Transient Delayed Facial Nerve Palsy After Inferior Alveolar Nerve Block Anesthesia

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Facial nerve palsy, as a complication of an inferior alveolar nerve block anesthesia, is a rarely reported incident. Based on the time elapsed, from the moment of the injection to the onset of the symptoms, the paralysis could be either immediate or delayed. The purpose of this article is to report a case of delayed facial palsy as a result of inferior alveolar nerve block, which occurred 24 hours after the anesthetic administration and subsided in about 8 weeks. The pathogenesis, treatment, and results of an 8-week follow-up for a 20-year-old patient referred to a private maxillofacial clinic are presented and discussed. The patient's previous medical history was unremarkable. On clinical examination the patient exhibited generalized weakness of the left side of her face with a flat and expressionless appearance, and she was unable to close her left eye. One day before the onset of the symptoms, the patient had visited her dentist for a routine restorative procedure on the lower left first molar and an inferior alveolar block anesthesia was administered. The patient's medical history, clinical appearance, and complete examinations led to the diagnosis of delayed facial nerve palsy. Although neurologic occurrences are rare, dentists should keep in mind that certain dental procedures, such as inferior alveolar block anesthesia, could initiate facial nerve palsy. Attention should be paid during the administration of the anesthetic solution.

Key Words: Inferior alveolar nerve block; Facial nerve palsy.

The administration of local anesthesia is an integral procedure of everyday practice in dentistry. The attainment of adequate analgesia in the operating field is essential in order to achieve the required cooperation with the patient and complete the session successfully. However, this common procedure may trigger the appearance of a variety of complications, systemic or localized.

Systemic complications as a result of accidental intravascular injection, drug overdose, rapid absorption, delayed metabolism of the anesthetic solution, or even allergic and anaphylactic reactions mostly affect the cardiovascular and the central nervous system.¹ Localized complications include, among others, hematoma

formation with the risk of trismus or infection, needle breakage, persistent postinjection paresthesia, soft tissue necrosis, spread of infection, self-inflicted soft tissue trauma, and ocular complications.¹ A rarely reported in the literature, yet alarming, localized neurologic complication after inferior dental nerve block anesthesia is facial nerve palsy.

The paralysis could be either immediate or delayed, based on the time elapsed from the moment of the injection to the onset of the symptoms. In the immediate type, the paralysis occurs within minutes of injection with a recovery period of 3 hours or less. However, in the delayed type the symptoms appear within several hours to several days, while recovery may expand from 24 hours to several months.^{2,3}

The purpose of this article is to report a case of delayed facial palsy as a result of inferior alveolar nerve block, which occurred 24 hours after the anesthetic administration and subsided in about 8 weeks.

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Figure 1. Obliteration of the nasolabial fold and drooping of the corner of the mouth.

CASE REPORT

In June 2008, a 20-year-old female patient visited her local dentist for a routine restorative procedure, carried out on the lower left first molar. She was in good health with no history of underlying diseases or hospitalization. A left inferior alveolar block anesthesia was administered, using a cartridge dental syringe with a needle of 25 gauge and 34 mm length. The local anesthetic used was articaine hydrochloride 4% with adrenaline 1 : 100.000. A total amount of 1.7 mL (one cartridge) was delivered. The injection of the anesthetic solution was uneventful and resulted in adequate anesthesia. The session was completed successfully.

However, the following day, the patient returned to the dental office concerned over a generalized weakness of the left side of her face and especially her inability to close her left eye, symptoms which appeared

Figure 3. Inability of the patient to raise her left eyebrow.

that same morning. She was referred from her dentist to a private oral and maxillofacial clinic.

The anamnesis was carefully undertaken to obtain information regarding her medical and dental history. On clinical examination the patient exhibited generalized weakness of the left side of her face with a flat and expressionless appearance, while the muscles of that side were immobile. Obliteration of the nasolabial fold and drooping of the corner of the mouth were also present (Figure 1). On the attempt to smile, her mouth was drawn to the right side (Figure 2). The patient was unable to raise her left eyebrow or close her left eyelid (Figures 3 and 4). When she was prompted to close her eyes, the left eyeball rolled upwards.

After the comfort and the reassurance of the patient, differential diagnosis followed. In order to exclude facial paralysis of central origin, the patient was referred to a neurologist and a computed tomography scan was acquired. No deviation from normal was found. After ruling out pathologic entities such as

Figure 2. On the attempt to smile, the mouth of the patient was drawn to the right side.

Figure 4. Inability of the patient to close her left eyelid.

Figure 5. Four weeks after the beginning of the treatment, the patient exhibited improved eye closure.

Figure 6. Four weeks after the beginning of the treatment, the patient exhibited partial recovery of her ability to raise the left eyebrow.

the Ramsay-Hunt syndrome, Lyme disease, Guillain-Barré syndrome, and sarcoidosis the list of differential diagnoses was narrowed down to transient delayed facial nerve palsy.

In order to prevent ophthalmic damage, hygienic measures were taken. An eye patch was applied, especially during the night, while an eye lubricant was prescribed for the first weeks of the patient's palsy. In cooperation with the neurologist, prednisolone was prescribed as follows:

- 20 mg, 3 times a day for the first week;
- 20 mg, 2 times a day for the second week;
- 20 mg, once a day for the third week; and
- 10 mg, once a day for the fourth week.

Four weeks after the beginning of the treatment, the patient exhibited improved eye closure and partial recovery of the ability to raise the left eyebrow (Figures 5 and 6). An additional 4-week follow-up was carried out until the symptoms completely subsided.

DISCUSSION

As mentioned above, there are two types of facial palsy following inferior alveolar block anesthesia, whose differences in clinical appearance derive from their separate pathogenic backgrounds.

The immediate type is due to the direct accidental anesthesia of one or more branches of the facial nerve.⁴ This is possible when an intraglandular injection of the anesthetic solution occurs. More specifically, if the injection is administered too far posteriorly, the anesthetic solution could be injected into the parotid substance, whose deep lobe extends around the posterior ramus of the mandible and projects forward on the medial surface of the ramus. Most often, the gland envelopes the facial nerve,

thus leading to the direct anesthesia of the latter.¹ It is worth noting, that some authors stress the difficulty of anesthetizing the facial nerve through the oral cavity, making this mechanism unlikely.^{5,6} Additionally, this inferior alveolar nerve block was successful, indicating that it was done properly.

However, there are cases in which the gland fails to envelop the nerve and its divisions,³ or the branches of the facial nerve appear to be aberrant in the retro-mandibular space.^{2,5} Such deviations from normal anatomy increase the chances of direct exposure to local anesthetic solution even if the anesthesia is administered properly.

The pathogenesis of the delayed type, from which our patient suffered, is more complicated. The following suggestions can be offered.

Firstly, the palsy could result from a sympathetic vascular reflex, leading to ischemic paralysis in the stylomastoid foramen region. The anesthetic solution, its breakdown products, or even the mechanical action of the needle itself, may lead to stimulation of the sympathetic plexus associated with the external carotid artery, which in turn communicates with the plexus covering the stylomastoid artery^{3,7} as it enters the parotid gland. The stimulation of the latter plexus causes delayed reflex spasm of the vasa nervorum of the facial nerve, resulting in ischemic neuritis and secondary edema.³

Secondly, the trauma involved in the procedure of dental anesthesia could act as a releasing factor, reactivating a latent viral infection such as herpes simplex virus (HSV) or varicella-zoster virus (VZV). The above could be responsible for neural sheath inflammation and consequent facial nerve palsy.^{3,5} Schirm and Mulken⁸ suggested that the reactivation of HSV genomes from the geniculate ganglia is the most im-

portant cause of Bell's palsy. The reactivation of HSV-1 can be detected by examining antibody titers against HSV by ELISA, by seclusion of viral DNA in saliva with the use of polymerase chain reaction and virus isolation in oral mucosa by cell culture. However, polymerase chain reaction has been proved to be the most useful followed by the detection of antiHSV IgM antibody (ELISA).⁹ The detection rates of HSV-1 reactivation have been found to be 19.3% in a recent study of Kawaguchi et al.⁹ In the same study, VZV reactivation rates were 18.7%.⁹ Peripheral facial palsy caused by VZV reactivation has been known as Ramsay-Hunt syndrome and zoster sine herpette. Because zoster sine herpette does not involve herpetic lesions, vertigo, or hearing loss, it is often clinically diagnosed as Bell's palsy. To detect VZV reactivation, serologic assay is quite useful.⁹ In a low percentage of patients, approximately 4%, reactivation of both viruses has been observed.⁹

Thirdly, alternative pathways for the breakdown of local anesthetic solutions may cause aromatic alcohols to form around the nerves. According to the dental literature, this may result in the equivalent of an alcohol block, leading to prolonged nerve damage.^{6,10,11}

Fourthly, prolonged instrumental opening of the mouth has been associated with facial palsy, due to stretch of the facial nerve.⁴ However, this has not been the case in our patient.

Finally, a different mechanism has been proposed in the literature involving direct intravascular administration of the anesthetic solution. Rood¹² showed that the pressure created during an intra-arterial injection is more than enough to cause backward flow of the anesthetic agent. There are several different anatomic pathways that the solution can traverse, triggering complications, ranging from simple numbness of the skin to facial palsy or even aphasia, if the central nervous system is affected.¹³

The occurrence of a complication after the infusion of an anesthetic requires emergency treatment immediately after an evaluation and a proper diagnosis. The patient suffering from facial nerve palsy exhibits hallmark clinical features, including generalized weakness of the ipsilateral side of the face, inability to close the eyelids, obliteration of the nasolabial fold, drooping of the corner of the mouth, and deviation of the mouth toward the unaffected side.³ The disappearance of the forehead creases of the unilateral side is a greatly valued clinical sign in differential diagnosis for the exclusion of facial palsy of central origin. The latter is an upper motor neuron lesion, synonymous with "central" paralysis, commonly recognized after a middle cerebral artery stroke. The upper facial nucleus, which supplies the upper facial muscles, receives bilateral cortical projection. Thus, the muscles of the forehead remain

unaffected in the case of facial palsy of central origin. On the other hand, the peripheral nerve palsy is a lower motor neuron lesion and therefore affects all muscles of the face. The lower facial nucleus receives only unilateral contralateral cortical projection and supplies the lower facial muscles.^{3,14}

Apart from the "central" paralysis, peripheral facial nerve palsy should be distinguished from a number of pathologic entities that manifest similar clinical features. The list of differential diagnoses includes trauma, operative injury, acoustic neuroma, otitis media, malignant parotid tumors, Ramsay-Hunt syndrome (geniculate herpes zoster), Lyme disease, Guillain-Barré syndrome, Melkersson syndrome, underlying HIV infection, infectious diseases, particularly syphilitic or tuberculous basilar meningitis, and sarcoidosis.^{7,15} When a paralysis occurs without an attributed cause, it is termed Bell's palsy.

A definitive diagnosis requires careful consideration of the patient's medical history as well as an evaluation of the accompanying symptoms.

As far as treatment of the delayed type of palsy is concerned, there is an obscurity in the literature. Similar to the idiopathic facial nerve palsy (Bell's palsy), treatment remains controversial due to the lack of large, randomized, and controlled trials.¹⁶ The main drug therapy, to date, consists of steroids.¹⁶ Although their efficacy has not been clearly demonstrated, they have been proven to be beneficial in improving the outcome of the palsy, when given immediately.^{16–18} These drugs hasten the recovery and lessen the ultimate degree of dysfunction.¹⁸ The addition of dextran or pentoxifylline to the standard steroid treatment has also been reported, in association with a lower rate of adverse effects. However, which of these drugs is the one actually responsible for the beneficial effects is so far unknown.¹⁶ Some antiviral medications such as acyclovir, or its prodrug valacyclovir, have also been prescribed based on the evidence that in patients with Bell's palsy HSV-1 has been detected in the neural fluid,¹⁹ and HSV antigens have also been detected in the facial nerve, geniculate ganglion, and facial nerve nucleus.²⁰ Antiviral drugs can be combined with steroids. In fact, there are indications that the aforementioned combination exhibits better results than monotherapy.^{18,21}

However, studies have produced somewhat conflicting results, and there is a debate over the effectiveness of steroid and antiviral monotherapy in comparison to antiviral-steroid combined therapy. Most authors conclude that the effect of combination therapy with prednisolone and valacyclovir on recovery of Bell's palsy is not significantly higher than that of prednisolone or valacyclovir alone.^{9,19,22} In cases of HSV reactivation, the cumulative recovery rates tend to be higher for the combined therapy, but no significant difference was detected.⁹ In addition, antiviral therapy is suggest-

ed to be beneficial particularly for patients with more severe facial paralysis at presentation.²³ On the contrary, the results of a study conducted by Gok et al,²⁰ indicated that neither monotherapy nor combined therapy had an effect on the ratio and period of recovery. It is also worth mentioning that the study of Quant et al,¹⁹ does not support the routine use of antivirals. The most recent guidelines from the American Academy of Neurology suggest that acyclovir combined with prednisone is “possibly effective” for Bell’s palsy.²⁴ Due to the above controversial results, future studies should use improved HSV diagnostics and newer antivirals (valacyclovir) to assess whether combination therapy exhibits significant benefits.

Concerning other treatments, such as acupuncture, electrotherapy, facial exercises, or even botulinum toxin injections, the studies in the literature seem to be inadequate to allow any conclusion about their efficacy.^{16,18}

In any case, management of facial palsy should include proper protection and lubrication of the eye. An eye patch should be applied, especially during night time, while artificial tears can be used during the day, along with sunglasses, to prevent exposure keratitis. Any corneal abrasion or infection should be treated immediately to avoid possible visual function complications.¹⁸

There are certain prognostic factors indicating poor prognosis of Bell’s palsy. Ipsilateral pain around the ear and in the face or neck as a prognostic factor appears to be a controversial issue. According to some authors, the presence of pain indicates a worse prognosis for facial recovery.^{25–28} More specifically in a prospective study conducted by Berg et al,²⁹ no correlation between pain within 72 hours of onset of palsy and recovery rates at 12 months was found. On the other hand, patients with pain at the 11th to the 17th day had significantly lower recovery rates at 12 months.²⁹ The latter indicates that pain at 2 weeks is a negative prognostic factor. The same study concluded that there was no treatment effect of prednisolone or valacyclovir on the incidence or intensity of pain in Bell’s palsy. The pain was therefore treated with the use of nonsteroidal anti-inflammatory drugs or paracetamol.²⁹ However, other authors claim that pain is of no prognostic value.^{30–32} The pathogenesis of pain is unclear. It has been stated that anoxia of the nerve, caused of a primary or secondary ischemia, followed by compensatory dilatation of the blood vessels supplying the nerve is part of the pain process.³³ It might be speculated that inflammation also affects the facial’s nerve branch that carries sensation from the skin in the region of the external ear and mastoid process, with ipsilateral pain as a result.²⁹

Age is another prognostic factor. More specifically, as age increases the full recovery is reduced.²⁵ This could

be owing to the fact that psychologic or physical stress increases in middle-aged people, acting as an aggravating factor to the recovery of Bell’s palsy.⁹ Patients with incomplete palsy have a better prognosis than patients with complete palsy.¹⁶ In patients with incomplete palsy, up to 94% make a full recovery.³⁴ Some authors claim that early treatment, within 72 hours after the onset of the symptoms, with prednisolone alone²² or combined with acyclovir,³⁵ improves the possibility of full recovery. However, Kawaguchi et al,⁹ state that there was no significant difference in the recovery rate between the treatment within 3 days and that of 4 to 7 days after onset. In addition, Ramsay-Hunt syndrome, the presence of conditions causing secondary facial nerve palsy,¹⁶ the absence of acoustic stapedius reflex during the first days, the familiar incidence, and the presence of repeated ipsilateral palsies are also considered as indicators of poor prognosis of Bell’s palsy.²⁸ According to Pitts et al,³⁶ about 10% of the patients experience one or more recurrences after a mean latency of 10 years. Prognosis may be evaluated clinically, by nerve conduction studies, transcranial magnetic stimulation, or the quantitative analysis of magnetic resonance imaging.¹⁶

CONCLUSION

Although neurologic occurrences are rare, dentists should keep in mind that certain dental procedures, such as inferior alveolar block anesthesia could initiate facial palsy. Attention should be paid during the administration of the anesthetic solution. Standard precautions such as aspiration, slow injection, and continuous monitoring of the patient could minimize possible side effects.

Upon the appearance of facial palsy, the patient should be reassured and fully informed about any symptoms that occur. The patient should be referred to a neurologist for further evaluation and a clinical follow-up must be organized. The recovery is often total, but slow and progressive.

REFERENCES

1. Blanton PL, Jeske AH. Avoiding complications in local anesthesia induction: anatomical considerations. *J Am Dent Assoc.* 2003;134:888–893.
2. Ling KC. Peripheral facial nerve paralysis after local dental anesthesia. *Oral Surg Oral Med Oral Pathol.* 1985;60:23–24.
3. Crean J, Powis A. Neurological complications of local anaesthetics in dentistry. *Dent Update.* 1999;26:344–349.
4. Bernsen PL. Peripheral facial nerve paralysis after local upper dental anaesthesia. *Eur Neurol.* 1993;33:90–91.
5. Chevalier V, Arbab-Chirani R, Tea SH, Roux M. Facial palsy after inferior alveolar nerve block: case report and re-

view of the literature. *Int J Oral Maxillofac Surg*. 2010;39:1139–1142.

6. 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 Pathol Oral Cir Bucal*. 2006;11:175–178.

7. Tiwari IB, Keane T. Hemifacial palsy after inferior dental block for dental treatment. *Br Med J*. 1970;1:798.

8. Schirm J, Mulkens PS. Bell's palsy and herpes simplex virus. *APMIS*. 1997;105:815–823.

9. Kawaguchi K, Inamura H, Abe Y, et al. Reactivation of herpes simplex virus type 1 and varicella-zoster virus and therapeutic effects of combination therapy with prednisolone and valacyclovir in patients with Bell's palsy. *Laryngoscope*. 2007;117:147–156.

10. Pogrel MA, Bryan J, Regezi J. Nerve damage associated with inferior alveolar nerve blocks. *J Am Dent Assoc*. 1995;126:1150–1155.

11. Richardson MF, Straka JA. Alcohol block of the mandibular nerve report of a complication. *J Natl Med Assoc*. 1973;65:63–64.

12. Rood JP. The pressure created by inferior alveolar injections. *Br Dent J*. 1978;144:280–282.

13. Weinberg A, Shohat S, Stabholz A, Findler G. Transient hemiparesis following mandibular nerve anesthesia. *Endod Dent Traumatol*. 1985;1:116–119.

14. Mahadevappa K, Vora A, Graham A, Nesathurai S. Facial paralysis: a critical review of accepted explanation. *Med Hypotheses*. 2010;74:508–509.

15. Miles PG. Facial palsy in the dental surgery. Case report and review. *Aust Dent J*. 1992;37:262–265.

16. Finsterer J. Management of peripheral facial nerve palsy. *Eur Arch Otorhinolaryngol*. 2008;265:743–752.

17. Gilchrist JM. Seventh cranial neuropathy. *Semin Neurol*. 2009;29:5–13.

18. Ahmed A. When is facial paralysis Bell palsy? Current diagnosis and treatment. *Cleve Clin J Med*. 2005;72:398–405.

19. Quant EC, Jeste SS, Muni RH, Cape AV, Bhussar MK, Peleg AY. The benefits of steroids versus steroids plus antivirals for treatment of Bell's palsy: a meta-analysis. *BMJ*. 2009 Sep 7;339:b3354, doi: 10.1136/bmj.b3354.

20. Gok U, Alpay HC, Akpolat N, et al. Comparisons of steroid, acyclovir, lipoprostoglandin E1 and steroid + acyclovir treatments in facial paralysis: a rat study. *Int J Pediatr Otorhinolaryngol*. 2005;69:1199–1204.

21. Hato N, Sawai N, Teraoka M, et al. Valacyclovir for the treatment of Bell's palsy. *Expert Opin Pharmacother*. 2008;9:2531–2536.

22. Sullivan FM, Swan IR, Donnan PT, et al. Early treatment with prednisolone or acyclovir in Bell's palsy. *N Engl J Med*. 2007;357:1598–1607.

23. Hato N, Yamada H, Kohno H, et al. Valacyclovir and prednisolone treatment for Bell's palsy: a multicenter, randomized, placebo-controlled study. *Otol Neurotol*. 2007;28:408–413.

24. Grogan PM, Gronseth GS. Practice parameter: steroids, acyclovir and surgery for Bell's palsy: report of the quality standards subcommittee of the American Academy of Neurology. *Neurology*. 2001;56:830–836.

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

26. Hyden D, Sandstedt P, Odkvist LM. Prognosis in Bell's palsy based on symptoms, signs and laboratory data. *Acta Otolaryngol*. 1982;93:407–414.

27. Katusic SK, Beard CM, Wiederholt WC, Bergstralh EJ, Kurland LT. Incidence, clinical features, and prognosis in Bell's palsy, Rochester, Minnesota, 1968–1982. *Ann Neurol*. 1986;20:622–627.

28. Gavilan C, Gavilan J, Rashad M, Gavilan M. Discriminant analysis in predicting prognosis of Bell's palsy. *Acta Otolaryngol*. 1988;106:276–280.

29. Berg T, Axelsson S, Engstrom M, et al. The course of pain in Bell's palsy: treatment with prednisolone and valacyclovir. *Otol Neurotol*. 2009;30:842–846.

30. Adour KK, Byl FM, Hilsinger RL, Kahn ZM, Sheldon MI. The true nature of Bell's palsy: analysis of 1,000 consecutive patients. *Laryngoscope*. 1978;88:787–801.

31. Mey M, Wette R, Hardin WB, Sullivan J. The use of steroids in Bell's palsy: a prospective controlled study. *Laryngoscope*. 1976;86:1111–1122.

32. Chida K, Okita N, Takase S. Retroauricular pain preceding Bell's palsy: report of three cases and clinical analysis. *Tohoku J Exp Med*. 2002;197:139–143.

33. Adour KK, Wingerd J, Bell DN, Manning JJ, Hurley JP. Prednisolone treatment for idiopathic facial paralysis (Bell's palsy). *N Engl J Med*. 1972;287:1268–1272.

34. Holland NJ, Weiner GM. Recent developments in Bell's palsy. *Br Med J*. 2004;329:553–557.

35. Hato N, Matsumoto S, Kisaki H, et al. Efficacy of early treatment of Bell's palsy with oral acyclovir and prednisolone. *Otol Neurotol*. 2003;24:948–951.

36. Pitts DB, Adour KK, Hilsinger RL. Recurrent Bell's palsy: analysis of 140 patients. *Laryngoscope*. 1988;98:535–540.