were recommended to strengthen facial muscles.

At the 7-month follow-up, a marked recovery of facial muscle function was observed, with significant improvement in eyelid closure and near-complete restoration of facial mimic movements. At this stage, facial nerve function was graded as Grade II on the HB scale.

Discussion

Facial paralysis following dental procedures is a rare but clinically significant complication. Both acute and delayed-onset cases have been reported in the literature, and the mechanisms underlying these two clinical forms appear to differ.

In our series, three distinct clinical scenarios were observed: the first case developed after tooth extraction, the second after implant surgery, and the third represented spontaneous complete facial paralysis. The first two cases support the literature reports of rare but serious complications of facial paralysis associated with dental procedures, while the third case is consistent with the viral reactivation hypothesis underlying spontaneous Bell's palsy.

Acute Bell's palsy is most frequently associated with complications of local anesthesia. It may occur immediately following inferior alveolar nerve block administration and is generally attributed to inadvertent infiltration of the anesthetic agent into the facial nerve. In such cases, recovery usually occurs within 12–24 hours (Chevalier *et al*., 2010; Gray, 1978; Ling, 1985; Vasconcelos *et al*., 2006). More rarely, it has been suggested that air pressure irrigation into the mandibular socket during tooth extraction may cause direct tissue injury or dissection into the facial spaces, leading to facial nerve stretching or inflammatory trauma (Burke and Adams, 1987). For this reason, the use of water rather than air irrigation has been recommended for socket debridement (Vasconcelos *et al*., 2006). In our post-extraction case (Case 1), although no direct traumatic or mechanical factor could be demonstrated, the acute onset and absence of pathological findings at the surgical site suggest a possible association with these mechanisms.

The etiology of delayed-onset facial paralysis remains a subject of debate. One hypothesis proposes that delayed facial paralysis occurring hours or days after inferior alveolar nerve block may result from a reflex vasospasm of branches of the external carotid artery. This mechanism could reduce blood flow in the stylomastoid artery, causing ischemia of the facial nerve and subsequent paralysis (Ling, 1985). However, this theory does not fully explain the prodromal symptoms observed in many patients (retroauricular pain, fever, paresthesia, etc.). Furthermore, in some series, more than 60% of cases developed facial paralysis without inferior alveolar nerve block administration (10 out of 16 patients), suggesting that the vascular hypothesis alone cannot fully account for the condition.

The viral reactivation hypothesis appears to be more consistent with current evidence. Stress, trauma, pain, infection, or jaw hyperextension following dental interventions may reactivate latent herpes simplex virus or varicella-zoster virus. Indeed, stress-related factors such as pregnancy, diabetes, immunodeficiency, fatigue, and infections have been reported as triggers for Bell's palsy and Ramsay Hunt

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syndrome (Gaudin *et al*., 2016; Hohman and Hadlock, 2014; Jowett and Hadlock, 2015). Dental procedures may therefore be added to this list of triggers. Our second case (facial paralysis following implant surgery) and particularly the third case (spontaneous paralysis with prodromal symptoms) support the viral reactivation theory.

A review of the cases reported in the literature indicates that facial paralysis developing after dental procedures is generally transient. In one case (Evangelista De Leon *et al*., 2024), facial paralysis that occurred immediately after local anesthesia resolved completely within a few weeks following corticosteroid therapy. In another reported case (Cakarer *et al*., 2010), facial paralysis that developed 24 hours after upper third molar extraction showed near-complete recovery within three months with early corticosteroid therapy, ocular protection, and physiotherapy. Similarly, in a third case (Vasconcelos *et al*., 2006), a Grade IV facial nerve dysfunction that developed after lower third molar extraction, likely related to local anesthesia, completely recovered within three months following neurotrophic supportive therapy. These cases, similar to those in our series, demonstrate that early therapeutic intervention is a key determinant of prognosis. However, in our cases, facial paralysis developed a few hours after the procedure or even spontaneously in some instances, suggesting that, in addition to anesthetic complications, viral and inflammatory mechanisms may also play a role in the etiology.

Patients with a history of recurrent facial paralysis should be considered as a separate subgroup. Although prophylactic antiviral and/or corticosteroid therapy has been suggested prior to head and neck surgeries or dental procedures in such patients, especially in those with recurrent severe herpetic gingivostomatitis, there is no conclusive evidence regarding the efficacy of this approach (El Hayderi *et al*., 2013; Miller *et al*., 2005). On the other hand, the literature emphasizes that initiation of therapy within the first 72 hours of symptom onset accelerates recovery and significantly improves outcomes in Bell's palsy patients (Jowett and Hadlock, 2015). In all our cases, early initiation of treatment contributed to marked functional recovery, which became evident between the 5th and 7th months of follow-up.

Clinically, this case series highlights the importance of informing patients about potential risks prior to dental implant surgery and tooth extraction. Although facial paralysis is rare, its occurrence can cause considerable anxiety and functional impairment. Therefore, clinicians are advised to inform patients that, while uncommon, this complication may occur and that early treatment typically leads to complete recovery. Increased awareness among dental practitioners may also facilitate early diagnosis and appropriate referral, thereby helping to prevent permanent damage.

Conclusion

Timely diagnosis and a multidisciplinary management approach are essential to prevent long-term sequelae and to ensure optimal functional recovery in cases of facial paralysis associated with dental procedures.

Ethical Approval: Not applicable.

Patient Consent: Written informed consent was obtained from all patients for the use of their clinical data and photographs for scientific publication purposes.

References

Al-Muharraqi MA, O'Sullivan EC. Unilateral facial nerve paralysis following an infected lower third molar. *Int J Oral Maxillofac Surg* 2010; 39: 192-195.

Burke RH, Adams JL. Immediate cranial nerve paralysis during removal of a mandibular third molar. *Oral Surg Oral Med Oral Pathol* 1987; 63: 172-174.

Cakarer S, Can T, Cankaya B, Erdem MA, Yazici S, Ayintap E, Özden AV, Keskin C. Peripheral facial nerve paralysis after upper third molar extraction. *J Craniofac Surg* 2010; 21: 1825-1827.

Chevalier V, Arbab-Chirani R, Tea SH, Roux M. Facial palsy after inferior alveolar nerve block: case report and review of the literature. *Int J Oral Maxillofac Surg* 2010; 39: 1139-1142.

El Hayderi L, Delvenne P, Rompen E, Senterre JM, Nikkels AF. Herpes simplex virus reactivation and dental procedures. *Clin Oral Investig* 2013; 17: 1961-1964.

Evangelista De Leon R, Cabrera R, Guerrero P. Routine Dental Procedure Induced Hemifacial Paralysis—Case Report. *SVOA Dentistry* 2024; 5: 202-206.

Eviston TJ, Croxson GR, Kennedy PG, Hadlock T, Krishnan AV. Bell's palsy: aetiology, clinical features and multidisciplinary care. *J Neurol Neurosurg Psychiatry* 2015; 86: 1356-1361.

Gaudin RA, Robinson M, Banks CA, Baiungo J, Jowett N, Hadlock TA. Emerging vs Time-Tested Methods of Facial Grading Among Patients with Facial Paralysis. *JAMA Facial Plast Surg* 2016; 18: 251-257.

Gray RL. Peripheral facial nerve paralysis of dental origin. *Br J Oral Surg* 1978; 16: 143-150.

Hohman MH, Hadlock TA. Etiology, diagnosis, and management of facial palsy: 2000 patients at a facial nerve center. *Laryngoscope* 2014; 124: E283-E293.

Jenyon T, Panthagani J, Green D. Transient facial nerve palsy following dental local anaesthesia. *BMJ Case Rep* 2020; 13: e234753.

Jowett N, Hadlock TA. Contemporary management of Bell palsy. *Facial Plast Surg* 2015; 31: 93-102.

Kang TS, Vrabec JT, Giddings N, Terris DJ. Facial nerve grading systems (1985-2002): beyond the House-Brackmann scale. *Otol Neurotol* 2002; 23: 767-771.

DOI: https://dx.doi.org/10.47746/FMCR.2025.6503

Ling KC. Peripheral facial nerve paralysis after local dental anesthesia. *Oral Surg Oral Med Oral Pathol* 1985; 60: 23-24.

Lydiatt DD. Medical malpractice and facial nerve paralysis. *Arch Otolaryngol Head Neck Surg* 2003; 129: 50-53.

Mavrikakis I. Facial nerve palsy: anatomy, etiology, evaluation, and management. *Orbit* 2008; 27: 466-474.

Miller CS, Avdiushko SA, Kryscio RJ, Danaher RJ, Jacob RJ. Effect of prophylactic valacyclovir on the presence of human herpesvirus DNA in saliva of healthy individuals after dental treatment. *J Clin Microbiol* 2005; 43: 2173-2180.

Peitersen E. Bell's palsy: the spontaneous course of 2,500 peripheral facial nerve palsies of different etiologies. *Acta Otolaryngol Suppl* 2002; 549:4-30.

Reich SG. Bell's Palsy. Continuum (Minneap Minn). 2017 Apr;23(2, Selected Topics in Outpatient Neurology): 447-466.

Stahl N, Ferit T. Recurrent bilateral peripheral facial palsy. *J Laryngol Otol* 1989; 103: 117-119.

Taverner D, Kemble F, Cohen SB. Prognosis and treatment of idiopathic facial (Bell's) palsy. *Br Med J* 1967; 4: 581-582.

Vasconcelos BC, Bessa-Nogueira RV, Maurette PE, Carneiro SC. Facial nerve paralysis after impacted lower third molar surgery: a literature review and case report. *Med Oral Patol Oral Cir Bucal* 2006; 11: E175-E178.