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Iatrogenic penetration of the mouth floor during mandibular molar extraction: A case of protracted bleeding in an emergency department and clinico-anatomical considerations

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SUMMARY

Iatrogenic perforation of the lingual cortical plate of the mandible is uncommon but it may result in injuries to various adjacent structures such as the lingual nerve, sublingual gland, submandibular duct, deep lobe of the submandibular gland, and branches of rich anastomosing plexus supplying the floor of the mouth (FOM). The aim of this article was to highlight an unusual case of protracted postoperative bleeding that was occurred due to a FOM injury during an attempt to extract a mandibular second molar. Various anatomical considerations with clinical relevance were summarized too. Even small and superficial injuries of FOM should be not misjudged but be carefully monitored, since there is a potential risk for significant haemorrhage and hematoma formation leading to airway obstruction.

Key words: floor of mouth, extraction, sublingual hematoma, haemorrhage, mandibular molar.

INTRODUCTION

Iatrogenic injury of the floor of mouth (FOM) has potential to occur due to tooth extraction, implant placement, tori removal or reduction, biopsies, genioplasty, and various osteotomies (1). If an arterial branch of the high vascular of FOM is also injured, the aforementioned complication may even lead to life-threating conditions, such as hypovolemic shock and airway obstruction caused by a massive hematoma (2). The aim of this article was to report the management of a case of copious postoperative bleeding that arose due to a FOM injury during the extraction of mandibular second molar.

CASE REPORT

A 32-year-old man was referred to surgical emergency department (ED) of Evaggelismos Hospital of Athens by the hospital dentist at 04:23 am due to uncontrolled intraoral bleeding occurred after a second molar extraction. The patient cited that the extraction was performed by another (private) dental practitioner; it was very laborious while the whole interventionlasted at least 2 hours.The bleeding was continuous as it had initiated approximately at 10:30 pm of previous day and it had never been completely controlledbefore his visit. His medical history was not remarkable for diagnosed disease, medication use, smoking, or alcohol abuse.

On examination, the patient was eupneic without signs of respiratory distress; his blood pressure was 98/62 mm Hg, while the heart rate was 84 bpm. A wound of the left half of FOM exhibited copious haemorrhage; it was approximately 5-10 mm in both width and depth, as well as in continuity with the haemorrhagic post-extraction socket (Figure 1, A). The flowing blood was bright in color. Several sutures and hemostatic gauzes had been already placed before the referral to ED, but the patient mentioned that even temporary cessation of bleeding had not been achieved.

Under local anesthesia with articaine 4% and epinephrine 1:200000, both the wound and socket were first cleansed by a solution contained a mixture of hemostatic agents for local vasoconstriction. The

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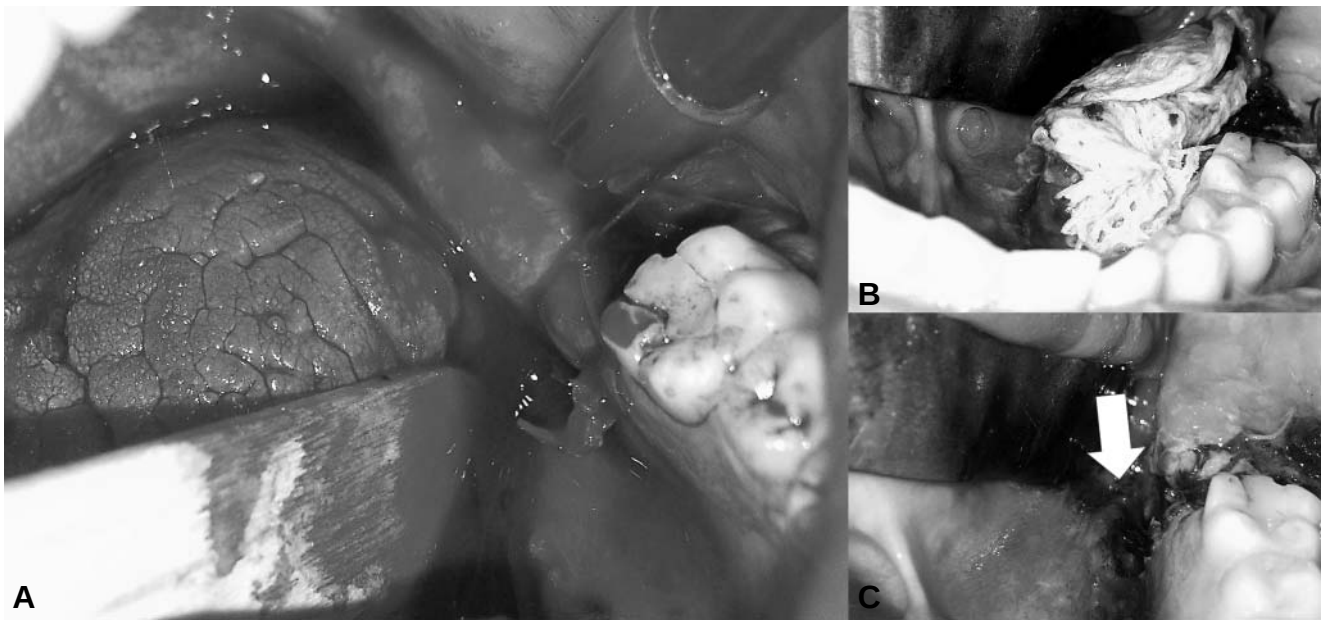

Fig. 1. A – a bleeding wound of the left half of FOM. Sutures and gauzes had been already placed before the referral and the blood was bright in color. B – a Xeroform gauze was placed to restrain the oxidized cellulose within the wound. C – the wound after the removal of the gauze and hemostatic materials. The arrowhead indicates the perforating injury of the FOM.

hemostatic agents included: 5-10 ml of tranexamic acid (500 mg/5 ml), 4-6 ml of epinephrine (1 mg/ml), 60 ml of 3% hydrogen peroxide, and 120 ml of normal saline 0.9%. Afterwards, several particles of an oxidized cellulose gauze gradually filled the bone cavity and covered the wound of FOM too.

Slow oozing continued, thus a sterile and fine mesh gauze, impregnated with a blend of 3% bismuth tribromophenate (Xeroform) and USP Petrolatum (jelly), was properly configured to form a "saddle" or "stopper" over the wound similarly to tie-over technique for split-thickness skin grafts. The gauze was sutured to restrain the hemostatic material within the wound, to exert stable compression, and to protect the clot from salivary enzymes. It was also placed to absorb quantities of hemostatic solution that could be released during mastication and tongue movements (because of the exerted pressure over it). Sutures (3-0 and 4-0 vicryl) were passed superficially through the adjacent tissues of mouth floor as well as over and through the gauze, to keep it fixed in place (Figure 1B). Diathermy was not initially applied for the risk of lingual nerve injury and was not eventually needed. Effective surgical hemostasis was accomplished within 2 minutes.

The patient felt faintness that lasted for a few minutes and resolved after placement in Trendelenburg position. He was admitted to hospital's maxillofacial clinic for monitoring and received antibiotic (IV amoxicillin/clavulanic acid 1.2gt.i.d. for 4 days), corticosteroid (IV methylprednisolone 1st day: 125 mg t.i.d., 2nd day: 125 mg b.i.d, and 3rd day: q.d.),and analgesic (IV paracetamol 1gr t.i.d.), and supportive fluid (normal saline 0.9% sodium chloride) therapy. A 100-ml oral solution containing the aforementioned mixture of hemostatics (placed in urine-sample container) was instructed for wound rinsing (5 ml of it with a blunt-tipped syringe) 4 times per day, after every meal and in case of recurrent bleeding. The removal of the Xeroform gauze was performed 32 hours later (Figure 1, C).

After bleeding cessation, the patient remained hemodynamic stable despite the hemoglobin drop of 1.6 mg/dl (hematocrit 41.6%). The coagulation panel did not reveal any coagulopathy. He did not confirm tongue hypoesthesia, while a panoramic radiograph (Figure 2) evidenced a residual distal root within the socket and widespread mandibular defect extended below the level mylohyoid line (absence of lingual cortical plate). There was no recurrence of bleeding during patient's hospital stay and until the complete healing of postextraction socket. He discharged 2 days later and the residual root was extracted 29 days later without complications.

DISCUSSION

Iatrogenic perforation of the lingual cortical plate may result in exposure and injury of various structures such as the lingual nerve,sublingual gland,submandibular duct,deep lobe of the submandibular gland, and branches of rich arterial network supplying the mouth floor (3).The main arteries forming this network are the sublingual and submen-

tal as well as mylohyoid and lingual branches of inferior alveolar artery (Figure 3, A and B) (4-8). The sublingual artery originates from the lingual artery at the anterior margin of the hyoglosseus muscle, and then courses between the genioglossus and mylohyoid muscles contributing on vascularization of the sublingual salivary gland, mylohyoid and adjacent muscles, mucous membranes of FOM, and lingual gingivae of the mandible.The submental artery branches off from facial artery, continues anteriorly along the inferior surface of the mylohyoid muscle, and provides blood supply to the submandibular triangle, the anterior belly of the digastric muscle, and the mylohyoid muscle. It also gives off a sizeable branch perforating the mylohyoid muscle. The mylohyoid artery arises from the inferior alveolar artery, descends within the mylohyoid groove, and ramifies on the periosteum of lower medial cortex and the undersurface of mylohyoid muscle. The lingual branch leaves the inferior alveolar artery near its entrance in the mandibular foramen, and continues inferiorly along the lingual nerve to supply the mucosa of the FOM.

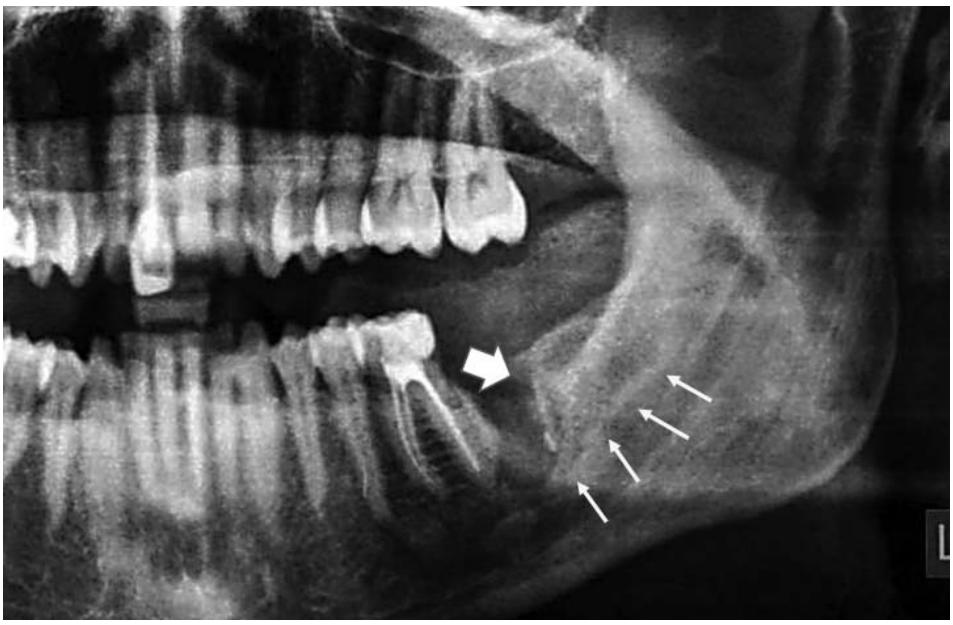

Fig. 2. The panoramic radiograph evidenced a residual distal root within the socket (arrowhead) and a defect of lingual cortex extended below the level of radiopaque mylohyoid line (arrows).

Of note, profuse hemorrhage can be provoked not only by severance or laceration of a vessel of the FOM, but also by trauma of local musculature (1). Indeed, the mylohyoid muscle is anatomically perceived as an anastomosing structure between sublingual (medial muscular aspect) and submental arteries (lateral muscular aspect) (9).

A completely transected vessel of FOM may not immediately bleed as a result of the retraction deep into local soft tissues and/or effects of used vasoconstrictor (rebound dilation) (3). The primary concern should always be the maintenance of the airway because there is risk for development of large dissecting hematoma (10, 11); the diagnosis of hypovolemic shock must be considered too (2, 12). Several case reports have described airway obstruction/compromise secondary to perforation of mandibular lingual cortex, mostly in anterior region of mandible, during implant placement procedures (9, 10). In the majority of these cases, either emergency intubation or tracheostomy was carried out (10). Profound swelling and elevation of FOM against palatal vault may be established either immediately or after the interval of few hours (9). Therefore, Vehmeijer *et al.* (13) suggested the use of awake fiberoptic intubation for acutelydeveloped hematomasof FOM to reduce the likelihood of a fatal ‘‘can’t intubate, can’t ventilate” situation.

Both the increase and extension of a hematoma are influenced bythe responsible vessel (capillary, venous, or arterial) and the surrounding tissues (muscle, fat, or interstitia)(12). The increase ofhematoma may be interrupted, as long as the pressure of the extravasated blood collections surpasses the vascular pressure of the haemorrhagic vessel (12).

The hemorrhage of the presented case was likely to be causedby iatrogenic perforation of the mandibular originof mylohyoid muscle and subsequent injury of its arterial tributary. Flanagan advocated that an injured vessel at the lingual aspect of the posterior mandible is more likely to be the mylohyoid artery (14). On the other hand, Kim *et al.* (15) underscored the risk of bleeding in second molar region, since their study found that the submental artery lies at its highest position at this site, with close horizontal distance (1.9 mm)from lingual cortical plate and increased diameter (1.3 mm) as well.According to medical information that patient gave, the mechanism of injury was probably attributed to inadvertent use of a dental elevator. After the fracture and perforation of lingual wall the alveolus, the instrument was gradually sliding and penetrating the soft tissues of FOMduring the maneuvers

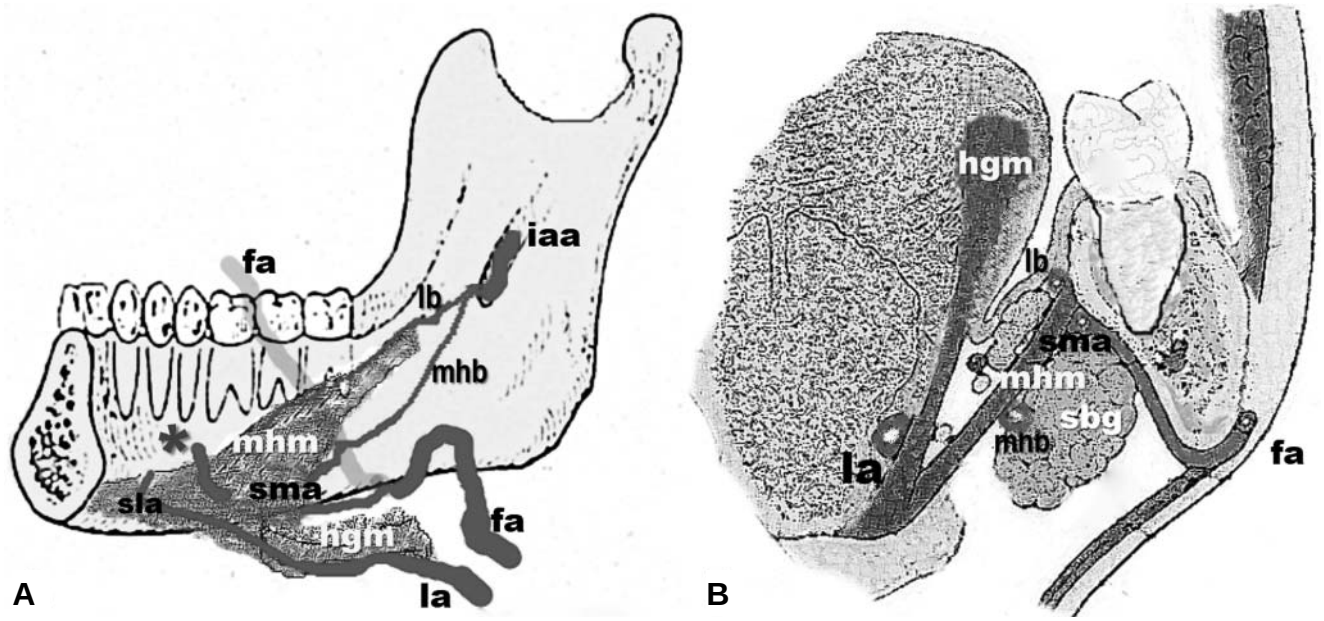

Fig. 3. Medial (A) and coronal (B) view of FOM. fa – facial artery; iaa – inferior alveolar artery; mhb – mylohyoid branch of inferior alveolar artery; lb – lingual branch of inferior alveolar artery; la – lingual artery; sma – submental artery; sbg – submandibular gland; mhm – mylohyoid muscle; hgm – hyoglosseus muscle (cutted in Fig 3A); *asterisk – perforating branch of submental arterty.

of strenuous extraction. Airway obstruction from posterior displacement of the tongue did not occur, since the extravasated blood drained into oral cavity through the extraction socket, without forming a life-threatening hematoma within lax soft tissues.

Such a hemorrhage may be initially controlled by exerting bimanual (external) pressure to the floor of mouth and lingual surface of the mandible (1, 3, 14). Pressure on facial artery can be applied on antegonial notch too, in case where the perforator of submental artery is recognized to be the injured vessel (1, 9). Any attempt for intraoral surgical ligation of the bleeding source may be proved arduous or impossible, as there are distortion of local anatomy and retraction of the vessel deep in tissues (3). However, in the event that hemorrhage is not cessated with all the aforementioned measures, surgical ligation via an extraoral (submandibular) approach can be attempted (1, 3, 9, 11). The ligation of the submental orfacial artery should be first performed, andif the bleeding continues, ligation of lingual artery should follow (14). In case of severe haemorrhage or vascular malformation, carotid angiography with selective endovascular embolization of the bleeding source should be a treatment option too (16).

The described hemostatic technique combines packing procedures with the use of hemostatic agents. It is routinely applied by the first author forcontinuous and recurrent episodes of intraoral hemorrhages in ED, and does not premise neither specific equipment nor special and expensive materials. It can be performed bedside and, potentially, not only by dentists and oromaxillofacial surgeons but also by those emergency physicians who are accustomed to wound packing and tie-over bandage techniques (of mucosal and bony cavities) in head and neck region.

CONCLUSIONS

This case underscored that both dental practitioners and oral surgeons should be pay attention to not harm adjacent anatomic structures even during minor and routine dentoalveolar surgeries. Profound knowledge of local anatomy and its variations is essential and should be also supplemented preoperatively by thorough radiographic evaluation. The rare complication of FOM injury in this case occurred in an otherwise healthy young man; a similar event of acute blood loss in a patient with history of ischemic heart disease could have caused life-threatening hemodynamic instability. Furthermore, considering that the interruption of either anticoagulant or antiplatelet medications is not currently recommended for minor dental surgeries, the avoidance of vascular surgical trauma grows in importance. Even small and superficial iatrogenic injuries of FOM should be not underestimated as there is risk for hematoma formation and airway obstruction, which can be apparent immediately or within few hours. In these circumstances, close monitoring for at least 48 hours is advisable.

There is no conflict among the authors

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