

Emergency Treatment of Massive Hemorrhage after Upper Third Molar Extraction : A Case Report

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Tooth extraction is a routine dental treatment, and massive hemorrhage rarely occurs in it. In the present case, emergency treatment was performed to stop massive hemorrhage after upper third molar extraction. The patient was a 50-year-old female with hypertension who underwent an upper third molar extraction at a dental clinic. Since massive hemorrhage continued after tooth extraction, the patient was taken to our hospital. General anesthesia was selected because hemostasis under local anesthesia was difficult, hematomas were present in the buccal mucosa and soft palate, and a respiratory risk was predicted. Awake nasotracheal fiberoptic intubation was selected because mask ventilation was difficult. Since the greater palatine artery was not injured, it was assumed that the hemorrhagic point was present in a branch of the pterygoid plexus. The tracheal tube was left for respiratory management after treatment, and removed on postoperative day 2 after complete hemostasis was confirmed. It is important to evaluate the necessity of general anesthesia for hemostatic treatment in consideration of the cause and volume of massive hemorrhage.

Key words : hemorrhage, pterygoid plexus, emergency treatment

Introduction

Tooth extraction is a routine dental treatment. Hemorrhage after tooth extraction is a frequent complication, but massive hemorrhage rarely occurs. The cause of massive hemorrhage after tooth extraction is roughly divided into systemic causes, such as hemophilia^{1,2)} and anti-thrombotic therapy³⁾, and local causes due to injury of the surrounding tissue^{4,5)}. In this patient, we performed emergency treatment for massive hemorrhage after the upper third molar extraction.

Case description

The patient was a 50-year-old female, 153 cm in height and 52 kg in weight, with hypertension. She underwent the upper right third molar extraction at a dental clinic, and massive hemorrhage occurred after extraction. The maxillary tuberosity was partially attached to the extracted tooth (Fig. 1). The dentist attempted hemostasis, but hemorrhage continued. The patient was urgently taken to OHU University Hospital. On arrival, the blood pressure was 171/113 mmHg ; pulse, 85 beats/min ; respiratory rate, 20 breaths/min; and percutaneous ar-

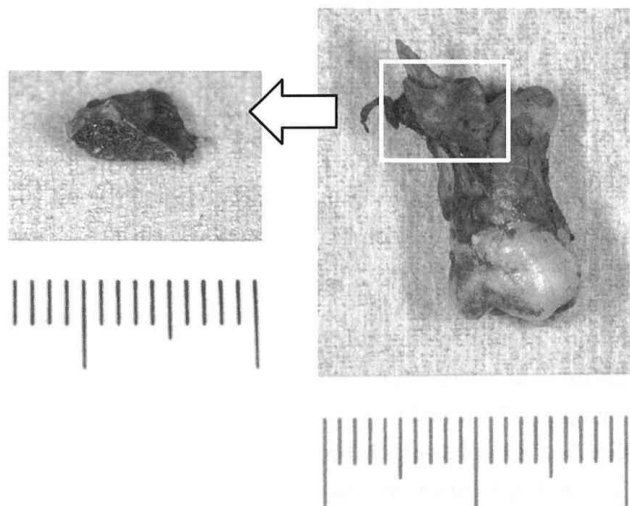


Fig. 1 Extracted tooth with a part of maxillary tuberosity

terial blood oxygen saturation, 98%. On blood testing, the red blood cell count ($334 \times 10^4 / \mu\text{l}$), hemoglobin level (10.8 g/dl), and hematocrit (31.5%) were decreased, and the platelet count ($16.3 \times 10^4 / \mu\text{l}$), thrombin test value (11.5 seconds), prothrombin time international normalized ratio (1.05), prothrombin time percentage activity (94.2%), and activated partial thromboplastin time (24.0 seconds) were normal. Hemorrhage could not be stopped by hemostatic treatment under local anesthesia. During this period, hematomas appeared and rapidly expanded in the buccal mucosa and soft palate. An oral surgeon of our hospital considered that inhibition of hemorrhage under local anesthesia was impossible, and selected hemostatic treatment under general anesthesia to avoid airway obstruction by the expanding hematoma.

After standard monitoring, general anesthesia was introduced by the intravenous administration of midazolam (1mg), propofol ($0.5 \mu\text{g}/\text{ml}$ by target controlled infusion), and remifentanyl ($0.5 \mu\text{g}/\text{kg}/\text{min}$) under oxygen inhalation (6 l/min) (Fig. 2). Two minutes later, the dose of continuous remifentanyl infusion was decreased to

$0.1 \mu\text{g}/\text{kg}/\text{min}$. Since an oral surgeon continued pressure hemostasis of the region around the upper third molar with his finger and gauze, bag ventilation and oral tracheal intubation using a laryngoscope were difficult. Thus, nasotracheal fiberoptic intubation was performed. General anesthesia was maintained with propofol ($1.0 \mu\text{g}/\text{ml}$ by target controlled infusion) and remifentanyl ($0.15 \mu\text{g}/\text{kg}/\text{min}$) under oxygen (1 l/min) and air (3 l/min) inhalation. Temporary hemostasis was achieved, but hemorrhage recurred when the blood pressure rose in the awakening process, for which general anesthesia was resumed. The greater palatine artery was not injured, and hemorrhage on the distal side of the extraction site was confirmed. After pressure hemostasis, the extraction socket was covered with a hemostatic agent, and the wound was sutured. The tracheal tube was not removed because of the risk of airway obstruction by the hematoma. After hemostatic treatment, a single dose of propofol (30 + 20mg) and continuous dexmedetomidine ($0.7 \mu\text{g}/\text{Kg}/\text{h}$) were administered for sedation, and a single dose of fentanyl was administered (50 + 20 + 30 μg) for postoperative

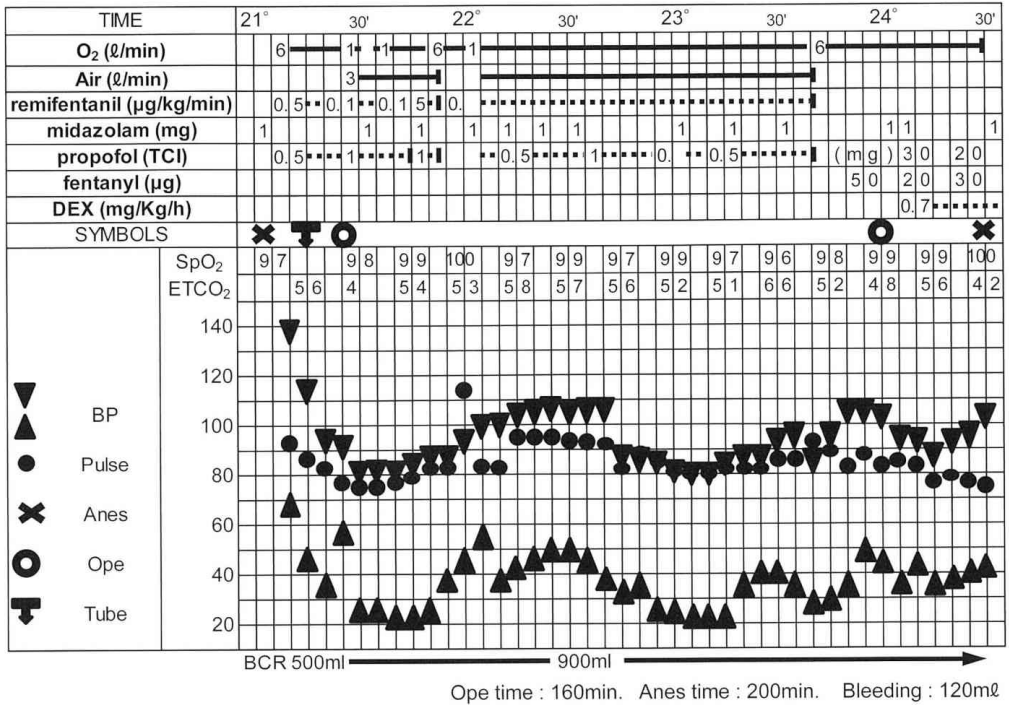


Fig. 2 Anesthesia record

analgesic treatment. The patient was transferred to an ICU. The tracheal tube was removed after confirming complete hemostasis on postoperative day 2.

Discussion

The most important focus of this case was to identify the cause of the massive hemorrhage. The systemic causes of massive hemorrhage include a hemorrhagic predisposition^{1,2)} and anti-thrombotic therapy³⁾, and the local causes include hemorrhage from the extraction socket and injury of the surrounding tissue⁴⁻⁶⁾. The blood coagulation system of this patient was normal, and the patient had received no anti-thrombotic therapy. Accordingly, systemic causes were excluded. Regarding the local cause, hemorrhage was observed on the distal side of the extraction site. It was considered that the cause of the bleeding is an injury of the

greater palatine artery, the descending palatine artery, the posterior superior alveolar artery, or the pterygoid plexus⁴⁾. Subsequently, it was assumed that the hemorrhagic point was present in the pterygoid plexus because the bleeding was not a pulsatile. There have been several case reports of complications associated with the pterygoid plexus after upper third molar extraction^{5,6)}. Shah and Bridgman⁵⁾ reported fracture of the maxillary tuberosity and pulsating massive hemorrhage after extraction of the upper right second molar. Their patient required general anesthesia for the control of massive hemorrhage and removal of fractured bone fragments, similarly to our patient. Warburton and Brahim⁶⁾ reported infraorbital hematoma that appeared 2 days after extraction of the upper bilateral third molars, and discussed communication of the pterygoid plexus with the infraorbital structure in the infratemporal fossa. It is

important to consider complications of upper third molar extraction associated with the pterygoid plexus and confirm the anatomical morphology of the maxillary tuberosity. The appropriate operation of tooth extraction tools is also necessary.

The blood loss was estimated from the body weight (Wt) (kg) and hemoglobin level (Hb) (g/dℓ) of the patient using the equation below :

$$\text{Bleeding} = \frac{\text{Wt} \times 0.7}{\text{pre-bleeding Hb}} \times (\text{pre-bleeding Hb} - \text{post-bleeding Hb})$$

On the assumption that the hemoglobin level before tooth extraction was 14 g/dℓ, the blood loss was calculated as follows:

$$\begin{aligned} \text{Bleeding} &= \frac{52 \times 0.7}{14} \times (14 - 10.8) \\ &= 8.32 \text{ dl (832 ml)} \end{aligned}$$

This is useful to judge the necessary volume of blood transfusion, although it is an estimate. The indication of blood transfusion is more than a 30% loss of the circulating blood⁷⁾ or a 6 g/dℓ or lower hemoglobin level⁹⁾. The circulating blood of this patient was estimated to be 36.4 dℓ (52 kg x 0.7 dℓ). If the blood loss was 8.32 dℓ, 22.9% of the circulating blood was lost. The hemoglobin level of the patient was 10.8 g/dℓ. Accordingly, blood transfusion was unnecessary for this patient. However, the blood loss was greater than 20% of the circulating blood, and so the infusion of colloid solution was necessary.

Awake nasotracheal fiberoptic intubation was selected because hemorrhage continued, tracheal intubation under laryngoscopic direct vision was difficult, and there was a risk of causing hemorrhage from the soft palatal hematoma by laryngoscope operation. Midazolam and remifentanil were intravenously administered because awake intubation without sedation is intolerable for patients. Several studies suggested that midazolam and remifentanil reduced pain during awake nasotracheal fiberoptic

intubation^{9,10)}.

In conclusion, it is important to judge whether general anesthesia is necessary for hemostatic treatment in consideration of the cause and volume of massive hemorrhage.

The authors declare no conflict of interest associated with this manuscript.

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