Palatal Perforation: A Rare Complication of Postanesthetic Necrosis

Vidisha Gargi, Ravi Prakash Sasankoti Mohan, Nagaraju Kamarthi, Swati Gupta

Department of Oral Medicine and Radiology, Subharti Dental College, Meerut, Uttar Pradesh, India

Abstract

The everyday practice of dentistry relies heavily on achieving adequate local anesthesia. Even though the safety record of local anesthetic agents is high, complications do occur. Palate is a favorable site for soft-tissue lesions. Various factors such as direct effects of the drug, blanching of the tissues during injection, relatively poor blood supply, and reactivation of the latent forms of herpes can all promote to tissue ischemia and a lesion in the palate. Among various complications, anesthetic necrotic ulcer is a rare and uncommon condition occurring mostly in the hard palate possibly after a local anesthetic infiltration. The ulceration is often deep and shows spontaneous but delayed healing. If proper treatment is not instituted on time, the necrosis can reach deep into the bone causing sequestrum formation and ultimately leading to palatal perforation. Here, we report a case of palatal perforation in a male patient followed by surgical interventions and follow-up.

Keywords: *Anesthetic necrosis, local anesthesia, palatal perforation, sequestrum*

Introduction

Administration of local anesthesia (LA) in the form of either injection or topically is an indispensible practice required during both operative and endodontic procedures.[1] It is an effective and safe means of pain control that allows routine procedures to be undertaken. Although uncommon, adverse local and systemic reactions to local anesthetic occur. These complications are multifactorial in origin and are related to toxic drug overdose, rapid absorption, intravascular injection, etc., Needle breakage, prolonged pain, paresthesia, trismus, hematoma, infection, edema, facial nerve paralysis, sloughing of tissues, and postanesthetic intraoral lesions are some of the complications occurring after administering LA.[2] Other rare complication includes postanesthetic necrosis commonly befalling in the palatal region. This occurs as the palatal mucosa is in close proximity to the underlying bone, thus leading to pressurized deposition of the local anesthetic solution and traumatic needle penetration. Other factors such as the direct effects of the drug directly being administered, blanching of the tissues during injection, a relatively poor blood supply, and the possibility of reactivating the latent forms of a disease process such as herpes may all serve to promote tissue ischemia and lead to tissue necrosis.[3] Trauma induced either by insertion of the needle or the solution itself can lead to burning and swelling of the tissues which could reactivate latent viruses such as herpes virus and cause vasculopathy of large or small artery causing ischemia.[4] Chronic necrosis destroys the palatal bone to leave a bony sequester which eventually leads to palatal perforation. We report here a case of palatal perforation causing oroantral communication secondary to postanesthetic necrosis, and the possible mechanism of this unusual presentation is discussed.

Case Report

A 45-year-old patient reported to the Department of Oral Medicine and Radiology with a chief complaint of difficulty in eating food and speaking for the past 5 days. Medical and family history were nonsignificant as reported by the patient. Dental history revealed an uneventful extraction of upper left back tooth 3 years back. The patient had no adverse habit. The patient was asymptomatic 2 months back but then noticed mobility in the upper left back tooth region of the jaw. History revealed that he had visited a private practitioner 15 days back for extraction of mobile teeth under LA. On investigation, he

How to cite this article: Gargi V, Mohan RP, Kamarthi N, Gupta S. Palatal perforation: A rare complication of postanesthetic necrosis. Contemp Clin Dent 2017;8:501-5.

Address for correspondence:
Dr. Ravi Prakash Sasankoti Mohan,
Department of Oral Medicine and Radiology, Subharti Dental College, Meerut, Uttar Pradesh, India.
E-mail: sasan_ravi@rediffmail.com

Access this article online

Website: www.contempclindent.org

DOI: 10.4103/ccd.ccd_123_17

Quick Response Code:

unveiled that injections were given on the palatal mucosa and buccal vestibule of the left side. Three days after extraction, he noticed a painless ulcer at LA infiltration site, which was refractory to the treatment prescribed by his dental surgeon. After a week, the patient felt mobile bone fragment on the palate, that was easily detached by him. This led to nasal regurgitation of food while eating. Furthermore, his speech was impaired.

Extraoral examination revealed no gross facial asymmetry [Figure 1]. Lymph nodes were nontender and nonpalpable. Intraorally, there was missing 25, 26, 27, and an oval-shaped palatal perforation, measuring approximately 1 cm × 1 cm with well-defined margins was evident on the left side of hard palate in relation to 25, 26 tooth region [Figure 2]. There was the absence of sinus opening, suppuration, and tenderness in that region. Furthermore, no erythematous region was present. Based on the site of defect and clinical appearance, a provisional diagnosis of palatal perforation secondary to postanesthetic necrosis was given. Differential diagnosis considered was perforation secondary to mucormycosis, syphilis, and tuberculosis. Investigations were done to rule out diabetes, bacterial and fungal infections. Patients' erythrocyte sedimentation rate was 20 mm/h, and following tests produced no pathologic results: complete blood count, blood sugar level, liver and renal function tests, serology for syphilis, bacteriologic culture including stains for fungi and acid-fast bacilli staining for tuberculosis.

Cone beam computed tomography was done to visualize bone defect. In the axial and three-dimensional section, a well-defined radiolucency measuring 11.89 mm × 10.32 mm in diameter was seen [Figure 3a and b]. Coronal and sagittal views revealed intact maxillary sinus and discontinuity in the nasal floor [Figure 4a and b].

Treatment was planned and the patient was referred to Oral Surgery Department where nasal floor reconstruction was done in which soft tissues around the defect were incised and were sutured together. After that, full thickness rotational palatal flap was harvested from the opposite side and sutured onto the defect to close it. At last benzocaine, tincture was placed on the bone from where the flap was raised so that it promotes healing by secondary intention [Figure 5]. The patient is on regular follow-up and showed healthy healing of lesion [Figure 6].

Discussion

The literature describes different local complications derived from a local anesthetic.[5] Post-anesthetic necrosis following pressurized injection of local anesthetic solution particularly those containing a vasoconstrictor is one of the complication and are well documented. The palate has a rich blood supply through the greater and lesser palatal arteries which will play a role in wound healing and sustaining metabolism by providing oxygen and nutrients.[6]

Palatal tissues are relatively dense, confined, unyielding, and firmly adherent to the underlying bone. An increase in pressure when the local anesthetic agent is administered rapidly and/or forcefully in the adherent palatal tissues causes pain and soreness in that area. This may provide an explanation into the etiology of such an event, or the absence of a good supply, through vasoconstriction, deprives the tissue of its necessary sustenance resulting in necrosis of the overlying epithelium. The contraction of smooth muscle within the arterial wall during vasoconstriction may lead to transient ischemia of structures distally to the injection site leading to tissue necrosis.[7] Furthermore, vasoconstrictors in local anesthetics reduce the oxygen supply to the injected tissue and promote the buildup of acidic by-products of metabolism. In addition, local anesthetic solutions with vasoconstrictors are adjusted to a lower pH to preserve the vasoconstrictors which however accentuate tissue acidity. Thus, epinephrine

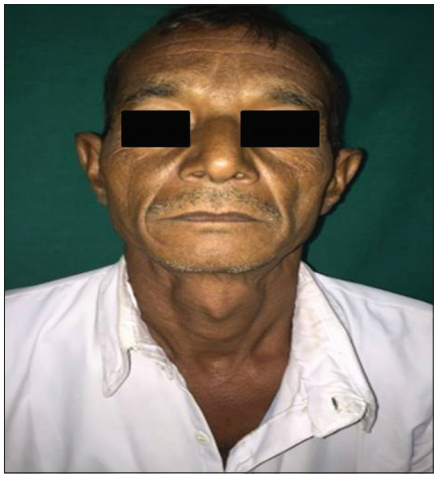

Figure 1: Extraoral view

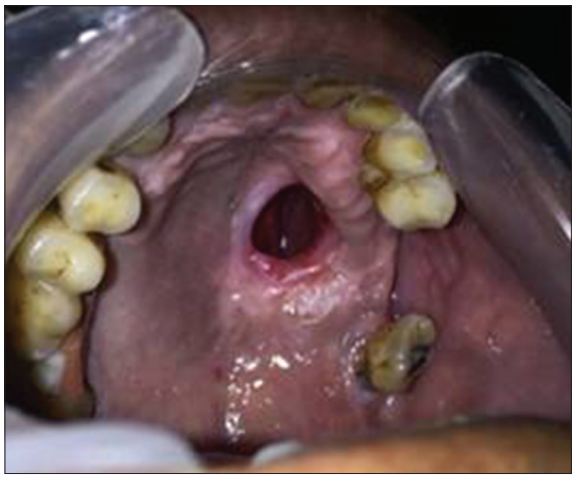

Figure 2: Single well-defined palatal perforation, oval in shape, measuring 1 cm × 1 cm

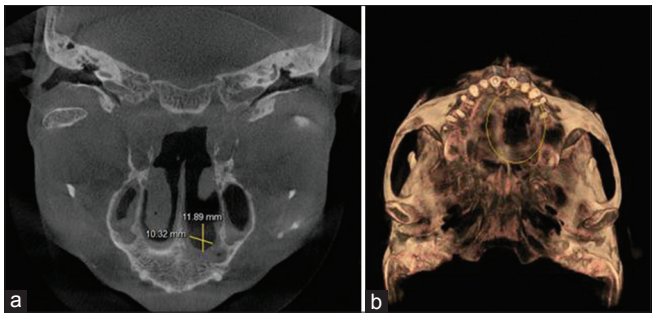

Figure 3: (a) Axial and (b) 3D Cone Beam CTview revealed a well-defined radiolucency on the bhard palate measuring 11.89×10.32 mm

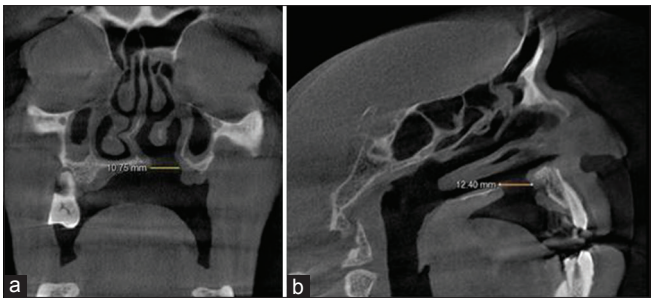

Figure 4: (a) Coronal and (b) Sagital Cone Beam CT view revealed intact maxillay sinus but discontinuous nasal floor

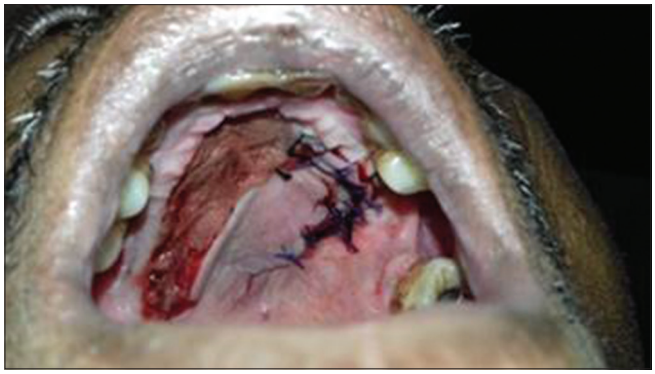

Figure 5: Postoperative view after 1 week

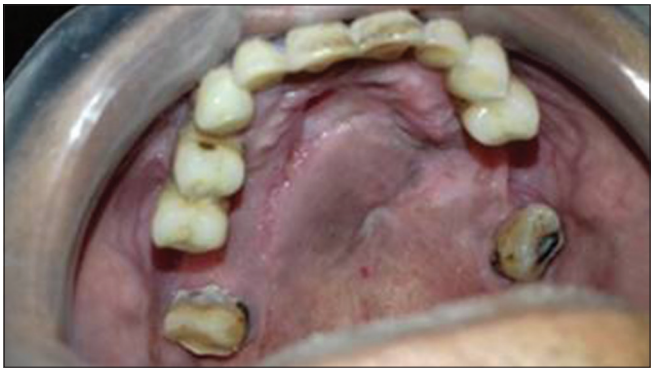

Figure 6: Follow-up image after 2 months

contained in many local anesthetics may be a possible cause of ischemia and secondary necrosis.[3]

Modern local anesthetics are relatively nonirritating to the tissues with the exception, perhaps, of skeletal muscle. Allergic reactions may manifest as circumscribed lesions while other may be derived from the pharmacological effects of the agents used. Allergic reactions to local anesthetics have been greatly reduced by the development of amide local anesthetics, for example, lidocaine. Cases of skin necrosis due to prilocaine have been reported.[8]

Epithelial desquamation may result from the application of a topical anesthetic agent to gingival tissues for a prolonged period of time. In the hard palate, a sterile abscess may form secondary to prolonged ischemia resulting from the use of a local anesthetic solution containing a vasoconstrictor. Other postanesthetic intraoral lesions may result from recurrent aphthous stomatitis and/or herpes simplex which can develop following any traumatic insult to the tissues.[9] Herpes simplex, although most commonly observed extraorally, can develop intraorally on tissues attached to the underlying bone; for example, tissues of the hard palate. Similarly, mucormycosis also causes ulceration of palate, which results from necrosis due to invasion of a palatal vessel. The lesion is characteristically large and deep, causing denudation of the underlying bone.[10]

The palatal perforation can pose a difficult diagnostic dilemma for the clinician [Table 1].[11] The perforation may present with the common characteristics and may be indistinguishable clinically. Emphasis is placed on the importance of obtaining a thorough and comprehensive history and collecting relevant laboratory information.

Table 1: Differential diagnosis of palatal perforation

Developmental	Cleft palate (secondary to maternal alcohol consumption and cigarette smoking, folic acid deficiency, teratogenic drugs, viral infection, corticosteroid use, and anticonvulsant therapy)
Infectious	Tertiary syphilis, tuberculosis, leprosy, typhoid, mucormycosis, actinomycosis, aspergillosis, paracoccidioidomycosis, histoplasmosis, naso-oral blastomycosis, leishmaniasis, diphtheria, rhinoscleroderma
Autoimmune	Lupus erythematous, sarcoidosis, Crohn's disease, and Wegener granulomatosis
Neoplastic	Lymphoma, carcinoma, melanoma, acute lymphoblastic leukemia
Drug related	Narcotics (cocaine, heroin, etc.)
Iatrogenic	Oroantral fistula resulting from procedures like tumor surgery (maxillectomy), corrective surgeries (e.g., septoplasty) or intubation
Rare causes	Rhinolith
	Patients with psychological problems may present with a fictitious palatal perforation

Management of patients with intraoral lesions following the administration of local anesthetic solution is very conservative and consists of reassuring the patient and

Table 2: Recorded cases of postanesthetic necrosis

Authors	Age/gender	Location
Giunta *et al*., 1975[16]	10 years, female	Medial to the pterygomandibular fold
Hartenian and Stenger, 1976[17]	NA	Right posterior hard palate
Ghanem and Suliman 1983[3]	16 years, male	Hard palate
Ranjitha *et al*., 2015[6]	40 years, female	Left posterior hard palate
Gogna *et al*., 2015[7]	44 years, female	Right posterior hard palate
Gupta *et al*., 2016[18]	50 years, male	Left posterior hard palate
Sharma, 2017[4]	16 years, female	Hard palate

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prescribing analgesics and/or topical antiseptics/anesthetic preparations. In many cases, healing occurs within 10 days of the onset of lesion. In certain instances, where ulceration has taken a prolonged course, surgical intervention has been deemed necessary.[12] The common rule for two-layer closure of perforation by turning over the adjacent mucosa for nasal floor reconstruction and rotation mucoperiosteal flaps from intact neighboring palatal mucosa may be applicable in this situation. Large palatal defect can also be closed using a tongue flap. The success is attributed to the excellent vascular supply and the proximity of the donor and recipient sites.[13] Prosthetic obturators avoid nasal reflux, facilitating correct swallowing and sufficient speech performance. The only contraindication to such devices are patients' tolerance to the obturator, as in some cases the obturator size required to fully seal the defect can cause nausea.[14] Another temporary option that is available is the positioning of a silicone button to close the perforation and thus improve the air flow in the nose and reduce progression of local necrosis.[15]

Although not uncommon, a case of an inferior alveolar block resulting in a postanesthetic necrotic defect has also been reported where deep punchedout lesion medial to the pterygomandibular fold was present following repeated doses of anesthesia given 2 months previously.[16] A case of chronic nonspecific palatal ulceration after 5 days has been reported following greater palatine nerve block.[17] Another case reported one large and small palatal ulcerations, each covered by a necrotic slough after palatal injection.[3] Yet another case of acute necrotic ulcer on the palate in a 16-year-old girl following greater palatine nerve block was reported.[4]

To minimize the incidence of palatal lesions following the administration of LA, the following precautions are recommended:[7]

1. Knowledge of the proper anatomy of the area, before the administration of the anesthetic, is a must to avoid local complications
2. Topical anesthetic preparation should be used according to the recommendation of the manufacturer. Application should be limited to 1–2 min to maximize the effectiveness and minimize toxicity
3. Anesthetic solutions containing relatively high concentrations of epinephrine (i.e., 1:50,000; 1:30,000) should be used with caution.

As an alternative, anesthetic solutions not containing a vasoconstrictor, such as 3% mepivacaine, may result in effective palatal anesthetic without soft-tissue necrosis.

Conclusion

The anesthetic necrotic ulcer of the palate leading to palatal perforation is rarely reported in the literature. To our best knowledge, only six cases of postanesthetic palatal necrosis have currently been reported till now [Table 2]. Hence, it should be considered in the differential diagnosis of palatal perforation cases wherein the patient gives a history of previous extractions or administration of LA. It is advisable to avoid undue pressure while administering LA in tissue firmly bound to the underlying bone or to avoid vasoconstrictor containing LA. Mepivacaine, which has a less vasodilation effect, can be considered as an alternative depending on the duration of anesthesia required and the site to be injected.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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