Facial nerve paralysis after impacted lower third molar surgery: A literature review and case report

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ABSTRACT
Facial nerve paralysis (FNP) is the most common cranial nerve disorders and it results in a characteristic facial distortion that is determined in part by the nerves branches involved. With multiples etiologies, these included trauma, tumor formation, idiopathic conditions, cerebral infarct, pseudobulbar palsy and viruses. FNP during dental treatment is very rare and can be associated with the injection of local anesthetic, prolonged attempt to remove a mandibular third molar and subsequent infection. We report a case of a 21 years-old black woman who developed a Bell's palsy after an impacted third molar surgery under local anaesthesia, present a FNP classified like a grade IV by the House-Brackmann's grading system. The treatment was based of prescription of a citidine and uridine complex (NÚCLEO CMP™) one tablet twice per day and a close follow up. Three months later that had begining the treatment, the patient recovery her normal facial muscle activity.

Key words: Facial Palsy, bell’s palsy, third molar, postoperative complications, tooth extractions.

INTRODUCTION
Only few authors have described cases of facial nerve injury due the dental practice (1-6). Gray et al. (1) published multiple cases of peripheral facial nerve paralysis of dental origin, one of which was apparently related to a prolonged attempt to remove a mandibular third molar and subsequent infection. He also cited 3 cases caused by the local anesthesia technique that had a maximum recovery period of 7 hours. The case caused by a dental infection had not recovered 90% of full muscular function until five months.

REPORT OF CASE
A 21 years-old black woman was referred to an oral surgeon to have her third molar removed. At that time her chief complaints were a mild pain in the area of the lower right third molar, a light swelling on the right side of her face and a little difficulty to open her mouth wide. The oral surgeon therefore prescribed analgesics and instructed her to do a better local hygiene. After two weeks the patient was submitted to the surgical procedure. The anesthesia of inferior alveolar nerve and the lingual nerve were made with 3 cartridges of 2% lidocaine with 1:200.000 epinephrine; a disposable regular dental needle mounted on a dental syringe was used. Good anesthesia was achieved in normal time and the molar was removed without difficulties. There was no sign of facial paralysis at the end of the surgical procedure that could be noticed by the patient or by the oral surgeon. After 4 hours her lips and tongue was in deep anesthesia. In the morning after the surgery, the patient noticed that the right side of her face was too heavy and couldn’t smile
or wink or close her right eye with maximal effort. During the same day the patient returned to the oral surgeon. He noticed that the patient had lost the control of her right facial muscles; the patient couldn’t close her eyes with maximal effort or smile symmetrically. Therefore, he prescribed a vitamin B complex (CITANEURIN™) one tablet twice per day. After 3 days the patient stills complaining that her eye waters and the corner of her mouth droops. At the forth day the patient was referred to the service of oral and maxillofacial surgery of the Oswaldo Cruz Hospital - Recife - Brazil. On her the admission, physical examination showed that the patient could not move the right side of her face normally or raise her right eyebrow or close completely her right eye with effort (Fig. 1 and 2); in the intra-oral examination her right third molar region was healing quite normally. There was no preceding retro-auricular pain, no deafness or hyperacusis, and no loss of taste sensation in the tongue on her right side.

No herpetic vesicles were found and there was no fever. There was no past history of facial paralysis following a dental procedure. A panoramic radiography was taken and showed nothing uncommon (Fig. 3). A diagnosis of a moderately severe dysfunction of the seventh cranial nerve or grade IV of the House-Brackmann grading system for facial paralysis, due possibly to the local anesthetic injection, was made. The treatment consisted of administration of a citidine and uridine complex (NÚCLEO CMP™) one tablet twice per day. Her oral surgeon was consulted and he informed that the local anesthetic solution and the needle were in condition of use and there was no complication during the surgical procedure. During the first 3 weeks of follow up the patient showed a good recovery (grade II of the House-Brackmann grading system). However, her facial nerve activity was slowly responding. After three months of follow up the patient recovery from the facial musculature paralysis (Fig. 4).
DISCUSSION
The incidence of postoperative paralysis of the facial nerve is not well described by many authors (6-9). The literature describes different etiologies, such as: local anesthesia (6,9), tooth extraction (3,4,6), infections (1,5), osteotomies, preprosthetic procedures, excision of tumors or cysts, surgery of TMJ7,8 and surgical treatment of facial fractures and cleft lip/palate (10).

Facial nerve paralysis may be central or peripheral in origin, complete or incomplete. Its cause is varied and included trauma, tumor formation, iatrogenic problems, idiopathic conditions, cerebral infarct, pseudobulbar palsy and viruses. It results in a characteristic facial distortion that is determined in part by the nerves branches involved (3,11). It is rarely a complication of tooth extraction (3).

The literature reports three mechanisms, in which a dental procedure could damage a nervous structure: direct trauma to nerve from a needle, intraneural hematoma formation or compression and local anesthetic toxicity (3,9).

Direct trauma seems unlikely since many patients report experiencing trauma to the nerve when they feel the electric shock sensation on injection of the needle. However, virtually all these symptoms resolve completely with no residual nerve damage (9). In addition, nerves such as the inferior alveolar and lingual nerves are between 2 and 3 mm in diameter and consist of a number of fascicles; in comparison, a 25 or 27 gauge local anesthetic needle is smaller than 0.5 mm in external diameter, and when it encounters a nerve, its tendency is to separate the fascicles and pass between them (9).

In other hand the needle may hit one of the small blood vessels running within the epineurium, causing hemorrhage within the nerve, which results in compression and fibrosis. This compression could occur fairly quickly (within 20 to 30 minutes) such that the damage will have taken place by the time the local anesthetic would be expected to wear off. Thus, the patient would be unaware of the increasing pressure on the nerve and the resulting damage (9).

The literature reports a case of facial paralysis due to infection, the most likely mechanism of facial nerve paralysis was compression caused by an unusual swelling of the posterior auricular region. However compression of the nerve alone is unlikely the sole cause because minimal improvement in nerve function was noted despite early decompression through incision and drainage. Similarly, toxicity is unlikely to be the only cause of the paralysis, because only the frontal branch was involved. Although the exact mechanism remains unknown, multiple factors were probably involved in causing this unusual consequence of relatively common illness (6).

The facial paralysis as a complication of dental extraction, it may result from direct tissue damage from a blast of air into the tissue with dissection through the fascial spaces (3). Because of this potential, one should not use forced air when cleaning an extraction site. Careful water irrigation may accomplish the same task and minimize the risk of subcutaneous emphysema, secondary infection and nerve injury. One should consider prescribing steroids for the patient to decrease the edema and to provide neural membrane stabilization once the examination has been completed and a diagnosis established. Normally, nerve dysfunction was resolved within 1 week (5).

The literature describes a case of the recurrent peripheral facial nerve palsy after dental procedures (4). In this report a patient had unilateral facial paralysis on two separated occasions, each time within 24 hours of a dental procedure. 5 days earlier, 24 hours after extraction of the left lower wisdom tooth, he had developed a left facial nerve palsy, which had resolved within 2 weeks without any complication. Two years later he required removal of the other three wisdom teeth and once again, within 24 hours of the surgical procedure, he developed a typical left facial weakness and taste dysfunction, identical to the previous episode, again lasting for less than 2 weeks with no sequelae. The exact mechanism to explain this facial weakness after a dental procedure remains uncertain. Direct anesthesia of the facial nerve has been proposed, since this could explain the rapid onset occurring at the time when the anesthetic agent is being infused. Some authors, however, consider this to be unlikely and cite the difficulty with which the facial nerve can be anesthetized via the oral cavity. Furthermore, such local mechanisms cannot explain the involvement of the upper divisions of the facial nerve and the corda tympani or development of facial weakness when an upper tooth was extracted. Reflex vasospasm of the branches of the external carotid artery due to the stimulation of the sympathetic plexus, leading to ischemia of the facial nerve. Alternatively, another pathway was observed by the literature: a retrograde epidural compression edema with ischemia of the facial nerve is also possible. Such a mechanism is consistent with the pathogenesis of idiopathic Bell’s palsy where nerve compression in bony canal plays an important role in the pathogenesis of facial paralysis (4).

The local anesthetic itself may be neurotoxic and may damage the nerve. In fact, most local anesthetic is neurotoxic, although some present more risks than others. Procaine and tetracaine cause more nerve damage than bupivacaine or lidocaine, although lidocaine can also be neurotoxic. However, neurotoxicity normally occurs only when the local anesthetic is injected intrathecally, whereby it can cause the cauda equine syndrome or intrafascicularly in high concentrations. Again, we could only expect intrafascicular injections of local anesthetic to affect the skin or mucosal area and sensory parameters supplied by that fascicle and not the whole nerve. Some researchers have also suggested alternative pathways for the breakdown of commonly used local anesthetic agents, possibly resulting in the formation of aromatic alcohols around the nerves, which may result in the equivalent of an alcohol block that causes prolonged nerve damage (9).

There have been several attempts to grade facial palsy but none have been universally accepted (12). The House and Brackmann grading system has been recommend as a uni-
versal standard for assessing the degree of facial palsy (12). Some authors studied this grading system and concluded that the House and Brackmann grading system is a simple and robust method of assessing facial function (12). Others compared the House and Brackmann and Yanagihara grading system in relation to eletroneurographic (ENoG) technique in 30 consecutive patients with Bell’s palsy and concluded that the ENoG was more accurate in predicting a favorable prognosis compared with clinical grading (13). Initial Yanagihara grading appears to provide more prognostic information than the House-Brackmann grading. However, the two clinical grading system strongly resemble each other and comparable in the time course of Bell’s palsy. The pattern of clinical grading and ENoG depends on the degree of palsy, which in turn is dependent on the relation between neurapraxia and degeneration (13).

The literature reports a case of a peripheral facial nerve paralysis after local dental anesthesia (2,6). The onset of the paralysis was 13 days after the injection. The treatment was made with triamcinolone, 4 mg four times daily, for 10 days, with the dose being gradually reduced. The recovery period was 4 weeks. This steroid therapy was done in this case to prevent possible denervation of the facial muscles. Judging from the quite dramatic recovery on the second day of medication and the subsequent full recovery, the value of steroid therapy in this case was significant, but this author also reports that the prognosis have been good in the reported cases treated without or with steroids (2).

Talzi, Soichot and Perrin report that the treatment of Bell’s palsy is still controversial because the benefit of acyclovir has not been definitively established. However, the safety of this antiviral drug combined with prednisone and its possible effectiveness in improving facial functional outcomes in patients with Bell’s palsy make most experts favor its use with corticosteroids as soon as possible to treat patients with this disease (6).

In this presented case this temporary peripheral paralysis of the facial nerve could be caused by the postoperative edema in the region of the parotid gland or an alteration in the anesthetic solution. We could not say if the recovery of paralysis was due to use of NUCLEO CMP, although was observed that when the patient started to use the drug the recovery was abruptly.

REFERENCES