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Complications Associated with Local Anesthesia in Oral and Maxillofacial Surgery

Basak Keskin Yalcin

Abstract

One of the important attempts in clinical oral surgery practice is to maintain safe and effective local anesthesia. Dental procedures are frequently performed under local anesthesia; thus, drug-related complications are often encountered. It is mandatory to have a preoperative evaluation of the patient and choosing the proper local anesthetic agent. Various complications including hypersensitivity, allergy, overdosage, toxicity, hematoma, trismus, paresthesia, or neuralgia can be observed during practice. Therefore, the practitioner should be aware of the possible complications and management methods. The aim of this chapter is to review the preoperative and postoperative complications associated with the local anesthetic in oral and maxillofacial surgery practice. The prevention of measures and treatment of the complications is also emphasized.

Keywords: local anesthesia, complication, local complications, systemic complications, treatment

1. Introduction

Local anesthetic agents have been used in clinical dentistry to allay or eliminate pain associated with invasive operations as early as the nineteenth century [1]. Local anesthetics are used routinely also in oral and maxillofacial surgery. Despite that local anesthetics are reliable and efficient drugs, the risks that practitioners need to be aware of were also reported [2].

Complications associated with local anesthetics can be evaluated systemically and locally. Common systemic reactions due to local anesthesia are reported as psychogenic reactions, systemic toxicity, allergy, and methemoglobinemia. Common local complications associated with local anesthesia are reported as pain at injection, needle fracture, prolongation of anesthesia and various sensory disorders, lack of effect, trismus, infection, edema, hematoma, gingival lesions, soft tissue injury, and ophthalmologic complications [2, 3].

This chapter is presenting the local and systemic complications associated with the local anesthetics used in oral and maxillofacial surgery. The prevention of complications and management methods are also emphasized.

2. Classification and chemical structure of local anesthetics

Local anesthetics can be classified according to their chemical structure, a rate of onset, potency, and duration of action. Chemically, they are either amino esters or amino amides (i.e., an aromatic, lipophilic ring connected to a hydrophilic amine group by an intermediate chain containing either an ester or amide linkage). Ester local anesthetics are hydrolyzed in the plasma by pseudocholinesterase into para-aminobenzoic acid (PABA) and other derivatives, whereas amide-type local anesthetics are metabolized by the liver. The rate of hydrolysis has an effect on the potential toxicity of a local anesthetic. Allergic reactions that occur in response to ester local anesthetics usually are related to para-aminobenzoic acid, which is a major metabolic product of many ester local anesthetics. The rates of biotransformation of amide group lidocaine, mepivacaine, etidocaine, and bupivacaine are similar. Articaine, which contains both amide and ester, metabolizes both in the liver and blood. The ester group (benzoic acid esters) includes cocaine, procaine, chlorprocaine, tetracaine, and benzocaine. The amide group includes lidocaine, mepivacaine (Carbocaine), prilocaine (Citanest), bupivacaine (Marcaine), etidocaine (curanest), dibucaine (nupercaine), and ropivacaine (Naropin).

Ester local anesthetics are not available in dental cartridges essentially because of several reasons such as the lack of efficacy, the potential for allergenicity, and the advantages of amino amides [4–7].

3. Systemic reactions due to local anesthesia

3.1 Psychogenic reactions

This psychogenic answer is associated with either the patient's body counterbalance to an anxiety-inducing situation or due to adrenaline secreted by the vasoconstrictor agent. As a result of mood changes, heart rate, respiratory rate, and blood pressure are altered. Patients often have a blush or erythema which mimics allergic reactions, hyperventilation, nausea, and vomiting [3]. It is important to understand the patient and make them relax. In more severe cases, these reactions should be maintained as syncope and hyperventilation. For preventing psychogenic reactions, the patient should be relaxed before administering local anesthetic injections. Using oral sedatives is an efficacious method to manage dental fears. Initial dosage should be dependent on patient health, age, weight, and duration of the operation. For healthy adult patients in short-term operations antihistamine-diphenhydramine (Benadryl) 50 mg 1 hour prior to the operation, moderate length (1–2 hours) operation benzodiazepine, triazolam (Halcion) 0.125–0.5 mg 1 hour before the operation triazolam, for longer duration (2–4 hours) benzodiazepine such as lorazepam (Ativan) 1–4 mg may be given 1–2 hours prior to the operation or 30–60 minutes prior for the sublingual preparation may be described and given. Pharmacologically, mildly and moderately anxious dental patients can be managed using sedation or extremely anxious or phobic patients using general anesthesia [8, 9].

3.2 Systemic toxicity

Local anesthetic systemic toxicity develops when a sufficient (toxic) concentration of anesthetic drug in the blood level reaches to the central nervous system and cardiovascular systems.

Initial symptoms are characterized by central nervous system signs such as excitation, convulsions, followed by loss of consciousness and respiratory arrest. These symptoms are often accompanied by cardiovascular signs such as hypertension, tachycardia, and premature ventricular contractions. The clinical signs and symptoms usually show objective symptoms such as quick talking, flicker, and tremor in the extremities [10, 11].

Predisposing factors are associated with age, weight, other drugs, gender, the presence of disease, genetics, vasoactivity, concentration, dose, route of administration, the rate of injection, vascularity of the injection site, and the presence of vasoconstrictors [7].

In order to prevent systemic toxicity, the patient should be evaluated. The volume of local anesthesia should be decreased, young or lightweight patients should not be treated all four quadrants at one visit using local anesthetic alone; accurate and slow injection technique, adjustment of dosage divided administration and aspirating technique, using agents with low toxicity such as ropivacaine and levobupivacaine, and performing an aspiration test are recommended [11]. Preventing from a toxic dose complication, it should be evoked that for healthy adults, the suggested maximum safe dose of 2% lignocaine in 1:80,000 adrenaline is four-and-a-half 2 or 2.2 mL cartridges (180–198 mg lignocaine); for 3% prilocaine and felypressin 0.03 i.u./mL, the maximum safe dose is 400 mg (six 2 mL cartridges) [12]. Another strategy to reduce toxicity is using the guideline of 1/10th cartridge per kilo as a rough guide to the maximum dose [13].

Dentists should be aware that excessive doses of topical anesthetics while these agents are more concentrated to facilitate infiltration may lead to toxic effects, particularly in children.

Treatment at the office includes airway support, administration of 100% oxygen, supine positioning, and protection from injury in the event of seizure activity, treating convulsions (benzodiazepines or thiopental; propofol cannot be used in patients with unstable blood pressure, heartbeat) [14]. If severe hypotension arrhythmia occurs, administration of the infusion of a 1.5 mL/kg 20% lipid emulsion over approximately 1 minute and then starting with continuous application at 0.25 mL/kg/min = 1000 mL/h. Studies have reported a resuscitation effect at a total dose of ≤ 10 mL/kg; therefore, 12 mL/kg can be used as an approximate estimate for the maximum dose. The adrenaline dose should be based on resuscitation guidelines such as those of the American Heart Association. The American Society of Regional Anesthesia and Pain Medicine standard of <1 $\mu\text{g/kg}$ does not need to be strictly adhered to [11].

3.3 Allergy

Allergy is also known as hypersensitive reactions, initiated by immunological mechanisms acquired through exposure to a specific allergen; re-exposure to which produces a heightened capacity to react. The prevalence of allergic reactions to amide group local anesthetics is rare. It is predicted that less than 1% of all complications are caused by an allergy. Many of the complications doubt to be allergic are actually anxiety-induced reactions [15].

Ester-type local anesthetics are more allergenic than amide-type local anesthetics. Therefore, amide-type anesthetics are broadly used, among which lidocaine is the most commonly used for dental anesthesia epinephrine involving form. Adverse reactions to local anesthesia are caused by preservatives (e.g., methyl-p-hydroxybenzoate), antioxidants (e.g., bisulfate), antiseptics (e.g., chlorhexidine), vasoconstrictor (e.g., sulfites), and other antigens such as latex, as well as local anesthetic drugs themselves [5].

Allergic reactions may include mild symptoms, such as urticaria, erythema, and intense itching, as well as severe reactions in the form of angioedema and/or respiratory distress. Even more severe life-threatening anaphylactic responses include symptoms of apnea, hypotension, and loss of consciousness [15].

In order to diagnose allergies, the skin prick test is the most endorsed. When skin prick test results are determined to be negative, intradermal testing should be performed for patients who have a history of allergy to local anesthetics intradermal tests become obligatory [15, 16].

The following treatments a local anesthetic patient had tested negative in the allergy tests, should be used.

The initial treatment for an allergic reaction in office at the first step should be the removal of the causative agent. For the management of mild symptoms, oral or intramuscular antihistamine-diphenhydramine (Benadryl), 25 or 50 mg, should be given. Additionally, hydrocortisone cream may be prescribed to relieve skin itching or erythema. In life-threatening cases basic life support, intramuscular or subcutaneous epinephrine 0.3–0.5 mg, and hospitalization services should be given.

Anaphylaxis is an acute potentially life-threatening hypersensitivity reaction. The clinical symptoms of anaphylaxis are depending on the organ systems involved. Uncontrolled co-existing asthma, mast cell disorders, and patients with specific allergens such as peanut and tree nut allergy are the risk factors for anaphylaxis. In emergency management of anaphylaxis in the office, due to guidelines of the Australasian Society of Clinical Immunology and Allergy should be in these steps, the patient should lie flat, but also in the case of breathing difficulty, the patient is allowed to sit. Adrenaline 1:1000 dilution (0.01 mg/kg up to 0.5 mg per dose) should be administered intramuscular with 1-mL syringes, 21 gauge needles, and should be repeated every 5 minutes as needed. Another recommendation for epinephrine is or children and adults who weigh 30 kg or over is 0.3 mg. For those weighing 15 to 30 kg, the epinephrine dose is 0.15 mg. The use of adrenaline auto-injector can also be chosen, which is carried mostly by heavy allergic patients themselves.

Adrenaline should be administered for anaphylaxis by intravenous (IV) route only in the case of profoundly hypotensive patients or patients who develop a cardiopulmonary arrest or those who fail to respond to multiple doses of IM adrenaline because of the potential cardiovascular adverse effects of IV administration of adrenaline [17, 18].

Estelle and Simons evaluated evidence-based pharmacologic treatment of anaphylaxis. They agreed using epinephrine at the first step intramuscular in the treatment of anaphylaxis. But the use of antihistamines and glucocorticoids is controversial. Some authors claim using antihistamines is not effective because they are not effective on upper or lower airway obstruction, hypotension, or shock, while others advocate that these drugs decrease the side effects urticaria, flushing, headache, hypotension, and rhinorrhea. In the World Allergy Organization survey, glucocorticoids were reported to be the second most widely available medications (after epinephrine) for anaphylaxis treatment globally, even though some claim glucocorticoids have no proven benefit in anaphylaxis [19].

As a result first step of treatment must be epinephrine additionally glucocorticoids and antihistamines may use to treat severe systemic reactions.

3.4 Methemoglobinemia

Methemoglobinemia is a unique dose-dependent reaction where the iron in hemoglobin is stabilized in the ferric (Fe^{3+}) form, unable to attach oxygen, leading

to tissue hypoxia and causing a varying degree of cyanosis. Methemoglobinemia can be either inherited or acquired [20].

The risk of methemoglobinemia increased in infants and the elderly. Patients with underlying health problems; liver cirrhosis, with underdeveloped hepatic and renal function; heart disease; and pulmonary disease (chronic obstructive pulmonary disease, pneumonia) are under the risk of methemoglobinemia. When administered in excessive doses, the local anesthetics mostly prilocaine and benzocaine (90% of reported cases) and barely lidocaine and articaine may also lead to methemoglobinemia [21].

Symptoms of cyanosis will be observed in nail beds and mucous membranes. In more severe cases, headache, dizziness, fatigue, dyspnea, and tachycardia are seen. For diagnosis in the dental clinic, pulse oximetry and in-hospital arterial blood analysis play an essential role [21].

Management of methemoglobinemia begins with supplemental oxygen (100%) immediately. As a guideline, methylene blue, which is a heterocyclic aromatic chemical compound increasing the rate of conversion of methemoglobin to hemoglobin, may be given to a symptomatic patient. For severe cases, hyperbaric oxygenation may also be used if available. Methylene blue should be administered in 1 to 2 mg/kg doses, given as 0.1 mL/kg of a 1% solution (10 mg/mL) intravenously over 5–10 minutes every hour up to a 7 mg/kg maximum. Repeated doses may be necessary within 30–60 minutes of the initial dose [22, 23]. Guay summarizes 242 cases of methemoglobinemia complications related to dental local anesthetics lidocaine, bupivacaine, cocaine, mepivacaine, prilocaine, and tetracaine in children and adults. He concluded that benzocaine should be out of usage. In a specific patient group, in children younger than 6 months, in pregnant women, or in patients taking other oxidizing drugs, prilocaine should not be used. The dose should be limited to 2.5 mg/kg [21].

4. Local complications associated with local anesthesia

4.1 Pain on injection

Pain on injection can be due to specific circumstances such as a temperate of the solution, velocity of injection, dull needles, needles with barbs, or aggressive insertion of the needle, damaging soft tissues, blood vessels, nerves, or the periosteum and causing more pain and other complications.

The burning is dependent on the rate of injection and the acidity of the solution. Lidocaine causes an intense burning sensation when injected locally. When the needle penetrates a nerve, the patient may also feel a sudden “electric” shock, suddenly moving the head, with the risk of self-inflicted damage [24].

In order to prevent discomfort, topical anesthetic application, warming anesthetics to body temperature, using a smaller-gauge needle (27 gauge), switching to a fresh needle when you have to inject multiple times in the same lesion or when you have multiple injection sites, and injecting slowly and with low pressure which reduces pain are done. A rate of 30 seconds per mL of solution is recommended. An inadequate injection site can lead to an intramuscular or intraneural injection blunting of the needle, side of injection anatomic structure (palate) it is unacceptable to feel a little pain during injection [13, 24, 25].

4.2 Needle fracture

Needle breakage in the oral cavity after local anesthesia is a rare complication, since the establishment of non-reusable, stainless steel dental local anesthetic

needles. In most cases, needle fracture happened with 30-gauge needles and during inferior alveolar nerve block, as a result of either incorrect injection technique, improper choice of hypodermic needle magnitude, or unexpected motion of the patient or assistants [26].

In order to prevent needle fracture, first the injection needles should be checked; 30-gauge and short needles should not be used for inferior alveolar nerve block in adults or children (25–27 should be chosen). Needles should not bend while inserting them into soft tissue [26, 27].

In the case of a broken needle, if visible, it should be removed immediately with a hemostat. If this is inaccessible, computed tomographic (CT) scanning should be taken to ensure the location of the needle, and under general anesthesia, the patient should be operated. In the literature for the removal of the fragment, mostly superficial mucosal incision perpendicular to the trajectory of the needle followed by blunt supra-periosteal dissection to spare vital structures is recommended [28, 29].

Acham et al. in 2018 made an analysis of the literature complication of needle fracture following dental local anesthesia on 36 reports and 59 needle breakage events; they concluded that three-dimensional imaging techniques should be taken to see the broken fragment and also surrounding structures like vessels and the parotid gland. It is important because 27 out of 57 cannula fragments were located in the pterygomandibular space, and the choice of the removal of the fragment, whether general or local anesthesia, should be dependent on the patient's systemic condition [30].

4.3 Prolongation of anesthesia and various sensory disorders

Prolonged anesthesia, paresthesia, or neuralgia may occur following dental local anesthetic blocks. This may be temporary, where after a few days, weeks, or months, sensation returns or it may be permanent [31]. This mostly involves nervus lingualis or nervus mandibularis or both [32]. The nerve may be damaged during injection by direct injury, or the needle may damage the intraneural blood supply, resulting in a hematoma, or the needle may traumatize the medial pterygoid muscle which results in trismus. Neurotoxicity of the local anesthetic is another theory for nerve damage [33]. Procaine and tetracaine cause more damage than bupivacaine or lidocaine [34]. Paresthesia or neuralgia complication is mostly transient but may also be permanent if the anesthetic solution is injected directly into the nerve. Due to a numb feeling, the patient may have discomfort such as tongue biting, drooling, loss of taste, and speech impediment. Sullivan et al. conducted a randomized, double-blind, placebo-controlled trial on 496 patients with Bell's palsy. They maintain treatment with steroids within 3 days after onset quite advances the chance for full recovery at 3 or 9 months [35]. Piccinni et al. conducted an analysis of reports to the FDA Adverse Event Reporting System; about 573 cases of paresthesia and dysesthesia after local anesthetics between 2004 and 2011 were performed. They concluded that the use of prilocaine, articaine, or both drugs has a higher risk of paresthesia [36].

If a nerve is damaged due to dental local anesthesia, the first treatment should be managing the pain. In order to decrease local anesthesia-dependent nerve injury, avoiding high concentration of anesthetic agent for inferior alveolar nerve blocks (use 2% lidocaine as standard), preventing iterative injections, and avoiding inferior alveolar nerve blocks are done by using high concentration agents (articaine) infiltrations only. The use of a low daily dose of multivitamin B, to regaining nerve healing and function, has been recommended [37, 38].

4.4 Lack of effect

Reasons for unsucccess in obtaining local anesthesia can be dependent on anatomical variants, pathological and psychological factors, choice of technique and solution, and poor technique [24].

Anatomical factors comprise accessory nerve supply, alteration in foramen location, atypical development of the nerves (bifid mandibular canals), and bone density [39, 40].

Pathological reasons for the failure of anesthesia are trismus, infection, inflammation, and previous surgery or trauma. Inflammatory diseases altering the pharmacokinetics and pharmacodynamics of local anesthetics cause a response to decrease and unfavorable effects to increase [41].

Local anesthetic failure or difficulty to obtain satisfactory analgesia commonly occurs in the situations with inflammations such as pulpitis and apical periodontitis acute periodontal abscess or pericoronitis [42]. Psychological determinants such as angst and anxiety can also cause local anesthesia failure [39].

Poor technique failure mostly occurs to obtain mandibular anesthesia. If the needle is inserted and advanced too deeply and too far dorsally, the terminal branches of the facial nerve within the deep lobe of the parotid gland are affected. Direct anesthesia to the facial nerve can force a rapid onset that occurs while the anesthetic agent is being injected; reflex vasospasms of the external carotid artery can lead to ischemia of the facial nerve, so facial nerve palsy occurs. The patient is unable to wrinkle the forehead, raise the eyebrow, close the upper eyelid, retract the commissure of the lips to smile, and turn down the lower lip on the affected side. The removal of contact lenses and closing of the eye on the affected side in Bell's palsy prevent corneal abrasion or drying [43, 44].

In most cases, paralysis occurs immediately after mandibular anesthesia injection, but there are also some cases in which paralysis starts lately. Cakarar et al. have a case report for late paralyses. They extracted simple teeth, without any complication, and 1 day after the patient returned with complaints of a weakness of the muscles of the left side of his face. On the examination, they observed Bell's palsy sign on the left side and unilateral expressionless, and there was no pathologic sign in the wound or any herpetic lesions. They consulted the patient with the Department of Ophthalmology and the Department of Physical Therapy and Rehabilitation. For the treatment lubricant eye drop (4 × 1), tobramycin ophthalmic solution (4 × 1) and lanolin eye ointment (during night) supported by eye patch were used. For 4 weeks, galvanic stimulation of the affected side of the facial nerve was performed, and mime therapy was recommended. In 2 weeks all of the symptoms disappeared [45].

If the needle is inserted too high and deep, N. auriculotemporalis will be affected, and the feeling of "numbness" will occur. There has been a report of sudden unilateral deafness following inferior dental nerve anesthesia.

4.5 Trismus

Trismus is defined as a painful circumstance with inability to open the mouth normally. Several factors cause trismus such as multiple injections in a short period of time in the same area, intramuscular injections inside the muscle or trauma to muscles (either the lateral pterygoid muscle or the temporal muscle) which cause hematoma formation and fibrosis, needle fracture in the muscles inserting to styloid process, inaccurate positioning of the needle when giving the inferior nerve block or maxillary posterior injections or inflammation of the masseter and other

masticatory muscles, a low-grade infection, and excessive volumes of local anesthetic solution deposited into a bounded region which cause expansion of tissues. In the acute phase, pain from hemorrhage leads to muscle contraction and limitation of motion.

Once trismus develops, some cases will resolve spontaneously. Progression of trismus to chronic hypomobility and fibrous ankylosis may be prevented by the early institution of treatment consisting of heat therapy; soft diet; prescription of analgesics, anti-inflammatory drugs, antibiotics, muscle relaxants; or physiotherapy. Trismus caused by an infection needs to be treated by antibiotics. Usually, trismus will resolve in 6 weeks, with a range of 4 to 20 weeks.

Awareness of the anatomical landmarks and muscles: palpation of bony anterior ramus for temporalis muscle, pterygomandibular fold for pterygoid muscle, and appropriate angulation of the needle and bone contact before injecting are good methods for avoiding trismus via local anesthesia.

Intraorally the Vazirani-Akinosi technique, the closed-mouth mandibular nerve block technique, or extraoral techniques can provide anesthesia to trismus patients [43, 46].

4.6 Infection

Infection complication is rare since the usage of disposable needles and glass cartridges. Infection may extend to tissues by penetration of the needle through a contaminated tissue, because of the needle being contaminated before an operation or improper preparation of local anesthetic diluted solutions. On the other hand, a latent viral infection may be reactivated due to the trauma of the procedure which may be responsible for neural sheath inflammation.

The area to be penetrated should be cleaned with a topical antiseptic prior to insertion of the needle. Antiseptic mouthwash solutions such as chlorhexidine gluconate should be considered for all regional techniques. The local anesthesia should not be injected through the infected area.

Injecting local anesthesia during the presence of infection is important to increase the pH of anesthetic agent in order to increase efficiency because the infected tissue is more acidic. This process is called anesthetic buffering and leads to patient comfort during injection, fast onset of anesthesia, and lower postinjection tissue injury. Recommendation for treatment of infection is antibiotics (penicillin V 500 mg every 6 hour for 7–10 days), analgesics, heat, drainage, and physiotherapy [2, 31, 47].

4.7 Edema

Swelling of tissues can be due to trauma during injection, infection, allergy, hemorrhage, and injection of irritating solutions.

The management of edema is dependent on the cause. Allergy-induced edema treatment consists of intramuscular epinephrine injection as mentioned above and, additionally, antihistamine and corticosteroid administration and consultation with an allergist to determine the precise cause of the edema. Trauma-induced edema should be managed as a hematoma. For the treatment of edema produced by infection, antibiotics should be prescribed [27].

4.8 Hematoma

Hematoma formation as a complication of local anesthesia is the result of a venous or arterial laceration; intra-arterial blood pressure increase causes effusion of blood into the surrounding soft tissues. While injecting, if there is a high pressure, it may be a warning injecting against the bloodstream. The size of a hematoma

depends on the density and compactness of the affected tissue; when a vein rupture is concerned, hematoma does not necessarily occur. Discoloration on the area, a bruise may accompany hematoma [48].

From the anatomical point of view, different nerve effects cause hematoma on specific regions such as anterior superior alveolar (infraorbital) nerve block below the lower eyelid, incisive (mental) nerve block at the chin area, buccal nerve block or any palatal injection within the mouth, and posterior superior alveolar nerve block extraoral in the lower buccal region of the mandible, intraoral distal to maxillary tuberosity.

Hematoma formation can be prevented by aspirating before injecting the anesthetic solution, by using a short needle and a minimum number of needle penetrations into tissues. When swelling forms immediately after injection, localized pressure should be applied with a minimum of 2 minutes. This will stop the hemorrhage.

Both swelling and discoloration usually subside in 10 to 15 days. Ice packs should be held for the first 24 hours after surgery following which intermittent hot moist packs can be used to resolve the condition and massage therapy using a heparin cream. Antibiotics should be prescribed if the hematoma is large in order to prevent the development of a wound infection [14, 49].

4.9 Gingival lesions

Gingival lesions consist of recurrent aphthous stomatitis, and herpes simplex can occur intraorally after a local anesthetic injection or after any trauma to the intraoral tissues. The exact mechanism is unknown, but any trauma to tissues by a needle may activate the latent form of the disease process that was present in the tissues with previous injection.

No management is necessary until there is severe pain. In order to relieve pain, topical anesthetic solutions (e.g., viscous lidocaine) may be used on affected areas. A concoction of identical amounts of diphenhydramine and milk of magnesia rinsed in the mouth effectively covers the ulcerations and provides relief from pain. Triamcinolone acetonide without corticosteroid can remedy pain [14, 27].

4.10 Soft tissue injury

Lip or tongue biting or chewing can occur on children with special needs or disabled patients, following dental local anesthesia with the unfamiliar sensation of being numb [50]. Shorter-acting local anesthetics such as plain mepivacaine should be chosen, and the patient or the guardian should be warned about eating, drinking hot fluids, and biting on the lips or tongue to test for anesthesia; cotton rolls can be placed between the teeth and soft tissues to prevent chewing. In order to accelerate recovery time for sensation, an alpha-adrenergic receptor, phentolamine mesylate (OraVerse), may be injected. For adults, the proposed dosage is 1 to 2 cartridges of phentolamine mesylate (a dose of 0.4 to 0.8 mg), while for children the proposed dosage is 0.5 to 1 cartridge (0.2 to 0.4 mg) [50, 51] Malamed [52].

Swelling may decay after 2 to 3 days. The lesion will heal over the next 10 to 14 days. For pain complains, analgesics may be prescribed and topical local anesthetic gel may be applied to the area.

4.11 Ophthalmologic complications

The most common complications include diplopia (dual vision), ophthalmoplegia (paralysis or weakening of eye muscles), ptosis, and mydriasis (dilatation of

pupil). In extremely rare instances, amaurosis (partial/total blindness) can be seen. All these complications are transient and disappear on interruption of the anesthetic effects [53].

Intraarterial injection or perforation of the vascular wall would stimulate the sympathetic fibers running alongside the internal maxillary artery until reaching the orbit. The intravenous injection could reach the cavernous sinus via the pterygoid plexus and anesthetize the oculomotor, trochlear, or abducens nerves.

Horner's syndrome may occur after an inferior dental nerve block anesthesia because of penetration of the local anesthetic through the lateral pharyngeal and prevertebral spaces, causing barrier of the stellate ganglion [54, 55].

Alamanos et al. conducted a systematic review in 2016 on ophthalmologic complications following dental local anesthesia with 66 reports and 89 cases. They found that the Gow-Gates technique for mandibular block anesthesia is only associated with diplopia, vision impairment is more associated with inferior alveolar nerve blocks than with posterior superior alveolar nerve blocks, and the latter technique has rarely been reported as a cause of amaurosis. Ocular complications in the literature are mostly with an injection of lidocaine [56].

In order to minimize the possible complications, visualization of the regional anatomy, numerous aspirations while injection, and aspiration on at least two planes before administration local anesthetic are performed.

5. Conclusion

Administration of a local anesthetic can be associated with complications of adverse events. In order to prevent local anesthetic complications, the medical history of the patients should routinely be evaluated in details, and effective anxiety management should be performed. Doses of local anesthetics should be always strictly assessed with body weight, and the maximum recommended dosages should be considered. While administrating anesthesia, the painless injection should be performed, avoiding intravascular or intramuscular or direct trauma to the nerve. New developments should be followed by the practitioners to reduce possible complications associated with the local anesthesia.

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