

CASE REPORT

# Post-operative avascular necrosis of the maxilla: a rare complication following orthognathic surgery

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## Abstract

We present a patient with sickle cell trait who suffered avascular necrosis of the maxilla as a complication of maxillary osteotomy. Understanding the blood supply of the maxilla and how possible patient related, anaesthetic and operative factors affect it, is important in understanding how the vascularity of the maxilla can become compromised in a surgical procedure. The perioperative parameters were analysed to identify any prognostic elements. Avascular necrosis of the maxilla is a rare complication of orthognathic surgery with few cases reported in the literature. There are identifiable risk factors that can influence the blood supply of the maxilla. Careful preoperative assessment is required to exclude patient factors that have the potential to affect tissue vascularity. This in conjunction with sound anaesthetic and surgical technique should all minimize the risk of avascular necrosis. Even so it is still possible for this rare complication to occur.

## INTRODUCTION

Orthognathic maxillary surgery is a safe, predictable and stable procedure. The incidence of perioperative complications is low and the number of life-threatening complications with this surgery is even lower. However, avascular necrosis of the maxilla after Le Fort I osteotomy is a rare complication that has been reported to occur in <1% of cases [1].

During down fracture of the maxilla, the blood supply is by the ascending pharyngeal artery, the ascending palatine branch of the facial artery and the rich mucosal alveolar network overlying the maxilla [2]. Table 1 outlines the factors that may result in impaired blood supply to the maxillary segments and increase the risk of ischaemia. When the descending palatine artery is sacrificed, the main blood supply of the maxilla will

now be the soft tissue pedicle, which incorporates the anterior faucial pillar and the palatal mucosa (see Fig. 1).

## CASE REPORT

We report a case of a 45-year-old Afro-Caribbean female who underwent orthognathic surgery to correct her malocclusion. She was previously investigated for a fibro-osseous lesion, which presented as a hyperplastic right maxillary tuberosity (see Fig. 2). This was biopsied and reported as normal lamellar bone. Relevant past medical history included hypertension and sickle cell trait. Her medications included Lisinopril and Indapamide.

As seen in Fig. 3, there was good oral hygiene. The patient had lost her lower posterior teeth and there was downward

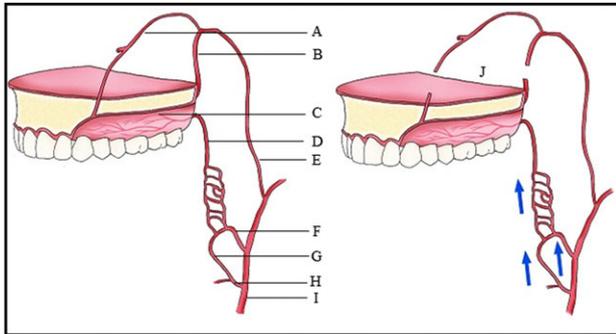
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**Table 1** Factors increasing the risk of avascular necrosis of the maxilla

Local	Systemic
Radiation treatment	Cigarette smoking
Infection	Pregnancy
Trauma	Chemotherapy
Surgery related	Haematological conditions
Sacrifice of descending palatine artery	Sickle cell disease
Perforation/stripping palatal mucosa	Leukaemia
Adrenaline injected into mucosa	Gaucher's disease
Perioperative vascular thrombosis	Thalassaemia
Segmental osteotomies	Caisson disease
Extensive advancement	Systemic lupus erythematosus
Anatomy related	Diabetes mellitus
Craniofacial dysplasias	Vasculitis
Orofacial clefts	Inflammatory bowel disease
Vascular anomalies	Drugs
Previous surgery	Vasoconstrictors
Cleft palate repair	High dose steroid
Surgically assisted rapid palatal expansion	



**Figure 1:** Blood supply of the maxilla. (A) Nasopalatine artery, (B) Descending palatine artery, (C) Greater palatine artery, (D) Lesser palatine artery, (E) Maxillary artery, (F) Ascending pharyngeal artery, (G) Ascending palatine artery, (H) Facial artery, (I) External carotid artery, (J) Le Fort I downfracture.



**Figure 2:** Preoperative orthopantomograph showing expansion of the right maxillary tuberosity.

displacement of the upper right posterior segment into the edentulous area of the lower posterior jaw.

The operation planned was a Le Fort I osteotomy with a right posterior segment osteotomy, which would set back the upper incisors by 6 mm, impact the upper left second molar by 4 mm and impact the posterior segment from the upper right first premolar (by 5 mm) to the upper right first molar (by 12-mm). No forward movement of the maxilla was planned. The



**Figure 3:** Pre-surgical photographs.

segmental osteotomy was performed following down fracture of the maxilla and the final occlusion was aligned to a full coverage occlusal splint. The right descending palatine artery was sacrificed.

The maxillary mucosa appeared pale during the procedure, but showed capillary refill. The patient presented with wound infection and avascularity of the maxilla 1 week after the procedure. She was treated with intravenous Co-amoxiclav for 2 weeks. A course of hyperbaric oxygen treatment was organized at 14 and 18 days post-operatively. She was started on therapeutic doses of Pentoxifylline and Vitamin E to improve capillary blood flow.

As shown in Fig. 4, most of the maxilla was affected, the right side to a greater degree. Despite treatment, over time those areas which remained devascularised began to demarcate. An oro-antral fistula developed in the upper right quadrant, as seen in Fig. 5.

## DISCUSSION

Blood flow to the maxilla is reduced by 50% in the first post-operative day after sacrifice of the descending palatine arteries [3]. However, there is excellent collateral blood supply particularly if only one artery is sacrificed, as in this case. Experimental studies have shown that loss of the descending palatine arteries results in a transient ischaemic period that is compensated



Figure 4: Photographs at 26 days post surgery.



Figure 5: Photographs at 6 months post surgery.

for by a vascular proliferation that allows tissue healing. The collateral microvasculature from other vessels including the ascending pharyngeal and facial arteries (Fig. 1), maintains the viability of the palatine pedicle. The pedicle can withstand stretching greater than 10 mm of anterior repositioning of the maxilla.

The patient had sickle cell trait. The haemoglobinopathy screen confirmed Hb A (Adult Haemoglobin) and Hb S (Sickle Haemoglobin). The Hb F (Foetal Haemoglobin) level was <0.5% and the Hb S level was 39.4%. These parameters are typical for sickle cell trait. However, for patients in Sickle Cell crisis or those expected to be exposed to severe hypoxia, Hb S level <30% is advised [4].

Craniofacial dysplasias may represent areas of anatomical variation where the blood supply may be susceptible to disruption. Although the patient had a biopsy showing normal bone, the maxillary tuberosity was expanded and hyperplastic clinically. This in itself would not however have accounted for the degree of avascularity seen in this case.

Literature suggests that segmental osteotomy is at a higher risk of ischaemic complications [5, 6]. Necrosis of the maxilla can be minimized in the following ways:

- Divide into as few segments as possible and avoid small segments anteriorly.
- Maintain the integrity of the palatal mucosa.
- Perform sagittal segmentation in paramedian sites as the mucosa is thicker and the bone thinner than the midline.

Although hypotensive anaesthesia was not purposely utilized for the procedure, it is a technique commonly used to help maintain a bloodless surgical field [7]. A mean arterial pressure (MAP) 30% below a patient's usual MAP, with a minimum MAP of 50 mm Hg in American society of

anaesthesiologists Class I patients and a MAP not <80 mm Hg in the elderly, is suggested to be clinically acceptable [8]. It is recommended that with respect to hypotensive anaesthesia:

- It should be adjusted in relation to the patient's preoperative blood pressure rather than to a specific target pressure.
- It should be limited to that level necessary to reduce bleeding in the surgical field.
- It should be confined to that part of the surgical procedure deemed to benefit by it.
- There is little need for blood transfusion perioperatively [9].

The anaesthetic concerns include the management of perioperative hypoxia, acidosis, hypovolaemia and hypothermia. Points to consider with this case are:

- (1) Hypoxia never occurred and is usually avoided as anaesthetised patients are always maintained with an  $\text{FiO}_2 > 0.35$ .
- (2) Acidosis may trigger a vaso-occlusive crisis and would affect all micro-circulation; it is easily prevented by proper perfusion and maintaining circulating blood volume.
- (3) Hypothermia was prevented by patient warming.
- (4) Hypovolaemia could be contentious, especially with hypotension.

With this patient, the mechanism of avascular necrosis could not clearly be explained. In the analysis of patient, surgical and anaesthetic factors, there seemed to be issues that may have implied a greater risk.

## CONFLICT OF INTEREST STATEMENT

None declared.

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