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# **NEOPLASTIC DISEASE**

# Canine and Feline Oral Cavity Tumours and Tumour-like Lesions: a Retrospective Study of 486 Cases (2015–2017)

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## Summary

Oral cavity tumours and tumour-like lesions are common in dogs and cats, and their diagnosis and classification requires histopathological examination. The aim of this study was to analyse retrospectively oral cavity lesions in dogs and cats in order to evaluate the distribution of inflammatory, hyperplastic and neoplastic lesions manifested as tumours. A total of 486 oral cavity tumours and tumour-like lesions (340 canine; 146 feline), diagnosed routinely from 2015 to 2017, were included. The lesions were classified as inflammatory, hyperplastic or neoplastic (benign and malignant). Histopathological diagnosis was based on haematoxylin and eosin staining and, when necessary, May-Grünwald–Giemsa (for mast cell tumours) or Masson's Fontana (for melanomas) stains or immunohistochemistry (for CD3, CD79 $\alpha$  and S100 markers). For dogs, 29.11% (99/340) of the lesions were benign tumours, 24.12% (82/340) were hyperplastic lesions and 14.7% (50/ 340) were inflammatory lesions. For cats, 4.79% (7/146) were benign tumours, 15.07% (22/146) were hyperplastic lesions and 57.53% (84/146) were inflammatory lesions. Furthermore, 23.24% (79/340) of canine cases were diagnosed with gingival hyperplasia and 19.12% (65/340) were diagnosed with peripheral odontogenic fibroma, while 43.84% (64/146) of feline cases were diagnosed with chronic lymphoplasmacytic stomatitis. Malignant tumours in dogs and cats constituted 32.06% (109/340) and 21.91% (32/146) of the lesions, respectively, with high-grade melanoma in dogs and squamous cell carcinoma in cats being the most common.

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#### Introduction

Oral cavity tumours and tumour-like lesions are detected commonly during routine clinical examination in both dogs and cats. These lesions can cause pain, discomfort and/or reluctance to eat or may be discovered accidentally, as they sometimes accompany periodontal diseases (Verhaert, 2010). Some oral cavity lesions in dogs and cats are consequences or manifestations of chronic gingivitis/stomatitis, but others can develop spontaneously, such as benign odontogenic neoplasia or malignancies (Lommer, 2013). However, it has been claimed that chronic irritation or persistent antigenic stimulation may play a role in malignant transformation (Coussens and Werb, 2002; Ekere *et al.*, 2010). It has also been suggested that canine and feline oral cavity tumours can develop due to passive tobacco smoke inhalation, flea control collars and dietary and environmental factors (Bertone *et al.*, 2003; Brønden *et al.*, 2009).

Canine and feline oral cavity tumour-like lesions may be true benign or malignant tumours, but hyperplastic and inflammatory lesions may clinically

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masquerade as neoplasia (Yoshida *et al.*, 1999; Kouki *et al.*, 2013). Despite the different nature of these lesions, they appear similar macroscopically, and therefore their clinical classification is difficult or impossible. There are some previous reports analysing the incidence of specific malignancies of the canine and feline oral cavity (Heldmann *et al.*, 2000; Ramos-Vara *et al.*, 2000; Nemec *et al.*, 2012; Liptak and Withrow, 2013; Bonfanti *et al.*, 2015; Gardner *et al.*, 2015; Wingo, 2018), but knowledge of the incidence of inflammatory, hyperplastic, benign and malignant oral cavity tumours and tumour-like lesions in dogs and cats in Eastern Europe is sparse.

The aim of this study was to analyse retrospectively oral cavity tumours (benign and malignant) and tumour-like lesions (inflammatory and hyperplastic) in dogs and cats, with regard to age, sex and breed, diagnosed in the Pathomorphological Laboratory of the Department of Pathological Anatomy, Faculty of Veterinary Medicine, University of Warmia and Mazury in Olsztyn, Poland, during the period of 2015–2017.

### **Materials and Methods**

The retrospective analysis was performed on 486 excisional biopsy samples (340 canine and 146 feline). These were archival diagnostic specimens diagnosed by two of the authors (IOD and KPC). The samples had been fixed in 10% neutral buffered formalin, processed routinely and embedded in paraffin wax. Sections were stained with Mayer's haematoxylin and (HE). When appropriate, May-Gruneosin wald-Giemsa stain (for mast cell tumours) or Masson's Fontana stain (for hypomelanotic melanomas) was also performed. In poorly differentiated tumours, immunohistochemistry (IHC) was performed at the request of the submitting veterinarian using markers specific for CD3 and CD79 $\alpha$  (for lymphoma) and S100 (for amelanotic melanoma). IHC involved initial heat-induced antigen retrieval (in Tris-EDTA buffer, pH 9.0), conducted in a microwave oven at 650W (samples were microwaved twice to the boiling point, and incubated in hot buffer for 20 min after boiling each time). Sections were subsequently overlaid with primary antibodies specific for CD3 (polyclonal rabbit anti-human CD3, 1 in 50 dilution; Dako, Glostrup, Denmark), CD79a (monoclonal mouse anti-human CD79a, clone HM57, 1 in 100 dilution; Bio-Rad Laboratories Inc., Hercules, California, USA) and S100 (polyclonal rabbit antibovine S100, 1 in 50 dilution; Dako). 'Visualization' of labelling was achieved by the immunoperoxidase method with 3, 3'-diaminobenzidine (DAB) as a substrate (Impress Universal Reagent anti-mouse/rabbit Ig peroxidase; Vector Laboratories, Burlingame, California, USA). The slides were counterstained with Mayer's haematoxylin. Positive and negative controls were processed together with the evaluated slides.

The data obtained from the archival records included sex, age, breed and histopathological diagnosis. The lesions were subsequently classified as hyperplastic, inflammatory or neoplastic (benign or malignant).

#### Results

# Canine Lesions

The sex distribution of the canine cases was 51.47% male (175 cases) and 48.53% female (165 cases). Most cases of the lesions were observed in crossbred dogs (Table 1).

Hyperplastic lesions constituted 24.12% (82/340) of the total oral lesions and were mostly gingival

Table 1Breed distribution of canine cases

Breed	Number of cases (%)
Crossbred	216 (63.53%)
Labrador retriever	15 (4.41%)
Yorkshire terrier	15 (4.41%)
German Shepherd dog	12 (3.53%)
Boxer	11 (3.24%)
American Staffordshire	6 (1.76%)
bull terrier	
Dachshund	6 (1.76%)
Bernese Mountain dog	5 (1.47%)
Spaniel	5 (1.47%)
Miniature schnauzer	4 (1.18%)
Other breeds (fox terrier,	Single cases <sup>*</sup>
French bulldog,	0
golden retriever,	
pointer, bloodhound,	
Chihuahua, great	
Dane, Irish setter,	
Maltese, poodle, pug,	
Alaskan malamute,	
black Russian terrier,	
border terrier, Cao	
File de San Miguel,	
Chinese crested,	
chow-chow,	
doberman pincher,	
English bulldog, giant	
schnauzer, Jack	
Russell terrier,	
miniature pincher,	
Shih-tzu, Siberian	
husky, St. Bernard,	
Wachtelhund, Welsh	
corgi)	

<sup>\*</sup>One to three cases each (0.29-0.88%).

hyperplasia (96.34% of all hyperplastic lesions; 79/ 82), with isolated cases of calcinosis circumscripta (2.44%; 2/82) and giant cell granuloma (1.22%; 1/ 82). The gingival hyperplasia was mostly accompanied by lymphoplasmacytic inflammation of various degrees (Fig. 1). Additionally, in nine cases of gingival hyperplasia, bony metaplasia of the connective tissue was also observed.

Inflammatory lesions (without distinct gingival hyperplasia) constituted 14.7% (50/340) of the total oral lesions and included lymphoplasmacytic (62% of all inflammatory lesions; 31/50), ulcerative (16%; 8/50), purulent (8%; 4/50), eosinophilic (6%; 3/50), pyogranulomatous (4%; 2/50) and mixed stomatitis (4%; 2/50). In six cases with inflammatory lesions, epithelial dysplasia was observed.

Benign neoplastic lesions constituted 29.11% (99/ 340) of the total oral lesions and included peripheral odontogenic fibroma (the second most common lesion in dogs; 65.66% of all benign tumours; 65/99), viral filiform papilloma (11.11%; 11/99), acanthomatous ameloblastoma (8.08%; 8/99), plasmacytoma (7.07%; 7/99) and single cases of granular cell tumour (3.03%; 3/99), ossifying fibroma (2.02%; 2/99), squamous papilloma (1.01%; 1/99), Schwannoma (1.01%; 1/99) and amyloid-producing odontogenic tumour (1.01%; 1/99). Peripheral odontogenic fibroma (Fig. 2) was diagnosed in nine out of 11 boxers included in this study.

The malignant tumours constituted 32.06% (109/ 340) of the total oral lesions in dogs. The most frequent malignancy was melanoma (Fig. 3) (35.78% of all malignant tumours; 39/109). Thirtyfive of these were high-grade and four were lowgrade melanomas according to the grading schemes of Spangler and Kass (2006), Bergin *et al.* (2011),



Fig. 2. Peripheral odontogenic fibroma, dog. Odontogenic epithelial islands (arrows) in hyperplastic connective tissue. HE.

Smedley *et al.* (2011) and Meuten *et al.* (2016). Other commonly diagnosed malignancies were squamous cell carcinoma (26.61%; 29/109), fibrosarcoma (12.84%; 14/109) and osteosarcoma (10.09%; 11/109). Other less frequent malignancies included mast cell tumour (5.5%; 6/109), lymphoma (3.67%; 4/109), including non-epitheliotropic (75%; 3/4) and epitheliotropic (25%; 1/4) lymphoma, undifferentiated sarcoma (2.75%; 3/109), haemangiosarcoma (1.83%; 2/109) and myxosarcoma (0.92%; 1/109).

Most cases of oral cavity tumours and tumour-like lesions were observed in middle-aged dogs (range 4-11 years), and few cases, particularly those with calcinosis circumscripta, eosinophilic stomatitis and viral filiform papilloma, were observed in young animals (range 0.5-3.5 years). Detailed data regarding



Fig. 1. Gingival hyperplasia, dog. Epithelial and connective tissue proliferation with plasma cell and lymphocyte infiltration (arrow). HE.



Fig. 3. Amelanotic melanoma, dog. Neoplastic cells with marked anisocytosis and anisokaryosis. HE. Inset: amelanotic melanoma, dog. S100 protein immunoexpression in neoplastic cells.

histological diagnosis, the number of cases and the mean age of dogs affected with oral cavity tumours and tumour-like lesions are summarized in Table 2.

#### Feline Lesions

The sex distribution of the feline cases was 79 (54.11%) male and 67 (45.89%) female. Most cases were seen in European shorthair cats (Table 3).

Hyperplastic lesions constituted 15.06% (22/146) of all cases and were represented by gingival hyperplasia (90.91% of all hyperplastic lesions; 20/22) and single cases of adenomatoid hyperplasia of the minor salivary glands (4.55%; 1/22) and peripheral giant cell granuloma (4.55%; 1/22). Similar to dogs, gingival hyperplasia was often accompanied by lymphoplasmacytic inflammation of various degrees. In two cases of gingival hyperplasia, bony metaplasia was observed.

Inflammatory lesions (without distinct gingival hyperplasia) constituted 58.22% (85/146) of the feline oral cavity lesions and included lymphoplasmacytic

 Table 2

 Canine oral cavity tumours and tumour-like lesions

Histological diagnosis	Number of	Age		
	cases (%)	$(\text{mean}\pm\text{SD})$		
HYPERPLASTIC 82 (24.12% of total oral lesions)				
Gingival hyperplasia	79 (23.24%)	$9 \pm 3.6$		
Calcinosis circumscripta	2 (0.59%)	$2 \pm 1.6$		
Giant cell granuloma	1 (0.29%)	8		
INFLAMMATORY 50 (14.7 % o	of total oral lesions	)		
Lymphoplasmacytic stomatitis	31 (9.12%)	$9 \pm 2.7$		
Ulcerative stomatitis	8 (2.35%)	$10 \pm 4.9$		
Purulent stomatitis	4 (1.18%)	$9 \pm 4.1$		
Eosinophilic stomatitis	3(0.88%)	$2 \pm 1$		
Pyogranulomatous stomatitis	2 (0.59%)	$6.5 \pm 2.1$		
Mixed stomatitis	2(0.59%)	$8.5 \pm 0.7$		
NEOPLASTIC: BENIGN 99 (29.11% of total oral lesions)				
Peripheral odontogenic fibroma	65 (19.12%)	$8 \pm 2.6$		
Viral filiform papilloma	11 (3.24%)	$2 \pm 1.83$		
Acanthomatous ameloblastoma	8 (2.35%)	$9 \pm 2.97$		
Plasmacytoma	7 (2.06%)	$10 \pm 3.6$		
Granular cell tumour	3(0.88%)	$12 \pm 0.58$		
Ossifying fibroma	2(0.59%)	$14 \pm 1.41$		
Squamous papilloma	1 (0.29%)	7		
Schwannoma	1 (0.29%)	5		
Amyloid-producing odontogenic	1 (0.29%)	8		
tumour				
NEOPLASTIC: MALIGNANT 109 (32.06% of total oral lesions)				
Melanoma	39 (11.47%)	$11.5\pm2.35$		
Squamous cell carcinoma	29~(8.53%)	$10 \pm 3.22$		
Fibrosarcoma	14 (4.12%)	$11 \pm 4.08$		
Osteosarcoma	11 (3.24%)	$9.5 \pm 3$		
Mast cell tumour	6 (1.76%)	$8.5\pm3.02$		
Lymphoma	4 (1.18%)	$8 \pm 4.57$		
Undifferentiated sarcoma	3(0.88%)	$9 \pm 7$		
Haemangiosarcoma	2 (0.59%)	$11 \pm 4.24$		
Myxosarcoma	1 (0.29%)	11		

Table 3 Breed distribution of feline cases

Breed	Number of cases (%)	
European shorthair	129 (88.36%)	
Maine Coon	5 (3.42%)	
Persian	5 (3.42%)	
British shorthair	4 (2.74%)	
Ragdoll	1(0.68%)	
Siamese	1 (0.68%)	
Sphinx	1 (0.68%)	

(75.29% of all inflammatory lesions; 64/85), eosinophilic (eosinophilic ulcer; Fig. 4) (18.82%; 16/85), purulent (2.35%; 2/85), pyogranulomatous (2.35%; 2/85) and mixed type (1.18%; 1/85) reactions. In three cases, the inflammatory infiltrates were accompanied by epithelial dysplasia. Additionally, in three cases, lymphoplasmacytic stomatitis was accompanied by tooth resorption.

Benign neoplastic lesions constituted 4.79% (7/146) of the feline oral cavity tumours and included peripheral odontogenic fibroma (28.57% of all benign tumours; 2/7), plasmacytoma (28.57%; 2/7), squamous papilloma (28.57%; 2/7) and Schwannoma (14.29%; 1/7).

Malignant tumours constituted 21.92% (32/146) of the feline oral cavity tumours. The most common malignancy of the feline oral cavity was squamous cell carcinoma (Fig. 5) (75% of all malignant tumours; 24/32), followed by fibrosarcoma (12.5%; 4/32). Other malignancies included a few or single cases of non-epitheliotropic lymphoma (6.25%; 2/32), melanoma (3.13%; 1/32) and osteosarcoma (3.13%; 1/32).

Most cases of oral cavity tumours and tumour-like lesions were seen in middle-aged cats (4–12 years), and only plasmacytomas were observed in two 3-



Fig. 4. Eosinophilic ulcer, cat. Ulceration (arrow) and eosinophilic infiltration of the gingiva. HE.



Fig. 5. Squamous cell carcinoma, cat. Neoplastic cells with marked anisocytosis and anisokaryosis. HE.

year-old cats. Detailed data regarding histological diagnosis, the number of cases and the mean age of cats affected diagnosed with oral cavity tumours and tumour-like lesions are summarized in Table 4.

#### Discussion

The results of this study showed that the majority (53.2%) of oral cavity lesions in dogs were either hyperplastic lesions (mostly gingival hyperplasia) or benign tumours (most commonly peripheral odontogenic fibroma), while in cats inflammatory lesions

Table 4
Feline oral cavity tumours and tumour-like lesions

Histological diagnosis	Number of	Age (mean + SD)		
	04303 (70)	(mean ± 5D)		
HYPERPLASTIC 22 (15.07 % of total oral lesions)				
Gingival hyperplasia	20 (13.7%)	$8 \pm 4.06$		
Adenomatoid hyperplasia of the minor salivary glands	1 (0.68%)	7.5		
Giant cell granuloma	1(0.68%)	12		
INFLAMMATORY 85 (58.22 % of total oral lesions)				
Lymphoplasmacytic stomatitis	64 (43.84%)	$7.5\pm3.8$		
Eosinophilic ulcer	16 (10.95%)	$7 \pm 4.51$		
Purulent stomatitis	2 (1.37%)	$6.5 \pm 2.12$		
Pyogranulomatous stomatitis	2(1.37%)	$6 \pm 5.66$		
Mixed stomatitis	1 (0.68%)	6		
NEOPLASTIC: BENIGN 7 (4.79 % of total oral lesions)				
Peripheral odontogenic fibroma	2(1.37%)	$8 \pm 1.41$		
Plasmacytoma	2(1.37%)	$3 \pm 0$		
Squamous papilloma	2(1.37%)	$9.5\pm0.71$		
Schwannoma	1 (0.68%)	8		
NEOPLASTIC: MALIGNANT 32 (21.92 % of total oral lesions)				
Squamous cell carcinoma	24 (16.44%)	$11 \pm 2.12$		
Fibrosarcoma	4(2.74%)	$9 \pm 3.52$		
Lymphoma	2(1.37%)	$9 \pm 0$		
Melanoma	1 (0.68%)	15		
Osteosarcoma	1 (0.68%)	12		

were most common (58.2%). In dogs and cats, both sexes were almost equally affected. The age of dogs and cats included in the study was similar, and ranged from 0.5 to 16.5 years (mean 9 years) and from 0.5 to 17 years (mean 8 years), respectively. Most tumours and tumour-like lesions occurred in middle-aged animals, which is in agreement with the findings of other studies (Gorlin and Peterson, 1967; Dorn and Priester, 1976; Svendenius and Warfvinge, 2010; Verhaert, 2010; Wingo, 2018). The most common lesions in young dogs (age range 0.5-2 years) were calcinosis circumscripta and viral filiform papilloma, as reported previously (Tafti et al., 2005; Uzal et al., 2016).

Gingival hyperplasia was the most common oral cavity lesion in dogs. Gingival hyperplasia is an excessive proliferation of overlying epithelium and stromal connective tissue, occurring as a focal or, more frequently, multifocal lesion (Verhaert, 2010). Gingival hyperplasia can be caused by dental plaque accumulation, gingivitis or certain medications, such as calcium channel blockers (i.e. amlodipine) or ciclosporin (Pariser and Berdoulay, 2011; Desmet and van der Meer, 2017). In the present study, gingival hyperplasia in dogs was accompanied by lymphoplasmacytic infiltrates in most cases, suggesting an inflammatory background to this lesion. However, inflammation secondary to chronic irritation cannot be excluded, as these cases lacked a full clinical history.

Bony metaplasia of the connective tissue was observed in nine cases of gingival hyperplasia in dogs. Metaplastic bone formation can result from chronic gingival inflammation (Reichart *et al.*, 1989). Nevertheless, because bony metaplasia is not uncommon within various gingival tumours and tumour-like lesions in dogs, it likely has little or no diagnostic importance (Reichart *et al.*, 1989; Uzal *et al.*, 2016), as is the case in man (D'Astous, 2015).

Peripheral odontogenic fibroma was the second most common oral cavity lesion in dogs, which was in accordance with the findings of other studies (Verstraete *et al.*, 1992; Gardner, 1996; Verhaert, 2010). Additionally, in the present study, peripheral odontogenic fibroma was diagnosed in nine out of 11 boxers, suggesting a breed predisposition, which has also been reported previously (Gorlin and Peterson, 1967; Verhaert, 2010).

Although canine acanthomatous ameloblastoma is a common odontogenic tumour in dogs (Wingo, 2018), only eight cases were reported in the present study. This tumour originates from the epithelial rests of Malassez or the reduced enamel epithelium of the Serres rests (Poulet *et al.*, 1992; Wingo, 2018). Macroscopically, acanthomatous ameloblastoma may look similar to peripheral odontogenic fibroma. However, unlike the latter, the lesion is highly invasive, albeit ostensibly benign, and therefore wide surgical excision with partial mandibulectomy or maxillectomy is recommended (Wingo, 2018).

Inflammatory lesions constituted approximately 15% of the canine cases. Stomatitis in the gingival sulcus involves interactions between the host immune system and various antigens. Although the precise aetiology in most cases is unknown, various infectious agents have been described, including bacteria, fungi and viruses (Lyon, 2005). The most common infectious agents of canine stomatitis include Actinobacillus spp., Actinomyces spp., Porphyromonas spp., Bacterioides spp., Streptococcus spp., Staphylococcus spp. and Proteus spp. (Senhorinho et al., 2011; Polkowska et al., 2014). Other possible factors include genetics, nutrition, environment and domestication (Lyon, 2005). Dogs affected by oral cavity inflammation had higher levels of alanine transaminase, aspartate transaminase and urea concentration in serum compared with healthy animals, which suggested co-existing health problems connected with the liver, kidney and/or heart (Senhorinho et al., 2012; Polkowska et al., 2014).

Our study showed that 32.06% of the evaluated oral cavity lesions in dogs were malignant tumours. A similar result was obtained by Wingo (2018). This result is inconsistent with the results of another study, in which 15% of evaluated canine oral biopsies were diagnosed as malignant lesions (Svendenius and Warfvinge, 2010). The most common malignancy was high-grade melanoma, which was also previously reported (Gorlin and Peterson, 1967; Borthwick et al., 1982; Smith et al., 2002; Bergman, 2007; Svendenius and Warfvinge, 2010; Bonfanti et al., 2015), followed by squamous cell carcinoma and fibrosarcoma. All evaluated cases of oral melanoma in dogs occurred in older individuals; however, this malignancy can also be observed in young dogs (Ramos-Vara et al., 2000; Bergman, 2007). Highly pigmented melanomas are not diagnostically however, hypomelanotic challenging; and amelanotic tumours are difficult to diagnose and often mimic other lesions, therefore requiring immunophenotyping (Bergman, 2007).

The second most common malignancy in dogs was squamous cell carcinoma. Previous studies also showed that squamous cell carcinoma is a common malignant tumour of the oral cavity of dogs (Dorn and Priester, 1976; Verhaert, 2010; Munday *et al.*, 2017). The neoplasm is locally invasive and has low metastatic potential; however, squamous cell carcinoma arising from lingual epithelium has higher metastatic potential than its counterpart arising elsewhere in the oral cavity (Carpenter *et al.*, 1993; Hayes *et al.*, 2007; Buckley and Nuttall, 2012). Fibrosarcoma was the third most common oral cavity malignancy in dogs. This tumour is locally invasive and can infiltrate the adjacent bone. Furthermore, in dogs, metastases to regional lymph nodes and distant organs are common (Munday *et al.*, 2017).

In cats, the majority (58.2%) of oral cavity lesions were stomatitis (mostly lymphoplasmacytic), which was also reported previously (Wingo, 2018). Stomatitis with ulceration is mostly associated with infection by feline calicivirus, feline immunodeficiency virus, feline leukaemia virus, feline herpesvirus or anaerobic bacteria (Healey *et al.*, 2007). Cats with stomatitis had differences in cytokine expression and immunoglobulin profiles compared with healthy animals, and this may contribute to oral cavity inflammation (Harley *et al.*, 1999). It has been suggested that immunosuppression caused by unconnected health problems may play a role in oral cavity inflammation (Williams and Aller, 1992).

Gingival hyperplasia in cats was less common than in dogs and in all cases was accompanied by lymphoplasmacytic inflammation of various degrees, suggesting a primary inflammatory origin. In two cases, bony metaplasia was observed. Knaake and Verhaert (2010) suggested that gingival hyperplasia with bone- or cementum-like hard tissue formation in cats should be diagnosed as peripheral ossifying fibroma. Peripheral odontogenic fibroma was diagnosed in only two cats, confirming that it is a rare condition in this species (Stebbins *et al.*, 1989; Wingo, 2018).

Malignant neoplasms constituted 21.91% of the feline cases. Squamous cell carcinoma constituted the majority of malignant tumours, consistent with previous reports (Dorn and Priester, 1976; Stebbins et al., 1989; Bertone et al., 2003; Hayes et al., 2007; Verhaert, 2010; Bonfanti et al., 2015; Wingo, 2018). This tumour often clinically resembles a non-healing ulcer and mimics foreign body reactions or eosinophilic ulcers (Verhaert, 2010; Martin et al., 2011). As in dogs, feline oral cavity squamous cell carcinomas are locally invasive and have low metastatic potential. Several factors have been described that can lead to the development of oral squamous cell carcinoma in cats, including contact with carcinogens in flea collars, topical tick and flea medications or grooming behaviour (Bertone et al., 2003; Wingo, 2018). Chronic stomatitis has also been proposed as a predisposing factor for squamous cell carcinoma in both dogs and cats (Stebbins et al., 1989).

In this study, fibrosarcoma was reported in only four cats. The low incidence of oral cavity fibrosarcoma in cats is in contrast to the high incidence of its dermal counterpart (Fox, 1995). The tumour in cats has low metastatic potential (Stebbins *et al.*, 1989; Munday *et al.*, 2017). Oral melanoma was reported only in one cat (15 years old). Oral melanoma occurs rarely in cats (compared with dogs) and the mean age of affected cats is 12 years (Munday *et al.*, 2017).

In conclusion, most cases of oral cavity tumours and tumour-like lesions in dogs and cats are hyperplastic, inflammatory or benign tumours, and occur in middle-aged animals, without sex predilection. The most common lesion in dogs was gingival hyperplasia, followed by peripheral odontogenic fibroma, while in cats lymphoplasmacytic stomatitis was most common. Boxers appear predisposed to peripheral odontogenic fibroma. Young age may be a risk factor for calcinosis circumscripta and viral papilloma in dogs. The most common oral cavity malignancy in dogs and cats is high-grade melanoma and squamous cell carcinoma, respectively. Feline oral melanoma is a rare disease. Due to the wide spectrum of observed oral cavity tumours and tumour-like lesions and their macroscopic similarity, clinical diagnosis may be challenging, therefore histopathological examination is recommended in each case.

#### References

- Bergin IL, Smedley RC, Esplin DG, Spangler WL, Kiupel M (2011) Prognostic evaluation of Ki67 threshold value in canine oral melanoma. *Veterinary Pathology*, **48**, 41–53.
- Bergman PJ (2007) Canine oral melanoma. Clinical Techniques in Small Animal Practice, 22, 55-60.
- Bertone ER, Snyder LA, Moore AS (2003) Environmental and lifestyle risk factors for oral squamous cell carcinoma in domestic cats. *Journal of Veterinary Internal Medicine*, **17**, 557–562.
- Bonfanti U, Bertazzolo W, Garcis M, Roccabianca P, Romanelli G *et al.* (2015) Diagnostic value of cytological analysis of tumour and tumour-like lesions of the oral cavity in dogs and cats: a prospective study on 114 cases. *Veterinary Journal*, **205**, 322–327.
- Borthwick R, Else RW, Head KW (1982) Neoplasia and allied conditions of the canine oropharynx. *Veterinary Annual*, **22**, 248–269.
- Brønden LB, Eriksen T, Kristensen AT (2009) Oral malignant melanomas and other head and neck neoplasm in Danish dogs – data from the Danish Veterinary Cancer Registry. Acta Veterinaria Scandinavica, 51, 54.
- Buckley L, Nuttall T (2012) Feline eosinophilic granuloma complex(ities): some clinical clarification. *Journal of Feline Medicine & Surgery*, **14**, 471–481.

- Carpenter LG, Withrow SJ, Powers BE, Ogilvie GK, Schwarz PD et al. (1993) Squamous cell carcinoma of the tongue in 10 dogs. *Journal of the American Animal Hospital Association*, **29**, 19–24.
- Coussens LM, Werb Z (2002) Inflammation and cancer. *Nature*, **6917**, 860–867.
- D'Astous J (2015) Periodontology: an overview of alveolar bone expansion. *Canadian Veterinary Journal*, 56, 295–300.
- Desmet L, van der Meer J (2017) Antihypertensive treatment with telmisartan in a cat with amlodipineinduced gingival hyperplasia. *Journal of Feline Medicine and Surgery Open Reports*, **3** 2055116917745236.
- Dorn CR, Priester WA (1976) Epidemiologic analysis of oral and pharyngeal cancer in dogs, cats, horses, and cattle. *Journal of the American Veterinary Medical Association*, **169**, 1202–1206.
- Ekere AU, Adiela VU, Kejeh BM (2010) Malignant change in chronic irritation: a case series. West African Journal of Medicine, 29, 38-40.
- Fox LE (1995) Feline cutaneous and subcutaneous neoplasms. Veterinary Clinics of North America: Small Animal Practice, 25, 961–979.
- Gardner DG (1996) Epulides in the dog: a review. Journal of Oral Pathology & Medicine, 25, 32−37.
- Gardner H, Fidel J, Haldorson G, Dernell W, Wheeler B (2015) Canine oral fibrosarcomas: a retrospective analysis of 65 cases (1998–2010). *Veterinary and Comparative Oncology*, **13**, 40–47.
- Gorlin RJ, Peterson WC Jr. (1967) Oral disease in man and animals based on analysis of 1,135 cases in a variety of species. Archives of Dermatology, 96, 390-403.
- Harley R, Helps CR, Harbour DA, Gruffydd-Jones TJ, Day MJ (1999) Cytokine mRNA expression in lesions in cats with chronic gingivostomatitis. *Clinical and Diagnostic Laboratory Immunology*, 6, 471–478.
- Hayes AM, Adams VJ, Scase TJ, Murphy S (2007) Survival of 54 cats with oral squamous cell carcinoma in United Kingdom general practice. *Journal of Small Animal Practice*, **48**, 394–399.
- Healey KA, Dawson S, Burrow R, Cripps P, Gaskell CJ et al. (2007) Prevalence of feline chronic gingivostomatitis in first opinion veterinary practice. *Journal of Feline* Medicine & Surgery, 9, 373–381.
- Heldmann E, Anderson MA, Wagner-Mann C (2000) Feline osteosarcoma: 145 cases (1990–1995). Journal of the American Animal Hospital Association, 36, 518–521.
- Knaake FAC, Verhaert L (2010) Histopathology and treatment of nine cats with multiple epulides. *Vlaams Diegeneeskundig Tijdschrift*, **79**, 48–53.
- Kouki MI, Papadimitriou SA, Kazakos GM, Savas I, Bitchava D (2013) Periodontal disease as a potential factor for systemic inflammatory response in the dog. *Jour*nal of Veterinary Dentistry, **30**, 26–29.
- Liptak JM, Withrow SJ (2013) Oral tumours. In: Withrow & MacEwen's Small Animal Clinical Oncology, 5<sup>th</sup> Edit.,
   SJ Withrow, DM Vail, Eds., Elsevier Saunders, Philadelphia, pp. 381-398.

- Lommer MJ (2013) Oral inflammation in small animals. Veterinary Clinics of North America: Small Animal Practice, 43, 555–571.
- Lyon KF (2005) Gingivostomatits. Veterinary Clinics of North America: Small Animal Practice, **35**, 891–911.
- Martin CK, Tannehill-Gregg SH, Wolfe TD, Rosol TJ (2011) Bone-invasive oral squamous cell carcinoma in cats: pathology and expression of parathyroid hormone-related protein. *Veterinary Pathology*, **48**, 302–312.
- Meuten DJ, Moore FM, George JW (2016) Mitotic count and the field of view area: time to standardize. *Veterinary Pathology*, **53**, 7–9.
- Munday JS, Löhr CV, Kiupel M (2017) Tumors of alimentary tract. In: *Tumors in Domestic Animals*, 5<sup>th</sup> Edit., DJ Meuten, Ed., Wiley Blackwell, Raleigh, pp. 499-602.
- Nemec A, Murphy B, Kass PH, Verstraete FJ (2012) Histological subtypes of oral non-tonsillar squamous cell carcinoma in dogs. *Journal of Comparative Pathology*, 147, 111–120.
- Pariser MS, Berdoulay P (2011) Amlodipine-induced gingival hyperplasia in a great Dane. *Journal of the American Animal Hospital Association*, **47**, 375–376.
- Polkowska I, Sobczyńska-Rak A, Gołyńska M (2014) Analysis of gingival pocket microflora and biochemical blood parameters in dogs suffering from periodontal disease. In Vivo, 6, 1085–1090.
- Poulet FM, Valentine BA, Summers BA (1992) A survey of epithelial odontogenic tumors and cysts in dogs and cats. *Veterinary Pathology*, **29**, 369–380.
- Ramos-Vara JA, Beissenherz ME, Miller MA, Johnson GC, Pace LW et al. (2000) Retrospective study of 338 canine oral melanomas with clinical, histologic and immunohistochemical review of 129 cases. Veterinary Pathology, 37, 597–608.
- Reichart PA, Philipsen HP, Dürr UM (1989) Epulides in dogs. Journal of Oral Pathology & Medicine, 18, 92-96.
- Senhorinho GN, Nakano V, Liu C, Song Y, Finegold SM et al. (2011) Detection of Porhyromonas gulae from subgingival biofilms of dogs with and without periodontitis. Anaerobe, 17, 257–258.
- Senhorinho GN, Nakano V, Liu C, Song Y, Finegold SM et al. (2012) Occurrence and antimicrobial susceptibility of *Porphyromonas* spp. and *Fusobacterium* spp. in dogs with and without periodontitis. Anaerobe, 4, 381–385.

- Smedley RC, Spangler WL, Esplin DG, Kitchell BE, Bergman PJ et al. (2011) Prognostic markers for canine melanocytic neoplasms: a comparative review of the literature and goals for future investigation. Veterinary Pathology, 48, 54–72.
- Smith SH, Goldschmidt MH, McManus PM (2002) A comparative review of melanocytic neoplasms. *Veterinary Pathology*, **39**, 665–678.
- Spangler WL, Kass PH (2006) The histologic and epidemiologic bases for prognostic considerations in canine melanocytic neoplasia. *Veterinary Pathology*, **43**, 136–149.
- Stebbins KE, Morse CC, Goldschmidt MH (1989) Feline oral neoplasia: a ten-year survey. *Veterinary Pathology*, 26, 121–128.
- Svendenius L, Warfvinge G (2010) Oral pathology in Swedish dogs: a retrospective study of 280 biopsies. *Journal of Veterinary Dentistry*, 27, 91–97.
- Tafti AK, Hanna P, Bourque AC (2005) Calcinosis circumscripta in the dog: a retrospective pathological study. *Journal of Veterinary Medicine*, **52**, 13–17.
- Uzal FA, Plattner BL, Hostetter JM (2016) Alimentary system. In: Jubb, Kennedy & Palmer's Pathology of Domestic Animals, 6<sup>th</sup> Edit, Vol. 2, MG Maxie, Ed., Elsevier, St. Louis, pp. 1–257.
- Verhaert L (2010) Oral proliferative lesions in the dog and cat. European Journal of Companion Animal Practice, 20, 252-264.
- Verstraete FJM, Ligthelm AJ, Weber A (1992) The histologic nature of epulides in dogs. *Journal of Comparative Pa*thology, **106**, 169–182.
- Williams CA, Aller MS (1992) Gingivitis/stomatitis in cats. Veterinary Clinics of North America: Small Animal Practice, 22, 1361–1378.
- Wingo K (2018) Histopathologic diagnoses from biopsies of the oral cavity in 403 dogs and 73 cats. *Journal of Veterinary Dentistry*, **35**, 7–17.
- Yoshida K, Yanai T, Iwasaki T, Sakai H, Ohta J et al. (1999) Clinicopathological study of canine oral epulides. *Journal of Veterinary Medical Science*, 61, 897–902.

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