

Facial Palsy after Inferior Alveolar Nerve Blocks

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Introduction

A few weeks back with the help of one of my colleagues a patient had entered the Emergency room late at night. We had taken the radiograph and could clearly see that her 48 had gross caries on it with a large rarefying osteitis present. The only option was an extraction to relieve the patient of pain. I had given her two carpules of 2% Lidocaine 1:100k epi as an IAN block. After 10 mins the patient reported profound anesthesia and the extraction was completed without complications and within 5 mins. As my colleague was putting the instruments away and I was writing my note the patient stated that she couldn't close her right eye anymore. To both of our surprised the entire right side of her face was drooping.

The following review of literature is based on facial palsy after inferior alveolar nerve block:

The administration of local anesthesia is an integral procedure that is performed thousands of times by any practicing dentist in a given year. The attainment of adequate analgesia in the operating field is an essential for getting the cooperation of the patient. However, this common procedure may trigger the appearance of a variety of complications, systemic or localized.

The most alarming of all these complications is a neurologic complication after an IAN being facial nerve palsy.

The paralysis could be either be immediate or delayed, which is determined from the time elapsed from the moment of injection to the onset of symptoms.

In the immediate type, the paralysis occurs within minutes and has a recovery period of 3 hour or less. In the delayed type, the symptoms appear several hours to several days after administration of the local anesthetic. Recovery for a delayed response can take 24hrs to several months.

The immediate type is due to the direct accidental anesthesia of one or more branches of the facial nerve. This is possible when an intra-glandular injection of the anesthetic solution occurs. This happens when the injection is given too far posteriorly, and the local anesthetic is injected into the parotid substance. This happens because the deep lobe extends around the posterior ramus of the mandible and projects forward on the medial surface of the ramus. The parotid gland contains the facial nerve and hence the paralysis occurs. Interestingly though that many authors stress the difficulty of anesthetizing the facial nerve through the oral cavity and state that this is very unlikely.

Of course, deviations from normal anatomy also factor into an increased risk of facial nerve paralysis.

For the delayed type of appearance, we must rule out that the facial paralysis isn't from a central origin and it is recommended that a CT is done.

For the transient delayed facial nerve palsy, the palsy could result from a sympathetic vascular reflex, leading to an ischemic paralysis in the stylomastoid foramen region. This reflex can occur from the anesthetic solution, its breakdown products, or even the mechanical action of the needle itself. The sympathetic vascular reflex is associated with the external carotid artery and communicates with the plexus covering the stylomastoid artery as it enters the parotid gland. The stimulation of that plexus causes a delayed reflex spasm of the vasa nervorum of the facial nerve resulting in ischemic neuritis and secondary edema.

The trauma from the injection itself may also act as a releasing factor of herpes simplex virus or varicella-zoster virus. These are both involved in neural sheath inflammation and facial nerve palsy. This is best known as Ramsay-Hunt syndrome.

Prolong instrumental opening of the mouth has been associated with facial palsy due to the stretch of the facial nerve.

Lastly, a different mechanism is now being proposed in the literature involving the direct intravascular administration of the anesthetic solution. It has been showed that enough pressure can be created to cause a backflow of the anesthetic agent. This can lead to any different anatomic pathways triggering complications.

Treatment

A proper evaluation and diagnosis must be done before any treatment is done. A 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. 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 upper facial nucleus which supplies the upper facial muscles, receives bilateral cortical projections and thus the muscles of the forehead remain unaffected in the case of facial palsy of central origin. Peripheral nerve palsy is a lower neuron lesion and affects all the muscles of the face.

Peripheral nerve palsy can appear in the following differential diagnoses: trauma, operative injury, acoustic neuroma, otitis media, malignant parotid tumors, Ramsay-Hunt syndrome, Lyme disease, Guillain-Barré syndrome, Melkersson syndrome,

underlying HIV infection, infectious diseases, particularly syphilitic or tuberculous basilar meningitis, and sarcoidosis. When a paralysis occurs without an attributed cause, it is termed Bell's palsy.

For both immediate and delayed palsy, 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.

Treatment for delayed type of palsy can also be treated similarly for patients with idiopathic facial nerve palsy. The main drug therapy is steroids. 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. These drugs hasten the recovery and lessen the ultimate degree of dysfunction.

Take Home Message

For our patient that is what we had exactly done. We informed her that she would keep her eye well hydrated and her eye was covered, and she was told that it should resolve within a few hours and she had the option of staying so we could monitor or go home. She ultimately decided to head home that night and when she was called in the morning the palsy had resolved.

Although neurologic occurrences are rare, dentists should keep in mind that certain procedures like the IAN can initiate facial palsy. Attention should be paid during the administration of the anesthetic solution and continuous monitoring of the patient could minimize possible side effects.

References

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