

Gingival Hyperplasia and Granulation Tissue Associated with a Feline Dental Resorptive Lesion

A 9-year-old, spayed/female DSH cat was presented for evaluation of an intraoral mass of the left rostral mandible. Upon noticing the mass, the owner immediately brought the cat to the emergency service. The mandibular left canine tooth had been noted to be missing approximately 2-months earlier.

Physical examination was normal except for abnormalities seen in the oral cavity and bilateral enlargement of the mandibular lymph nodes. The patient was fractious, preventing a complete oral examination. Preanesthetic laboratory tests including a chemistry panel and complete blood count were within normal limits. The patient was premedicated with a combination intramuscular injection of hydromorphone^a (0.05mg/kg), midazolam^b (0.5mg/kg), ketamine^c (6.0mg/kg) and atropine^d (0.02mg/kg). A 20-gauge intravenous catheter was placed and an isotonic balanced electrolyte solution^e (10 ml/kg/hr IV) was administered. Profound sedation was obtained from the premedication enabling intubation. Oxygen was delivered via mask prior to intubation and isoflurane^f was delivered once a cuffed endotracheal tube was placed. Thoracic radiographs (3-view) were obtained after induction of anesthesia due to the fractious nature of the patient. These radiographs showed no signs of metastatic disease. Oral examination revealed a missing mandibular left canine tooth (304), and a 9 x 9 x 10-mm erythematous mass present at the area of the missing canine (Fig. 1). The surface of the mass was ulcerated and hemorrhagic. The mandibular left third premolar tooth (307) was also missing, with a smooth gingival bulge present indicative of previous resorption.

The maxillary second premolar (106, 206) teeth, mandibular left fourth premolar (308), and the mandibular right third premolar (407) teeth exhibited signs of dental resorptive lesions (RL) at the gingival margin, with hyperplastic gingiva present at the cervical defects of these teeth. Full-mouth radiographs were obtained and revealed Stage 5, Type II root replacement resorption of teeth 304 and 307 (Fig. 1).¹ Stage 4, Type II RLs were present radiographically affecting teeth 106, 206, 308, and 407.

A left inferior alveolar nerve block was performed using 0.3cc of 0.5 % bupivacaine^g. The mass was removed at its base using sharp dissection and submitted for histopathology. A second biopsy was obtained with rongeurs deep to the site of initial excision including a portion of adjacent gingiva that was grossly normal. There was no remnant of a fractured crown beneath the mass, however calcified tissue was level with the surrounding gingiva in the area of root resorption. The level of the exposed calcified tissue was reduced to approximately 1-mm below the level of the adjacent soft tissue with a high-speed handpiece and a #4 round bur. No attempt was made to close the defect surgically or provide treatment for the other affected teeth pending the results of histopathologic evaluation of the mass. Hemostasis was achieved with a gauze sponge and digital pressure. Fine needle aspiration of the enlarged mandibular lymph nodes was performed for cytologic evaluation. Postoperative analgesic medication was administered (hydromorphone^h 0.05mg/kg QID X 24-hrs). Upon recovery from anesthesia, the patient was discharged with instructions for the owner to provide a soft food diet for 2-weeks.

Figure 1

Oral photograph (A) of a gingival hyperplastic mass in the area of the mandibular left canine tooth (304) in a 9-year old spayed/female DSH cat. An intraoral dental radiograph (B) shows Stage 5, Type II root replacement resorption of 304 and the mandibular left third premolar tooth (307).

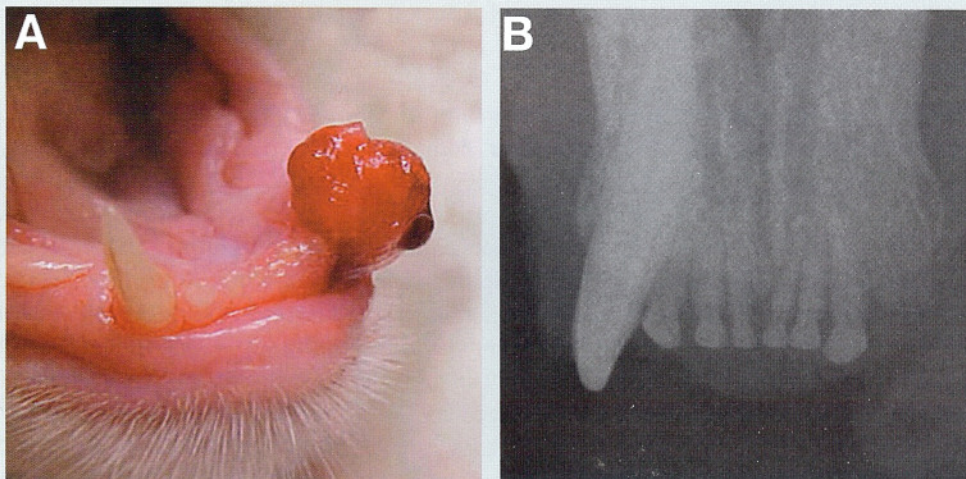
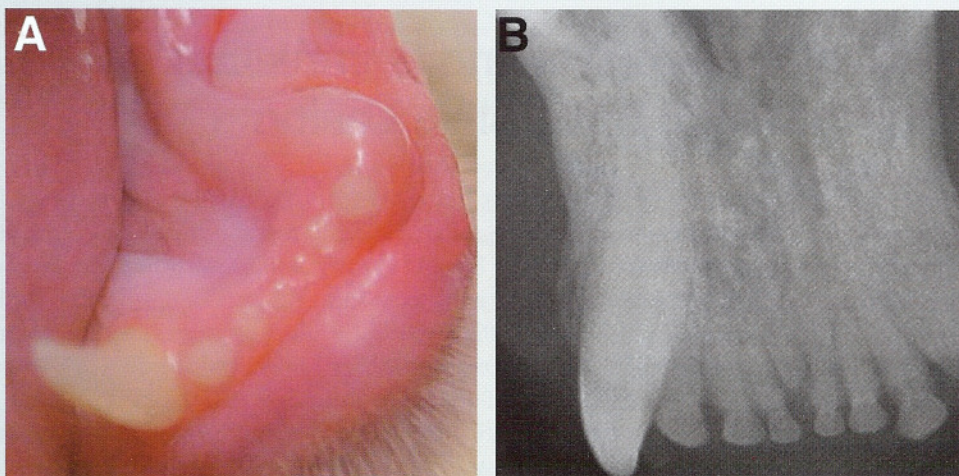


Figure 2

Oral photograph (A) 18-weeks following surgery for a gingival hyperplastic mass in the area of the mandibular left canine tooth (304) in a 9-year old spayed/female DSH cat. Note the smooth gingival bulge where the biopsy had been performed over the missing 304. An intraoral dental radiograph (B) shows no radiographic changes in the calcified tissue of the rostral mandible when compared to previous radiographs.



Cytological examination indicated reactive hyperplasia of the mandibular lymph nodes consisting of a mixed population of predominately mature small lymphocytes interspersed with low numbers of prolymphocytes, lymphoblasts, and plasma cells. No infectious organisms were seen. No cytologic evidence of neoplastic or metastatic disease was present in either lymph node. The mass had an ulcerated mucosal surface covering granulation tissue with a mixed inflammatory cell infiltrate consisting of plasma cells, lymphocytes, and neutrophils based on histopathologic examination. The deeper biopsy revealed hyperplastic gingiva supported by a fibrous stroma. There was no evidence of neoplasia in either sample.

The patient was returned for a 2-week postoperative examination. The surgical site had appropriate healing and the patient exhibited no signs of anorexia. The client, a third-year veterinary student, was advised to monitor the site and schedule an appointment to treat the other teeth affected by RLs. The patient was presented 18-weeks later for examination and admission to the dental service for treatment of teeth affected by RLs and follow-up evaluation of the previous surgical site. Teeth 106, 206 were completely resorbed and covered by healthy gingiva. Teeth 308 and 407 exhibited type II root resorption and were treated by crown amputation. Gingiva adjacent to the amputation sites was apposed with 5-0 poliglecaprone 25¹ in a simple interrupted pattern following elevation of buccal and lingual gingiva and mucosa. Full-mouth, intraoral dental radiographs and oral examination revealed no newly affected teeth. The previous surgical site revealed a smooth gingival bulge centered over the missing 304 (Fig. 2). There were no radiographic changes in the calcified tissue of the rostral mandible when compared to previous radiographs (Fig. 2).

Dental RLs are quite possibly the most common dental

disease of cats.² Recent studies suggest a 25.0 - 67.0 % prevalence rate, with variations seen in different populations and methods of diagnosis.^{3,4,5,6} Two distinct patterns of root resorption are commonly seen.¹ Type I appears clinically as a focal area of resorption commonly seen at the cervical portion of the tooth with invasion of adjacent gingiva and no loss of radicular density, whereas Type II resorption is more commonly seen radiographically as root replacement with loss of normal periodontal ligament space and decreased radiodensity of the roots. Type II resorption is associated less frequently with periodontal disease than Type I resorption.¹ The patient in this report exhibited gingivitis but no periodontal disease. Resorption has been previously classified into categories of surface, inflammatory, and replacement resorption.⁷ Type I root pathosis has radiographic characteristics of inflammatory root resorption, whereas Type II root pathosis is consistent with replacement resorption, or dentoalveolar ankylosis. The etiology of Type I and II resorption in cats is still unknown, although a recent study of calciotropic hormones in cats suggests excess dietary vitamin D may play a role.⁸

The increasing availability of dental intraoral radiography in companion animal practice has increased our ability to detect feline dental RLs. Radiography has been found to detect resorption which could not be seen on physical exam alone, increasing detection rates by 8.7 to 31.0 %.^{9,10} Although radiography is currently the best diagnostic tool for dental RLs, it is still relatively insensitive at detecting resorption, especially in its early stages. In a recent study, 60 % of clinically and radiographically normal teeth showed histologic signs of external root resorption and/or ankylosis in cats with overt lesions occurring elsewhere in the mouth.¹¹ The diffuse nature of the disease underlies the need to study "normal" teeth to elucidate the etiology of dental RLs in cats.

The degree of exuberant granulation tissue and gingival hyperplasia exhibited by the patient reported here is rarely seen as a sequela to dental RL in cats. The large mass of granulation tissue in this patient could have arisen from two possible sources. First, the granulation bed may have been a result of hyperplastic pulpitis. Commonly referred to as a "pulp polyp" in human dentistry, this condition is seen in young people as a result of pulp exposure through a carious lesion.¹² Due to the age of this patient, long period between loss of the canine tooth and clinical presentation, and the radiographic appearance of the root structure showing no overt pulp chamber, hyperplastic pulpitis seems less likely in this case. A second possible source of granulation tissue is the surrounding soft tissue structures, specifically the rich blood supply of the gingiva and attachment structures of the resorbing tooth. As tooth structure is lost due to resorption and inflammation, hyperplastic gingival epithelium commonly invades the excavated area of resorption at the gingival margin.¹³ When resorption weakens the tooth to the point of crown fracture, the hyperplastic gingiva continues to migrate across the defect, and in some cases, over the supragingival tooth remnants to create a prominent gingival bulge.

In skin, formation of a granulation bed is the first step in second intention wound repair.¹⁴ Granulation tissue consists of macrophages, fibroblasts, and budding capillaries within fibrin and fibronectin, acting as a scaffold for collagen deposition. Migration of epithelial cells requires establishment of a healthy granulation bed. Migration normally continues across this healthy wound bed until the cells come into contact with other epithelial cells, at which time contact inhibition prevents further epithelial migration. If blood clots are encountered, epithelial cells can secrete proteolytic enzymes, allowing epithelial cells to migrate below granulation tissue.¹⁴

There are some significant differences between wound healing in skin and oral mucosa. Migration of oral epithelial cells is more rapid (approximately 0.4-mm per day) than that of epidermal cells.¹⁵ Fibrous tissue that is formed in oral mucosa is remodeled to a greater extent, resulting in less contraction and scarring than in skin. It is suspected that fibroblasts in oral mucosa are phenotypically different from those in skin, as seen in their synthesis of glycosaminoglycans and response to certain cytokines.¹⁵

In this patient, the normal migration of epithelial cells may have been prevented by chronic trauma to the healing area, resulting in excessive granulation tissue formation. Alternatively, there may have been a portion of crown present prior to presentation that precluded epithelialization and fibrosis. By removing excess granulation tissue and reducing the level of bone apical to that of adjacent gingiva, a healthy wound bed was created, allowing epithelial cells to migrate across this area.

- ^a Dilaudid, Knoll Pharmaceuticals, Mt Olive, NJ
- ^b Versed, Abbott Laboratories, Chicago, IL
- ^c Ketaset, Fort Dodge Animal Health, Fort Dodge, IA
- ^d Atropine HCL, Phoenix Laboratories, St. Joseph's, MO
- ^e Norm-R, Abbott Laboratories, Chicago, IL
- ^f IsoFlo, Abbott Laboratories, Chicago, IL
- ^g Marcaine, Abbott Laboratories, Chicago, IL
- ^h Hydromorphone, Elkin-Sinn, Cherry Hill, NJ
- ⁱ Monocryl, Ethicon, Somerville, NJ

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