Amaurosis and transient diplopia, a rare complication secondary to mandibular nerve block: A case report

Mustafa KARAOĞLAN,1 Bilge KÜÇÜKÇAY,2 Levent Ertuğrul İNAN3

Summary

Ocular complications are one of the rare side effects that can be seen after a mandibular nerve block and have the most dramatic results. Since the mandibular nerve block is mostly performed by dentists, this complication is mostly seen after an intraoral mandibular nerve block. The mandibular nerve is the third division of the trigeminal nerve. It is the most caudal and lateral part of Gasser’s ganglion. It arises from the middle cranial fossa through the foramen ovale. This region, a block method, which is performed by passing through the coronoid process, has been defined. This block, usually made using anatomical markers, is used in the treatment of trigeminal neuralgia. A 42-year-old female patient was admitted to our department for a maxillary and mandibular block with a diagnosis of trigeminal neuralgia. Immediately after the administration of the local anesthetic, the patient described a complete loss of vision. The complaint of vision loss lasted for about 1 minute, after which the patient’s complaint of diplopia continued for 2 hours and 10 minutes. This case report presents the ocular complications after a mandibular block applied with the extraoral technique as an unexpected side effect.

Keywords: Mandibular nerve block; trigeminal neuralgia; ocular complication

Introduction

The mandibular nerve is the third division of the trigeminal nerve. It is the most caudal and lateral part of Gasser’s ganglion. It arises from the middle cranial fossa through the foramen ovale. In this region, a block method, which is performed by passing through the coronoid process, has been defined. This block, generally performed using anatomical markers, is used in the treatment of trigeminal neuralgia. Ocular complications are one of the rare side effects that can be seen after a mandibular nerve block and have the most dramatic results. Although these complications are benign and temporary, they have dramatic consequences for patients and practicing clinicians. A 42-year-old female patient was admitted to the clinic for a...
maxillary and mandibular block with a diagnosis of trigeminal neuralgia. Immediately after the administration of the local anesthetic, the patient described a complete loss of vision. After the vision loss lasted for about 1 minute, the patient had a complaint of diplopia which continued for 2 hours and 10 minutes. In general, amaurosis is a finding secondary to stroke. This case report presents ocular complications as unexpected side effects after a mandibular block has been performed with the extraoratal technique.

**Case Report**

A 42-year-old female patient was admitted to our clinic due to intense, stabbing pain in the left maxillary and mandibular nerve sensory area, which lasted for a maximum period of 1 minute. The pain of the patient, who had these complaints for about 2 years, was triggered by movements related to eating, drinking water, chewing, and washing the face. Neuroimaging of the patient, whose neurological examination did not reveal any findings, did not reveal any etiologically significant pathology. The patient’s current clinical situation was accepted as typical trigeminal neuralgia in accordance with the ICHD-3 guidelines.[5]

No additional disease was found in the medical history of the patient, who did not respond adequately to oral medication. She neither had a known allergy nor a coagulation disorder, as laboratory tests revealed. For the patient’s complaints, maxillary and mandibular nerve blocks were applied. The left maxillary block was successfully performed using anatomical markers from the coronoid notch, and no complications were observed. Immediately afterwards, the left mandibular block was performed with the help of a 25-gauge spinal needle using anatomical markers. While the patient’s neck was in a neutral position, and she was lying on her back, she opened and closed her mouth several times, and the coronoid notch was palpated in the anterior and inferior part of the ear. After preparing the skin over the coronoid notch with an antiseptic solution, the needle was advanced from the infrrazygomatic area at the midpoint of the coronoid notch. The needle was advanced approximately 1-2 cm vertically until it came into contact with the pterygoid plate. At this point, the needle was slightly withdrawn and guided posteriorly to reach the pterygopalatine fossa through the anterior margin of the lateral pterygoid plate. The needle was directed a few millimeters medially and cephalad to catch the mandibular nerve as it exited the foramen ovale. After the negative aspiration test, 3 ml of 2% prilocaine was administered to this area.[1,6]

Mydriasis and ophthalmoplegia were observed in the ipsilateral eye of the patient, which started immediately after the injection and lasted for approximately 1 minute. During this time, the patient described complete vision loss. After 1 minute, the patient’s vision loss improved. In eye movements, it was observed that the left pupil remained in the midline only when she looked outward. During this period, the patient was taken to the emergency department, and it took 2 hours and 10 minutes for her to fully recover from complaints of double vision and eye movements. Paresthesia continued in the maxillary and mandibular sensory area of the patient, who had no complaints at the time of discharge from the emergency department (Fig. 1). No hematoma formation was observed in the region in the ultrasound control performed after 72 hours.
Discussion

Although ocular complications due to mandibular nerve block are rare, similar case series exist in the literature, and most of the ocular complications were transient and benign. In these cases, the occurrence of ocular complications, similar to our case, has been attempted to be explained by three mechanisms under the main headings.

The first mechanism was presented by Blaxter and Britten in a 3-case series, describing ocular complications after blockade of the inferior alveolar nerve, a branch of the mandibular nerve. In the discussion, the authors suggested that these complications may occur after intra-arterial access to the maxillary and inferior alveolar arteries. In a study conducted by Alderete on rhesus monkeys, it was shown that the anesthetic solution administered from the maxillary artery could come to the external carotid artery with the retrograde flow and then reach the carotid bifurcation. It has been shown that the anesthetic solution passing from here to the internal carotid artery can mix with the cerebral circulation and pass into the ophthalmic artery, which is the first branch of the internal carotid artery. Predicting that this mechanism may also occur in humans, the authors tried to explain the ocular complications immediately following the injection of small volumes of local anesthetic into head and neck injections. Although they agree that retrograde flow along the carotid artery is possible, Blaxton and Britten have suggested a more likely mechanism. This mechanism is based on the connection between the middle meningeal artery and orbital blood supply, and this anastomosis has been clearly demonstrated in the literature. The lacrimal artery, a branch of the ophthalmic artery, runs along the lateral wall of the orbit and supplies the lacrimal gland and lateral rectus muscle. The lacrimal artery usually receives an anastomotic channel from the frontal artery, which is a branch of the middle meningeal artery. This canal usually enters the orbit from the upper corner of the fissura orbitalis superior or through its own foramen and shows variations in size. When this anastomosis is strong, it largely supplies the lacrimal artery and may even replace it.

The second mechanism is explained via the intravenous route. The maxillary vein, close to the block site and located around the maxillary artery, is formed by the fusion of many venules collectively called the pterygoid venous plexus. The pterygoid venous plexus within the infratemporal fossa forms many connections in all directions. Important for possible ocular complications, this plexus communicates with the cavernous sinus of the middle cranial fossa via the foramen ovale. There is also a direct connection of the pterygoid venous plexus with the orbit via the inferior orbital fissure.

The third mechanism can be described as follows: ocular complications occur after direct diffusion of local anesthetic through the anatomical foramen. It was stated by Penarrocha-Diago that this route is the most likely route for ocular complications. This mechanism may explain why the abducens nerve and lateral rectus muscle are more involved. There are also reports that diffusion of local anesthetics is limited for this mechanism, and this spread is not very realistic.

Since the mandibular nerve block is mostly performed by dentists, most of these mechanisms were used to explain ocular complications after intraoral mandibular nerve block. In our case, the mandibular nerve blockade was performed using the extraoral technique. The hypothesis of retrograde flow of anesthetic solution within the vascular system is the most commonly suggested cause of ocular complications following dental injection found in the literature. In this mechanism, there were two arteries close to our block site. One was the middle meningeal artery, and the other was the more likely maxillary artery. In this case, although the aspiration test was negative, an accidental intravascular injection may have been made. It was possible that a 25 G spinal needle was used during the block and that the open end of the needle abutted against the vascular intima. And so, the aspiration test could have been negative even though the needle was in a vessel. While the rapid onset of amaurosis suggested this mechanism, we had difficulty in explaining the restriction of outward gaze that continued afterward. In the second mechanism, while direct opening to the orbit could explain amaurosis, involvement of the abducens nerve with the cavernous sinus connection made us think that this might be the possible mechanism that explains the limitation of the

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outward gaze. Low pressures in the venous plexus could be a reason for false-negative aspiration. The third mechanism could provide a possible explanation, again considering the order in which the complications occurred. However, although we approached the fissura orbitalis inferior more during maxillary nerve blockade, we did not experience any complications after this block. It was expected that the infraorbital nerve would pass through the same fissure, and there would be paraesthesia in the under-eye area. However, since the maxillary nerve was blocked before, there was already paraesthesia here. As a result, unfortunately, it was not possible to know for certain what the true cause of this reported ocular complication was. However, it would be more rational to say that this complication is caused by a combination of many factors.

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