Case Report - Minor oral Surgery

Postanaesthetic Aseptic Palatal Necrosis - A Case Report

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Abstract

Rationale: In dentistry, the most common procedure to be applied is administration of a local anaesthetic agent. It is impossible to practice dentistry without local anaesthesia. In the oral cavity, the palatal mucosa is tightly adherent to the palatal bone and there is little space for anaesthetic solution to be deposited. If local anaesthetic is forcefully injected by the syringe, it creates pressure on blood vessels and causes palatal necrosis. Patient Concern: Here, we present a case report of a 25-year-old male patient who reported to us with chief complaint of an ulcer on the palate. Diagnosis: Patient was diagnosed with postanaesthetic aseptic palatal necrosis. Intervention: The patient was managed conservatively using copious irrigation and a palatal acrylic splint. Outcome: On the 6th month follow-up, the lesion was completely replaced by healthy mucosa. Take-away Lessons: We should avoid forceful injection of local anaesthetic agent to prevent further postoperative complications.

Keywords: Anaesthesia, erythematous, necrosis, palatal acrylic splint, palatal mucosa

Introduction

In our regular dental practice, local anaesthetic agent plays an important role. Without local anaesthesia, it is impossible to do a healthy dental practice. It is mainly given as local infiltration and nerve blocks.[1] In general, there are fewer complications encountered caused by local anaesthesia, but it cannot be denied that local anaesthesia does not cause any complications in dental practice. However, possible complications such as postoperative infection, prolonged pain, trismus, needle breakage, paraesthesia, haematoma, oedema, facial nerve paralysis, sloughing of tissues, and postanaesthetic lesions.[2] Due to its adherent feature, local anaesthesia is forcefully injected by the syringe, which creates pressure on blood vessels and causes palatal necrosis.[3] Here, we present a case report of a 25-year-old male patient who was diagnosed with palatal necrosis.

Case Report

A patient reported to us with a chief complaint of ulcer on the palate on the left side for 15–20 days. Patients provided a history of root canal treatment of the upper left tooth region that began around 25 days ago, after which he observed an ulcer that grew to its current size within 5–10 days. He reported to the dentist on the next visit and was on multivitamin and antibiotics till he reported to us.

During clinical examination, we observed a necrotic palatal mucosa of approximately 3 cm in diameter, grayish-black, and margins were erythematous [Figure 1]. Access cavities were opened in 24 and 26. On obtaining further history of present illness, the patient informed us about forcefully deposited local anaesthetic on the palatal side during treatment. Based on the examination and history of the patient provisional diagnosis was made as aseptic palatal necrosis. Accidental injection of formaldehyde and thermal burn were kept as differential diagnosis.

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On the first visit, the patient was advised to get orthopantomogram (OPG) and blood investigation done. OPG revealed no bony changes, and blood tests were also within the normal limits. We assured the patient about the lesion and started antioxidants along with multivitamin, gave betadine plus hydrogen peroxide gargle and recalled after 5 days.

The following visit, the biomechanical preparation of 24 and 26 was completed and an intracanal medicament was applied. Enucleating of necrotic palatal mucosa followed by irrigation and haemostasis was achieved. Finally, an occlusion splint was placed. Postoperative instructions were given and the patient was recalled after 1 week [Figure 2]. The patient was kept on regular follow-up and root canal treatment was completed after 1 month with fixed partial denture over 24, 25, and 26. Palatal mucosa granulated slowly and covered approximately 80% of the palatal bone in 3 months [Figure 3] and completely after 6 months [Figure 4].

The protocol followed for the management of this case is Vit Pro once a day for 3 months, Neomust Forte twice a day for 3 months, betadine + hydrogen peroxide mouthwash four times a day for 1 month followed by 0.2% chlorhexidine for 3 months and soft and nonspicy healthy diet for 3 months.

**DISCUSSION**

There are so many complications caused by local anaesthesia described in the literature. Postanaesthetic necrosis caused by the pressurized injection of local anaesthetic solution, particularly those containing vasoconstrictor, is one of the complications and is well documented. The palate has 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. According to Vidisha et al., only 6–7 cases were reported with aseptic palatal necrosis till 2017.

Palatal mucosa is relatively dense, confined, unyielding, and firmly adherent to the underlying palatal bone. The pain and soreness in the palatal mucosa may be caused by forceful
administration of local anaesthetic agents. The etiology of such an event is the absence of good blood supply through vasoconstrictions which deprive the tissue of its necessary sustenance, resulting in the necrosis of the overlying epithelium. The vasoconstrictor causes contraction of smooth muscles present in the arterial wall which may lead to transient ischaemia and reduces the oxygen supply to the distal structures of the injected site and promotes the production of acidic by-products of metabolism. In addition, to preserve the vasoconstrictors, the pH of the local anaesthetic solution is adjusted toward acidity, which, however, accentuates tissue acidity. Thus, epinephrine contained in many local anaesthetic agents may be a possible cause of ischaemia and secondary necrosis.\(^6\)

Modern local anaesthetic agents are relatively nonirritating to the tissues with the exception, perhaps, of skeletal muscle. Allergic reactions may manifest as circumscribed lesions while others may be derived from the pharmacological effects of the agents used. Allergic reactions to local anaesthetics have been greatly reduced by the development of amide local anaesthetics, for example, lidocaine. Cases of skin necrosis due to prilocaine have been reported.\(^7\)

Epithelial desquamation may result from the application of a topical anaesthetic agent to gingival tissues for a prolonged period. In the hard palate, a sterile abscess may form secondary to prolonged ischaemia resulting from the use of a local anaesthetic solution containing a vasoconstrictor. Other postanaesthetic intraoral lesions may result from recurrent aphthous stomatitis and/or herpes simplex which can develop following any traumatic insult to the tissues.\(^8\) 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 the 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.\(^9\)

Management of such lesions formed due to administration of local anaesthetic is usually conservative. It consists of reassuring the patient, prescribing analgesics, and a combination of topical antiseptic and anaesthetic preparations. Healing generally occurs within 8–10 days after the onset of the lesion. Rarely, surgical intervention is necessary when an ulcer does not heal. An oral protective emollient Orabase Paste can also be prescribed. The following measures can minimize such palatal lesions using topical anaesthetic preparation according to the manufacturer’s specifications. Moreover, it should be applied for 1–2 min to maximize the effectiveness and minimize toxicity.\(^10\)

Here, we presented a case of postanaesthetic aseptic palatal necrosis, in which palatal mucosa got granulated slowly and covered the palatal bone approximately 80% in 2–3 months and completely after 6 months.

**Conclusion**

Here, we conclude precautions should be taken to prevent postoperative complications. Complications can occur anywhere, but precautions can reduce the chances of postoperative discomfort.

**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.

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**Conflicts of interest**

There are no conflicts of interest.

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