Temporary Diplopia After Gow-Gates Injection: Case Report and Review

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Complications associated with various local anesthetic techniques have been recorded in case reports and reviews. This current case reports a transient incident of blurred, double vision (diplopia) following a Gow-Gates mandibular block injection. There is descriptive discussion on possibilities associated with intra-arterial injection, intravenous injection, diffusion through tissue planes, and the autonomic nervous system pathway to lend credence suggesting the etiology of the complication. For practitioners, recognizing when a complication arises from anesthesia delivery and managing the patient in an appropriate manner is essential to an overall agreeable outcome.

Key Words: Dental anesthesia; Local anesthesia; Mandibular nerve; Dentistry.

Delivery of local anesthesia for dental procedures is a very common and quite safe practice.^{1,2} Limited complications occur when providing either infiltration or block injections into the perioral tissues, and a number of these have been identified and presented in published case reports and reviews.^{2–6} Although most of these reported complications have been relatively minor, there have been several descriptions of temporary, and occasional permanent, ocular disturbances following injection of local anesthetics.^{7–16} This current case reports a transient incident of blurred, double vision (diplopia) following a Gow-Gates mandibular quadrant block injection. Management of the patient following such a localized complication is important for practicing clinicians to understand and to properly address.

CASE PRESENTATION

At the University of the Pacific, Arthur A. Dugoni School of Dentistry, students enter into agreement¹⁷ and learn basic techniques in local anesthesia delivery in a 3 students to 1 faculty member ratio. The Gow-Gates mandibular quadrant block injection is taught as one of several techniques for anesthetizing mandibular teeth for various dental therapies. This technique requires the operator to identify specific extraoral and intraoral landmarks. Also, the patient needs to open the mouth wide for the duration of the injection and keep it open wide for 1–2 minutes postinjection.¹⁸ As part of one of their local anesthesia block rotations, students simulate the Gow-Gates injection technique 3 times on a patient prior to providing the injection. Simulation exercises consist of showing the injection site with a cotton tip applicator, practicing the injection with the needle cap on the assembled standard syringe, and, finally, placing topical anesthetic at the injection site. During the simulation process, a trained and certified instructor provides feedback to the student regarding technique, landmarks, and positioning of the needle and syringe.

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Immediately following the 3 simulated injections, the student provides the Gow-Gates injection technique. The armamentarium set up within the university setting consists of a standard syringe, long 27-gauge needle (32 mm long), and anesthetic cartridge.

In this case, the patient was a 27-year-old Caucasian male given the injection at approximately 10:00 AM. His medical history was nonsignificant, with the patient reporting no past surgeries, no current medications (prescribed or over-the-counter), and no known drug allergies. A limited-focus oral examination revealed no significant findings, specifically no limitation of maximal incisal opening or range of mandibular motion. The patient weighed 97.5 kg (215 pounds) and measured 1.88 m (74 inches) tall. During the injection, the patient reported the sensation of “hot water flowing up his face” on the ipsilateral side. Several aspiration tests performed were negative, and approximate needle depth was 28 mm as confirmed by the operator and the supervising instructor.

Within minutes of delivery of 0.9 mL 2% lidocaine HCl with 1 : 100,000 epinephrine, the patient reported that he was experiencing blurred and double vision. The patient was immediately returned to an upright and seated position in the operatory chair and a full exam was performed to identify all subjective and objective symptoms. Subjective findings included the sensation of numbness on the ipsilateral nose and infraorbital soft tissue. Vision was reported to be “heavily” blurred with binocular diplopia, with objective findings of difficulty reading a nearby clock and computer screen. No signs or symptoms of blanching or drooping of the eyelids (ptosis) were noted. Furthermore, all extraocular movements were noted to be intact. The patient was instructed to keep his eyes closed to minimize sensations of nausea or dizziness due to his blurred vision, and was offered a piece of 2 × 2 gauze to have placed over his affected eye under his safety goggles if he preferred. No cold or electronic pulp tests were performed to confirm pulpal anesthesia, yet an unusual occurrence report was filed and submitted. The patient was intermittently monitored every few minutes until all symptoms were reported to have resolved, which lasted approximately 15 minutes. He was reevaluated to confirm he had returned to his baseline and no longer had symptoms. The patient was dismissed and reported he was able to attend to his schedule for the remaining part of the day, and has reported no residual symptoms since the time of the incident.

DISCUSSION

Although millions of anesthetic injections are administered worldwide every year, reports of complications in

general are uncommon, and ocular alterations are quite rare, representing less than 0.1% of all complications.¹⁹ One recently published literature review counted 131 reported ophthalmologic complications after intraoral injections between the years 1936 and 2011.²⁰ Of these 131 ophthalmologic complications, the vast majority were temporary, usually lasting from a few minutes to a few hours, although permanent loss of ocular function was reported for 5 of these cases, 4 with permanent loss of vision (amaurosis). Diplopia was the most commonly reported ocular complication, and occurred most frequently after maxillary injections (77.8% vs 57.1% for mandibular injections); amaurosis was most often associated with mandibular injections.²⁰

Diplopia may be caused by changes in the cornea, the lens, the muscles of accommodation, the extraocular muscles, the optic nerve pathway, or the cerebral cortical processing centers for vision. From anatomical analysis of the signs and symptoms of diplopia following local anesthetic injections, the muscles of accommodation and the extraocular muscles are the 2 most likely structures that have been affected. When the eye focuses in on a close object, 3 different actions occur, which are known as the accommodation reflex: (a) the extraocular muscles turn the eyeballs inward, (b) the constrictor pupillae muscle contracts to reduce the amount of light entering the eyeball, and (c) the ciliary muscles contract, decreasing the tension on the suspensory ligaments of the lens, thus allowing the lens to assume a more bulging shape, which in turn reduces the focal length of the lens to accommodate for close vision. The motor innervation to the ciliary muscles is carried in the oculomotor nerve, cranial nerve (CN) III. These are parasympathetic fibers that leave the oculomotor nerve to synapse in the ciliary ganglion, located just behind the eyeball, and then travel to the eye as the short ciliary nerves to the ciliary muscles. Because the oculomotor nerve also innervates the constrictor pupillae muscle of the iris, the levator palpebrae muscle of the upper eyelid, and the superior, medial, inferior, and inferior oblique extraocular muscles, anesthesia of this nerve will produce ptosis, loss of most eye movements, and dilation of the pupil in addition to loss of accommodation. Motor innervation to the superior oblique extraocular muscle is from the trochlear nerve, CN IV, and the lateral rectus muscle is innervated by the abducens nerve, CN VI. Sensory innervation to the contents of the orbit, to the upper eyelid, and to the skin above the orbit is provided by the ophthalmic division of the trigeminal nerve, V₁. All of these nerves enter the orbit posteriorly through the superior orbital fissure. The optic nerve, CN II, enters the orbit just medial to the superior orbital fissure through the optic canal, and is accompanied by the ophthalmic artery, the primary blood supply to the orbit.

Anatomy of infratemporal injection site.

Although it is not possible to know the precise mechanism for any ocular complication resulting from an injection of a dental local anesthetic, the following anatomical hypotheses have been offered in the literature. See the Figure when referring to the anatomy of this area.

INTRA-ARTERIAL INJECTION

A series of 3 case reports by Blaxter and Britten²¹ in 1967 reported temporary loss of vision in 1 case and diplopia of varying duration in all 3 cases following local anesthetic injections using the conventional inferior alveolar (IA) mandibular block technique. In their discussion, the authors suggested that the likeliest explanation for all of the observed signs and symptoms in these cases (transient blanching, amaurosis, and diplopia along with broad areas of cutaneous anesthesia in the periorbital regions) may have been accidental intra-arterial injection of the local anesthetic. Because the IA artery is in close company with the IA nerve at the site of anesthetic deposition for a conventional IA nerve block injection, they cited the IA artery as the most likely entry point for an intra-arterial injection. Blanton and Roda²² have also cited convincing evidence that the maxillary artery may take a distinct loop down towards the mandibular foramen as it courses across the infratemporal fossa (in 1 case placing it within 4 mm of the foramen), thus placing the maxillary artery also at significant risk for possible accidental intra-arterial injection of anesthetic with a conventional IA nerve block technique. From the IA artery, it has been

proposed that the injection pressure of the local anesthetic may be sufficient to cause retrograde flow of the anesthetic up to the maxillary artery.²³ Aldrete and colleagues²³ showed in experiments with rhesus monkeys that retrograde flow of the anesthetic solution could pass from the maxillary artery back into the external carotid artery, then down to where the internal and external carotid arteries bifurcate from the common carotid artery in the neck, and then up the internal carotid artery to the cerebral circulation. Because the ophthalmic artery is the first branch off of the internal carotid artery, the anesthetic solution could then enter the orbit via the ophthalmic artery and cause effects to the innervation of the eye and extraocular muscles. Aldrete et al²³ offered that this mechanism could also occur in humans and may account for some of the untoward reactions that occur soon after an injection of relatively small volumes of local anesthetics in the head and neck region.

Although agreeing that such a retrograde pathway through the carotid bifurcation may be possible, Blaxter and Britten²¹ described the following as a more likely intra-arterial pathway to the orbit: the middle meningeal artery branches off the maxillary artery a short distance anterior to the origin of the IA artery and passes superiorly through the foramen spinosum to enter the middle cranial fossa. Anastomoses between branches from the middle meningeal artery and the arterial blood supply of the orbit have been well documented.^{24,25} The lacrimal artery, a branch off the ophthalmic artery within the orbit, lies along the lateral wall of the orbit and supplies the lacrimal gland and lateral rectus muscle. The lacrimal artery commonly receives an anastomotic

channel from the frontal branch of the middle meningeal artery. This channel enters the orbit either through the upper corner of the superior orbital fissure or through its own foramen and varies markedly in size. When this anastomotic channel is large, it clearly reinforces the lacrimal artery, and it may even replace it entirely.²⁴ More rarely, the anastomotic channel from the middle meningeal artery has been shown to completely replace an occluded ophthalmic artery, following the ophthalmic artery's normal intraorbital course and giving off all of the ophthalmic artery's usual branches.^{24,25}

More recent case reports and literature reviews of ocular complications during delivery of dental anesthetic injections have suggested additional etiologies to explain this infrequent complication.^{4,11,13,14,19,20,26–33} In addition to the intra-arterial pathway described above, these papers proposed the possibility of intravenous pathways to the orbit, direct diffusion of the anesthetic solution through tissue planes and skeletal openings, and possible autonomic nervous system pathway disruption.

INTRAVENOUS INJECTION

The IA neurovascular bundle includes the IA vein, which drains superiorly into the maxillary vein. The maxillary vein is formed by the coalescence of numerous small venules that are collectively termed the pterygoid venous plexus. The pterygoid venous plexus within the infratemporal fossa is a very dense meshwork that surrounds the maxillary artery as the maxillary vein progressively forms into a distinct vessel towards the posterior of the infratemporal fossa. The pterygoid venous plexus has numerous communications in all directions. Significant to possible ocular complications, this plexus communicates superiorly with the cavernous sinus of the middle cranial fossa through the foramen ovale. The abducens nerve, CN VI, traverses through the lumen of the cavernous sinus itself, whereas the oculomotor, trochlear, ophthalmic, and maxillary divisions of the trigeminal nerve, CN III, IV, V₁, and V₂, respectively, are contained within the lateral wall of the sinus. Because veins are a low-pressure vascular system and there are no valves in the veins of the head and neck region, an intravenous injection of local anesthetic could flow from the pterygoid venous plexus into the cavernous sinus and produce the effect of innervation into the orbit, particularly of the motor nerves to the extraocular muscles, CN III, IV, and VI, and the parasympathetic innervation for visual acuity accommodation contained within the oculomotor nerve. This pathway offers a good explanation for the significantly higher frequency of diplopia versus all other ocular complications for all injection techniques (77.8% for diplopia vs 38.1% or less

for all other complications).²⁰ Because the abducens nerve, the motor innervation to the lateral rectus muscle, would be most readily and completely bathed by entry of an anesthetic solution into the cavernous sinus, this pathway may also explain why case reports of diplopia are more often than not accompanied by simultaneous reports of impaired abduction of the lateral rectus muscle.^{20,30}

The pterygoid venous plexus also has a direct communication with the orbit anteriorly through the inferior orbital fissure, forming a second pathway by which intravenously injected local anesthetic could affect orbital innervations. This pathway allows anastomoses between the pterygoid venous plexus and the infraorbital and inferior ophthalmic veins, as well as smaller venous branches, of the orbit. Other, less direct venous communications also exist from the anterior aspect of the orbit. There are venous communications anteriorly between the inferior and superior ophthalmic veins and the facial vein at the medial corner of the eye, which in turn connects to the infraorbital vein of the midface just below the orbit. The infraorbital vein drains posteriorly into the pterygoid venous plexus. The facial vein also anastomoses with the deep facial vein, which drains from the superficial midface region posteriorly into the pterygoid venous plexus. Because the veins of the head do not have valves, venous blood may easily flow both forwards and backwards. This means that because of the numerous anastomoses of veins between the infratemporal fossa and the orbit, there are several pathways by which an intravenous injection of anesthetic solution into any of the veins within the infratemporal fossa could pass into the orbit. Veins are more numerous and more anatomically variable than arteries, which may further increase the likelihood of venous transmission of anesthetic into the orbit.

One further consideration is that the walls of veins may be more easily penetrated by a needle than the walls of arteries because the walls of veins have significantly fewer smooth muscle cell layers and larger lumens than their corresponding-sized arteries. Upon initial contact of a needle with a blood vessel wall, the smooth muscle reflexively contracts. This is one potential cause of the facial blanching that is sometimes observed following dental injections. Fortunately, this contraction may also make the blood vessel wall more difficult for the needle to penetrate through, unless the blood vessel is pressed up against bone or stretched taut within the tissue space. Intravenous injections would be expected to exhibit less immediate and/or less dramatic signs and symptoms of complications due to the lower blood pressure to potentially push the anesthetic into neighboring regions, and postural changes may quickly reverse the direction

of intravenous anesthetic flow, because of the absence of valves, and thus minimize any adverse effects.

DIFFUSION THROUGH TISSUE PLANES

Skeletally, there is communication from the infratemporal fossa through the pterygomaxillary fissure into the deeper-lying pterygopalatine fossa, and then up into the orbit via the inferior orbital fissure (the same pathway of venous communication between the infratemporal fossa and the orbit). Some authors have proposed the possibility of an anesthetic solution traversing through these openings by means of the natural fascial tissue planes.^{13,28–30} The anesthetic solution could then potentially exert a direct action on the ciliary ganglion and its parasympathetic innervations to the eye and to the lateral rectus muscle and/or the abducens nerve. Peñarrocha-Diago and Sanchis-Bielsa¹³ advocated that direct diffusion of anesthetic solution along this pathway is the most likely cause for ocular disorders following injections, and that the proximity of the lateral rectus muscle to this pathway into the orbit explains why this muscle is more frequently affected than any other extraocular muscle. In contrast, Scott et al²⁸ felt that a direct diffusion pathway, although possible, was not likely because of the distances involved. However, it is possible that misdirection of the needle or needle deflection during the injection could result in deposition of an anesthetic solution much closer to the opening of the pterygomaxillary fissure than expected. The greater depth of needle penetration inherent with block injections such as the IA or posterior superior alveolar technique increases the potential for needle deflection to occur, which may at least partially account for the higher number of complications reported with block injection techniques. Medial deposition of the local anesthetic solution was proposed as a possible factor in a case report of unusual middle ear problems following a Gow-Gates block injection.³⁴ Additionally, several authors have speculated that the greater diffusion properties of articaine could result in an increased incident of ocular complications via direct diffusion into the orbit.^{13,19,27,31}

AUTONOMIC NERVOUS SYSTEM PATHWAY DISRUPTION

Steenen et al²⁰ suggested that a needle could injure the surface of an artery within the infratemporal fossa, activating the sympathetic nerve plexus on the arterial wall and creating a vasospastic impulse that could pass back along the maxillary arterial wall to the external carotid artery, and then back up the internal carotid

artery to the ophthalmic artery and on into the orbit. This idea of “autonomic dysregulation” is largely supported by the roughly 10% occurrence of facial blanching that accompanies ocular complications. Van der Bijl and Meyer¹¹ proposed that in cases where Horner syndrome–like signs and symptoms such as ptosis, myosis, and papillary dilation have been reported, direct anesthetic blockage of the sympathetic nerve plexus on the surface of the arteries may be responsible. It is possible that in some cases these mechanisms may be the primary cause for the reported signs and symptoms whereas in other cases these mechanisms could be operating simultaneously with others of the cited hypotheses.

HYPOTHESES FOR THE CURRENT CASE

The advantages of the Gow-Gates mandibular quadrant block injection technique are reported to be a high success rate (>95%) for those experienced with the technique combined with a minimal positive aspiration rate (approximately 2% vs 10–15% with the conventional IA nerve block) and few reported postinjection complications.^{35,36} Very few reports could be found specifically pertaining to an ocular complication following a Gow-Gates mandibular block.^{37,38} Authors of a 1989 case report, which described temporary CN paralysis for III, IV, and VI, concluded that this complication was due to an intravenous injection into the pterygoid venous plexus with retrograde flow of the anesthetic solution to the cavernous sinus.³⁷ This article went on to emphasize the importance of understanding the local anatomy and the proper technique for delivery of the Gow-Gates injection. The target site for deposition of the anesthetic solution for a Gow-Gates injection is the lateral side of the condylar neck, just below the insertion of the lateral pterygoid muscle, in a relatively avascular fatty area.^{34,35}

The hypothesis of retrograde flow of anesthetic solution within the vascular system is the most commonly proposed cause for ocular complications following dental injections found in the literature. Although the aspiration tests in this case were negative, based upon the symptoms presented by the patient, it is possible that an accidental intravascular injection could have occurred. It is possible that the beveled opening of the needle could have been within a blood vessel, but the negative pressure of aspiration pulled the blood vessel wall against the opening so that no blood entered the syringe.³⁹ Although the injection site for the Gow-Gates technique is typically relatively avascular, anatomical variation in this patient may have placed blood vessels, more likely to be veins rather than arteries, at a higher than normal

level, and therefore at risk with this high injection technique. The sensation that the patient described of “hot water flowing up his face” lends further credence to the suggestion that the anesthetic may have traveled within the vascular system even though no signs of facial blanching were observed.

The use of a 25-gauge needle likely might have prevented the needle from being deflected or directed medially. Because a 27-gauge needle was used, it may have placed the needle close to the opening of the pterygomaxillary fissure and the numerous arteries and veins passing through this opening. This would have increased the possibility of an accidental intravascular injection or of the anesthetic solution diffusing directly through the inferior orbital fissure. The hypothesis of possible direct diffusion of the anesthetic into the orbit through the inferior orbital fissure is supported by the patient’s report of numbness of the tissue below his eye and along the side of his nose (anesthesia of the infraorbital nerve, which also enters the orbit through the inferior orbital fissure). However, effect on the lateral rectus muscle, either directly or via the abducens nerve, was not observed, as might be expected to occur via this pathway.^{20,30} Thankfully, additional symptoms were not observed, but this may be attributable to the total volume of 0.9 mL given, which may account for the short duration of symptoms.

Because no signs of extraocular muscle impairment or ptosis were reported, it is likely that anesthesia of the parasympathetic innervation to the ciliary muscles, which disrupted the patient’s ability to accommodate, was the cause of his blurred, double vision. This may have resulted from vascular transmission of the anesthetic via intravascular injection or by direct autonomic dysregulation via contact of the needle with a blood vessel wall. Unfortunately, it is not possible to know with any certainty what the true cause of this reported ocular complication was, yet this complication can be recognized as one of multicausal origin.

CONCLUSION

Risks and complications have been described for every block and infiltration technique and for every anesthetic agent, with or without a vasoconstrictor additive. Although it is quite rare to have ocular complications following dental injections, such occurrences are obviously very alarming to both the patient and the dentist. And although the exact etiology of these events may not be clear, the preponderance of anatomical information points to some type of accidental intravascular injection, be it intra-arterial or intravenous, as being the most likely cause. For this reason, it is highly recommended to

always aspirate prior to depositing the local anesthetic solution, and some authors recommend aspirating in at least 2 planes prior to anesthetic deposition to further minimize the risk of an intravascular injection.³⁹ Moreover, it must be recognized that even with careful aspiration techniques, anesthetic may still be accidentally injected into a blood vessel.

Immediately communicating with all patients after each injection helps to identify any potential risks or complications. In the event of a patient reporting an ocular complication, such as the diplopia in this case, or any other complication, treatment should be stopped immediately and the patient carefully examined and assessed for emergency care. With ocular complications, the eye movements should be assessed and carefully monitored over time. The patient can be reassured that these are usually transient events that resolve completely within a few minutes, or at least within the duration of the anesthetic action.^{19,20,30,33} Timely assurance to the patient that mild complications will resolve quickly may help to ease anxiety and minimize discomfort. It is best to cover the affected eye or to tape the eye shut to minimize the strain and disorienting effects that double vision may have on the patient.^{19,30} If it is mutually decided to continue with treatment and if any ocular impairment remains at the end of treatment, the patient should be escorted to a specialist for ophthalmological evaluation, or if the symptoms are improving, at least be escorted home and advised not to drive or operate any machinery until normal vision has returned.^{30,33} It is important to query all patients at the end of their treatment appointment as to their well-being. This point was emphasized by Blaxter and Britten,²¹ who reported a case where a patient was involved in a minor automobile accident following development of diplopia during a dental appointment. From the article by Blaxter and Britten,²¹ it is not clear whether the patient was unaware of his blurred vision at the end of the appointment or whether he failed to inform his dentist of the problem.

All complications should be carefully detailed in the treatment notes, along with any follow-up care or referrals. Clearly documenting the signs and symptoms and longevity of the complication in the patient’s treatment record may help prevent future complications and may also provide a better understanding of possible patient anatomical variations. Later the same day, telephone the patient or send an e-mail to check the patient’s welfare to maintain rapport.

Because these complications may to some degree be related to individual anatomical variations, it is worthwhile to consider using a different injection technique if it is necessary to anesthetize the same region at a future appointment. Dental providers should be comfortable with the extraoral and intraoral anatomical landmarks for

multiple injection techniques, and it is best to practice these alternative techniques on a regular basis. It is also important that dental providers and ophthalmologists be aware of the possibility of ocular complications and be prepared to properly manage and reassure their patients should they occur.

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