



# Surgically treated congenital cleft palate in a 4-month-old kitten: medium-term clinical and CT assessment

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# **Abstract**

Case summary A 4-month-old female domestic shorthair kitten was presented for a congenital cleft palate causing nasal discharge and sneezing episodes. CT revealed a palatal bone defect involving 20% of the palatal area. Surgical correction of both the hard and soft palate defects was performed using the overlapping and medially positioned flap techniques, respectively. Complete healing of the wound and full resolution of the clinical signs occurred within a 1-month period. At 2 months postoperatively, two punctiform oronasal fistulae were observed rostrally without associated clinical signs. Control CT, performed 6 months postoperatively, revealed a 50% enlargement of the palatal bone defect. At 12 months postoperatively, the cat was still in good general condition without any clinical signs.

Relevance and novel information To the best of our knowledge, this is the first report to describe the treatment of a congenital cleft palate in a kitten using the overlapping flap technique with a successful medium-term clinical outcome, despite the formation of two oronasal fistulae. This suggests that, as in dogs, full restoration of oronasal compartmentation is not mandatory to achieve functional outcome. The increase of the palatal bone defect over time may play a role in late oronasal fistulae formation and should be considered for surgical planning.

**Keywords:** Oral and maxillofacial surgery; wound healing; congenital cleft palate; overlapping flap technique; palatal bone defect

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## Introduction

Congenital cleft palates (CCPs) are not commonly described in cats; among the seven cases reported, only three were treated surgically. Two of them only had isolated soft palate defects, successfully managed in one case by bilateral overlapping mucosal single-pedicle flaps, whereas in the second case, the cat died at the time of recovery from anaesthesia. The third had a soft palate cleft with only a partial involvement of the hard palate, which was treated, after three failed surgical attempts, using an auricular cartilage grafting. Herein, we report for the first time the successful surgical treatment of a CCP in a kitten, using a combination of the recommended techniques in dogs, highlighting an increase in the palatal bone defect (PBD) over time.

# **Case description**

A 4-month-old female domestic shorthair kitten weighing 1.5 kg was presented for growth retardation, nasal discharge and sneezing episodes related to food intake since birth. Clinical findings revealed a body condition score (BCS) of 3/9, intermittent snoring but no abnormalities on pulmonary auscultation, and a cleft palate

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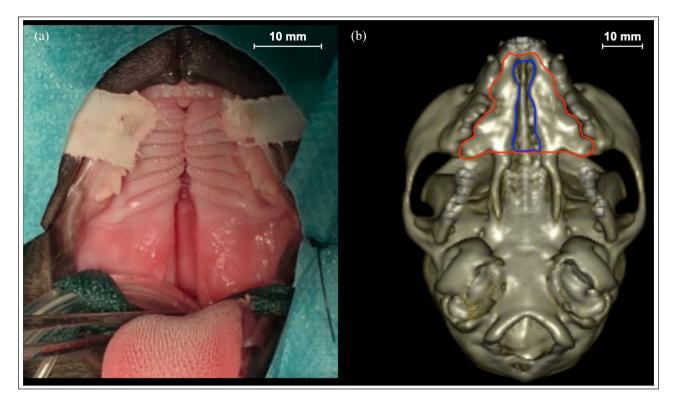
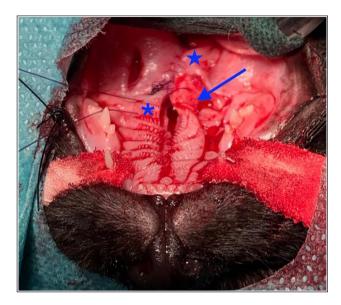


Figure 1 (a) Initial perioperative view of the cleft palate involving the soft and hard palates. (b) Three dimensional CT scan reconstruction: ventral view of the maxilla, highlighting the palatal bone defect (PBD), which was wider than the visible soft tissue defect. The red and blue lines surround the overall palatal area and the PBD area, respectively



Figure 2 Three dimensional CT reconstruction of the skull at initial evaluation (top row) and 6 months after surgery (bottom row): (a) rostral view; (b) ventral view; and (c) left lateral view. Note the skull symmetry and the absence of other visible congenital abnormalities apart from the cleft palate

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**Figure 3** Intraoperative view showing the overlapping flap (arrow) and the elevated contralateral flap to create a bed for the overlapping flap (asterisk). Note in the background the soft palate, reconstructed with medially positioned flaps (star)

involving the whole length of the secondary palate, with a relative width of soft tissue defect extending up to 15% of the maximal width of the palate (Figure 1a).

A CT scan of the skull (Canon Aquilion lightning SP 80-slice) revealed a large PBD, 2.6 times wider than the soft tissue defect, representing 20% of the total palatal surface area (Figure 1b). No other craniomaxillofacial congenital abnormalities were found and all relevant structures of the skull were normal (particularly dental occlusion and skull symmetry; Figure 2, top row).

Surgical treatment was provided. After premedication with methadone (0.2 mg/kg IM [Comfortan; Dechra]) and dexmedetomidine (7µg/kg IM [Dexdomitor; Zoetis]), anaesthesia was induced using alfaxalone (4 mg/kg IV [Alfaxan; Dechra]) and maintained with isoflurane (1–2.3%) in oxygen. Intravenous fluids were administered during surgery, and pain was managed using fentanyl (1µg/kg IV [Fentadon; Dechra]), as needed. Perioperative antibiotic therapy consisted of amoxicillin (20 mg/kg IV [Clamoxyl; Zoetis]). A mucoperiosteal incision was made along the right dental arch from the rostral to the caudal margins of the defect. An overlapping flap was created by mucoperiosteal elevation, taking care to preserve the major palatine arteries. On the left side of the defect, incisions were made 1-2 mm lateral to the cleft and along the dental arch; the bed flap was created by subsequent mucoperiosteal elevation on this side (Figure 3). The hard palate defect was closed by medial rotation of the overlapping flap, which was secured under the bed flap with full-thickness horizontal mattress sutures using glycomer 631 (Biosyn 4-0; Covidien). The



**Figure 4** Intraoperative view after completion of the overlapping flap. Particular attention is paid to preserve the major palatine artery (arrow)

medial margins of the soft cleft palate were incised and two partial thickness releasing incisions were made laterally. The soft cleft palate was closed by suturing the edges of the nasopharyngeal mucosa on one side and of the oropharyngeal mucosa on the other side, with two simple continuous patterns using 4-0 glycomer 631 (Biosyn; Covidien) (Figures 3 and 4). Postoperatively, buprenorphine (30µg/kg q8h for 1 day [Bupaq; Virbac]), meloxicam (0.05 mg/kg q24h for 5 days [Metacam; Boehringer Ingelheim]) and ampicillin–sulbactam (20 mg/kg q12h for 7 days [Unacim; Pfizer]) were prescribed along with a soft food regimen for 1 month. The cat recovered uneventfully and was discharged the day after surgery.

Granulation tissue was observed on the palatal bone 2 days later (Figure 5a). This granulation tissue covered all exposed palatal bone at 18 days (Figure 5b-d). At 1 and 2 months postoperatively, 70% and 100%, respectively, of the palatal bone surface exposed at the time of surgery was epithelialised (Figure 6a,b). However, two small oronasal fistulae (ONFs) were noted 2 months postoperatively in the mid-sagittal part of the hard palate, with no associated clinical signs (Figure 6b). Six months postoperatively, the ONFs size and BCS (4/9) improved vs assessment at the initial presentation (Figure 6c). Control CT revealed a 50% increase in PBD size, extending to 30% of the overall palatal area (Figure 7). However, harmonious growth of the skull was noticed (Figure 2, bottom row). At 9 (Figure 6d) and 12 months postoperatively, the two ONFs were consistently present, but the owners reported good quality of life without nasal discharge or sneezing episodes.



Figure 5 Short-term appearance of the surgical site. (a) Three days postoperatively: the granulation tissue (arrow) begins to cover the palatal bone (asterisk). Note the progression of the granulation tissue at (b) 7, (c) 10 and (d) 18 days postoperatively

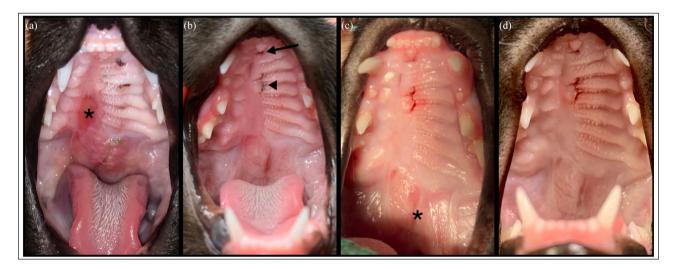


Figure 6 Medium-term appearance of the surgical site. (a) One month postoperatively: epithelialisation tissue covers 70% of the formerly exposed palatal bone surface (asterisk = granulation tissue). (b) Two months postoperatively: epithelialisation is complete. Note the resorption of suture knots and the presence of two oronasal fistulae (ONFs): a punctiform one in the rostral part of the hard palate (arrow) and a second in the middle part of the hard palate with hairs trapped inside (arrowhead). (c) At 6 months postoperatively, the two ONFs are still present. Note that the soft palate is completely healed (asterisk). (d) Nine months postoperatively no evolution of the ONFs is seen

# **Discussion**

Based on the literature search, this is the first report to describe the surgical management of a CCP involving both the hard and soft palates in a cat using a combination of the overlapping and sliding flaps techniques,<sup>6–9</sup> as well as the associated medium-term clinical and CT follow-up.

The overlapping flap technique used in the present report is recommended in dogs for the treatment of large congenital palatal defects.<sup>6-9</sup> However, the creation of mucoperiosteal flaps in young animals is challenging for several reasons: (1) the reduced surgical space, the small

amount of tissues available and their intrinsic friability; and (2), in an experimental study in dogs, flap creation has been suggested to hinder skull growth and result in maxillary deformity. In the present case, the surgical technique was performed in a 4-month-old kitten, despite the increased risk of failure, at the owners' request due to the time-consuming nursing and poor quality of life of the kitten. Although the overall bone growth of the skull was not affected by surgery, relevant conclusions cannot be drawn based on this single case. However, there is no consensus, in animals and in children, on the optimal timing to perform primary

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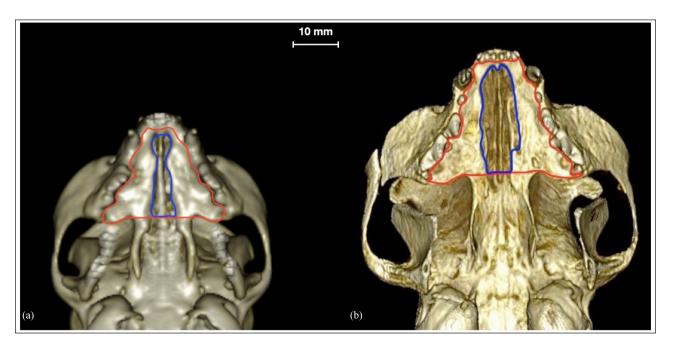


Figure 7 Three dimensional CT reconstruction. Ventral view of the maxilla, at (a) initial evaluation and (b) 6 months postoperatively, emphasising the augmentation of the palatal bone defect (PBD). The red and blue lines surround the total palatal area and the PBD area, respectively

repair of CCPs.<sup>3,8,10–16</sup> In dogs, some authors recommend performing the surgery at around 8 months of age.<sup>8</sup> However, performing a surgical repair either too early or too late (after 8 months) has been suggested to increase the risk of ONF formation.<sup>8,13</sup>

In this case, as previously reported in dogs, the relative PBD at admission was wider than the overlying soft tissue defect.<sup>14,17</sup> Indeed, PBD size is a critical issue to address, as large PBDs may increase the risk of ONF formation because the wound lacks bony support for healing and is more prone to trauma. 9,18-20 Moreover, the 50% enlargement of the PBD observed 6 months postoperatively has never been previously reported in animals or children, in which the PBD is rather reported to decrease due to bone bridge formation in about 70% of cleft palate repairs. 15 The reason for this increase remains unknown. Skull growth without bone bridge formation most probably accounted for that finding. Yet, the presence of an osteolysis secondary to preoperative osteomyelitis cannot be excluded, although CT findings were not consistent with this hypothesis. This increase must be taken into consideration for the surgical planning in order to define the technical choices, such as the use of specific flaps or prosthetic implants.9,18-20 However, recommendations are still lacking regarding the types of PBD on which these implants should be used.

Despite an uneventful recovery, two small ONFs, involving the rostral and middle portions of the hard palate, were noted 2 months postoperatively. In accordance with the literature in dogs, repair failure leading

to ONF formation is the most frequent complication, occurring in up to 50% of the cases in a study of 26 CCP repairs,8 being preferentially located at the hard and soft palates junction or at the rostral portion of the hard palate.8,21 The late onset of the ONF in our case contrasts with previous studies in which ONF formation has been recorded to occur between 3 days and 3 weeks after surgery.<sup>4,8,22</sup> ONF formation is supposed to be a result of excessive tension at the suture lines, infection, compromised blood supply, poor tissue quality, lack of underlying bony support, external trauma and poor body condition.<sup>8,9,14,16,21,23</sup> In this case, as the overlapping flap was sufficient to cover the defect without tension and so the blood supply was not altered, involvement of the surgical technique seems unlikely. However, several factors may be contemplated, including skull growth with concurrent PBD widening, poor initial body condition or the potential presence of preoperative rhinitis (although not clinically observed). Moreover, late self-inflicted trauma could not be excluded. Indeed, the lack of underlying bony support may enhance the risk of trauma-induced ONF.

# **Conclusions**

This is the first report to illustrate the feasibility of CCP repair in a kitten using the surgical technique recommended in dogs, with no noticeable impact on skull growth. In contrast to previous reports in dogs, ONF formation was observed at a later stage, after complete

epithelialisation of the palatal bone surface. This case also reported for the first time an enlargement of the PBD 6 months after its initial CT assessment, which may represent an additional risk factor to consider for postoperative ONF formation. Further studies on CCP repair in cats with long-term CT assessment are mandatory to draw definitive conclusions.

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**Ethical approval** The work described in this manuscript involved the use of non-experimental (owned or unowned) animals. Established internationally recognised high standards ('best practice') of veterinary clinical care for the individual patient were always followed and/or this work involved the use of cadavers. Ethical approval from a committee was therefore not specifically required for publication in *JFMS Open Reports*. Although not required, where ethical approval was still obtained it is stated in the manuscript.

**Informed consent** Informed consent (verbal or written) was obtained from the owner or legal custodian of all animal(s) described in this work (experimental or non-experimental animals, including cadavers) for all procedure(s) undertaken (prospective or retrospective studies). For any animals or people individually identifiable within this publication, informed consent (verbal or written) for their use in the publication was obtained from the people involved.

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