Clinicopathologic characterization of odontogenic tumors and focal fibrous hyperplasia in dogs: 152 cases (1995–2005)

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Objective—To characterize clinicopathologic features of the most common odontogenic tumors and focal fibrous hyperplasia (FFH) in dogs.

Design—Retrospective case series.

Animals—152 dogs evaluated for oral tumors of possible odontogenic origin at the William R. Pritchard Veterinary Medical Teaching Hospital of the University of California-Davis between 1995 and 2005.

Procedures—Information was collected from records, including dog breed, age, reproductive status, and location of lesion in the oral cavity. Histologic slides pertaining to each dog were reviewed by 3 investigators. Data regarding clinicopathologic features of the 3 most common lesions (canine acanthomatous ameloblastoma [CAA], peripheral odontogenic fibroma [POF], and FFH) were summarized.

Results—152 dogs with odontogenic tumors or FFH were identified. Sixty-eight (45%) dogs had CAA, 47 (31%) had POF, 24 (16%) had FFH, and 13 (9%) had other odontogenic tumors. Canine acanthomatous ameloblastoma was present most commonly in the rostral aspect of the mandible, with POF and FFH more common in the rostral aspect of the maxilla. Males and females were equally represented among dogs with CAA and FFH. Castrated males were overrepresented among dogs with POF. Golden Retrievers, Akitas, Cocker Spaniels, and Shetland Sheepdogs were overrepresented among dogs with CAA. No breed predisposition was detected for FFH or POF. Dogs with FFH had a greater mean age at initial evaluation than did dogs with CAA or POF.

Conclusions and Clinical Relevance—CAA, POF, and FFH have distinct clinical patterns that may help clinicians and pathologists identify such lesions more readily. (*J Am Vet Med Assoc* 2011;238:495–500)

Odontogenic tumors are neoplasms derived from ectodermal, ectomesenchymal, or mesenchymal components of the tooth-forming apparatus.[1] Reactive lesions of the gingiva can sometimes manifest with a gross appearance similar to that of odontogenic tumors, making histologic differentiation essential when determining an appropriate treatment approach. Odontogenic tumors are generally considered rare and their histologic identification challenging.[2] There is much confusion regarding the nomenclature and the origins of odontogenic tumors as well as reactive lesions of the gingiva in dogs. Various authors have attempted to standardize and classify these lesions on the basis of histologic appearance and, in some cases, the lesion's inductive abilities; however, such approaches have proven difficult.[2–6]

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Abbreviations

APOT	Amyloid-producing odontogenic tumor
CAA	Canine acanthomatous ameloblastoma
FFH	Focal fibrous hyperplasia
POF	Peripheral odontogenic fibroma
WHO	World Health Organization

The term epulis is often applied to expansile gingival lesions. The term is purely descriptive, derived from the Greek term epi-oulon, meaning on the gum; it does not provide additional information with regard to the histologic or pathological nature of a lesion.[7] Dubielzig et al[3] were among the first to attempt classification of canine epulides, which they believed to be of periodontal origin. They described 3 types: fibromatous epulis, ossifying epulis, and acanthomatous epulis. Another group then reclassified fibromatous and ossifying epulides as variations of the same lesion, the fibromatous epulis.[8] Further clarification was attempted through comparison of canine fibromatous epulis with the well-established but uncommon human POF (WHO type).[9,10] The comparison revealed that the 2 lesions were equivalent, and it was suggested that the canine lesion be referred to by the same name as the human lesion. To add further confusion, Dubielzig's group did not differentiate among fibromatous epulides, neoplastic lesions, and FFH, which are reactive inflammatory lesions with similar histological features. Several other authors have since attempted to clarify the difference between these histologically similar lesions, and the current acceptable term for the inflammatory lesion is FFH.[5,6,10,11] Because FFH constitutes a large proportion of gingival lesions in dogs and given the confusion surrounding its histologic identification and nomenclature, clarification of its nonneoplastic nature is important.[6]

The so-called acanthomatous epulis has some microscopic features in common with human ameloblastoma. Clinically, however, the canine lesions have similar invasive and destructive behavior to the human intraosseous ameloblastoma. The tumor is now named CAA because it is its own entity with no precise human equivalent.[7,12,13]

Several other less common odontogenic tumors have been identified in dogs, including APOT and odontoma; however, few case reports and studies exist to help elucidate their nature in dogs.[5,6,14]

Although an understanding of basic clinical features such as dog age, reproductive status, breed, and location of the masses within the oral cavity can play an important role in developing differential diagnoses for oral masses, little has been reported on this topic in the veterinary literature. The purpose of the study reported here was therefore to characterize clinicopathologic features of the most common odontogenic tumors and FFH in dogs.

Materials and Methods

Case selection—A search of the medical records database of the William R. Pritchard Veterinary Medical Teaching Hospital of the University of California-Davis was conducted to identify dogs in which oral tumors of possible odontogenic origin were diagnosed during a 10-year period (1995 to 2005). Keywords used to identify cases were epulis, odontogenic, ameloblastoma, and odontoma. Dogs were included in the study if they had complete medical records, including signalment and tumor location.

Medical records review—Data on tumor location within the oral cavity and dog breed, reproductive status, and age were collected. For classification of tumor location, the oral cavity was divided into 4 regions. The rostral aspect of maxilla and mandible extended from the level of the first incisor tooth to the level of the second premolar tooth. The caudal aspect of the maxilla and mandible was defined as the dentate region caudal to the second premolar tooth.

Histologic evaluation and classification—Historically, tissue samples from each dog had been fixed in neutral-buffered 10% formalin and embedded in paraffin. Five-micrometer sections were obtained and stained with H&E. For the study, the histologic slides of each dog were reviewed by 3 investigators (FJV, DPC, and a board-certified pathologist).

Specific histologic criteria were used to classify each of the 3 most common lesions. Canine acanthomatous ameloblastoma consists of islands and cords of squamous epithelium that have invaded irregularly through a connective tissue stroma (**Figure 1**).[13] Most of the basal cells are arranged in palisades and have vacuolated cytoplasm with reverse polarization of the nuclei.[5,13,15] The basal cells typically surround regularly arranged sheets of squamous epithelium.[5,12]

According to the WHO classification system for tumors of the alimentary system in domestic animals,[15] a fibromatous epulis is histologically described as primarily composed of periodontal ligament-type stroma characterized by the presence of regularly positioned stellate mesenchymal cells, smooth fibrillar collagen matrix, and regularly positioned and dilated but empty blood vessels.[15] Other features commonly observed are cords of odontogenic epithelium and cell-poor collagen matrix resembling alveolar bone, cementum, or even dentin. The WHO system also maintains that fibromatous epulides are benign tumors that many consider reactive hyperplasia rather than neoplasia. Because we believe that this description is in fact a combination of the histologic features of POF (neoplastic) and FFH (reactive),[5,6,9,11] we subdivided the lesions that fit the WHO histologic criteria of fibromatous epulides of periodontal origin into POF and FFH on the basis of whether odontogenic epithelium was present.[5,6,9]

In POF, there is typically a mass of cellular, fibroblastic connective tissue that is separated from the surface epithelium by a zone of histologically normal fibrous connective tissue.[5,9] The fibroblastic connective tissue is often in the form of strands interwoven within a looser connective matrix. The connective tissue is often highly vascularized. Rests of odontogenic epithelium, located in the connective tissue, are present but

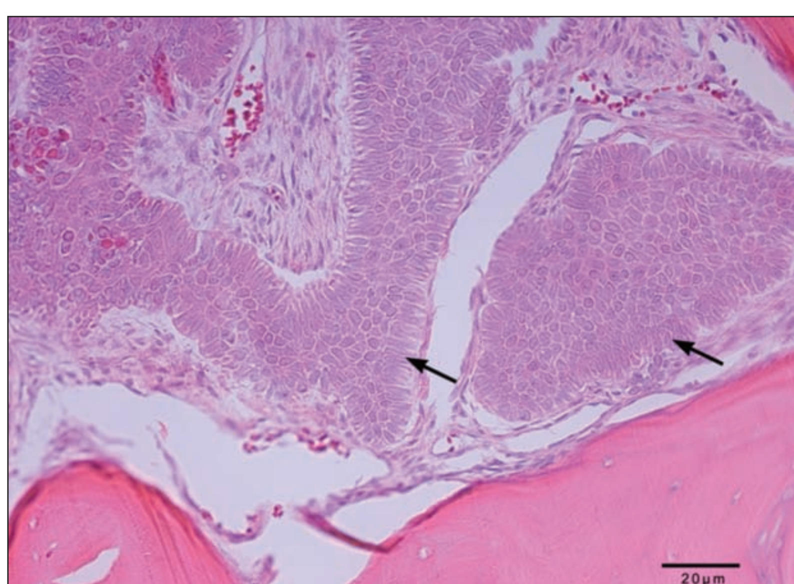

Figure 1—Representative photomicrograph of tissue from a CAA in the mouth of a dog. Islands and cords of squamous epithelium (arrows) have invaded irregularly through a connective tissue stroma. H&E stain; bar = 20 µm.

vary in number (**Figure 2**).[5,9] Bone and other foci of collagenous matrix, which may be considered as osteoid, dentinoid, or cementum-like, are often present in variable amounts.

Focal fibrous hyperplasia is characterized by dense fibrous connective tissue.[5] Fibroblasts are mature and widely scattered in a dense collagen matrix (**Figure 3**).[16] Foci of dystrophic calcification are often spread out amid the cellular fibrous tissue. The defining feature that distinguishes FFH from POF is the absence of odontogenic epithelium; however, strands of surface epithelium are often visible within the fibrous tissue.[6] The connective tissue in the immediate vicinity of the epithelium is highly cellular, and young stellate fibroblasts are present. The superficial aspect of the lesion may be ulcerated with associated inflammation.[5,6] Seven less common tumor types were also identified by use of the WHO tumor classification scheme[15] and specific descriptions.[1,5,6,14,17–21]

Statistical analysis—Computer software[a] was used to perform all statistical analyses. Continuous data are summarized as mean ± SD. The χ^2 test of homogeneity was used to evaluate associations between categorical variables (ie, age, reproductive status, breed, and tumor location). The χ^2 goodness-of-fit test was used to compare distributions of dog age, reproductive status, and breed with those of the general hospital population during the same period. Values of $P < 0.05$ were considered significant.

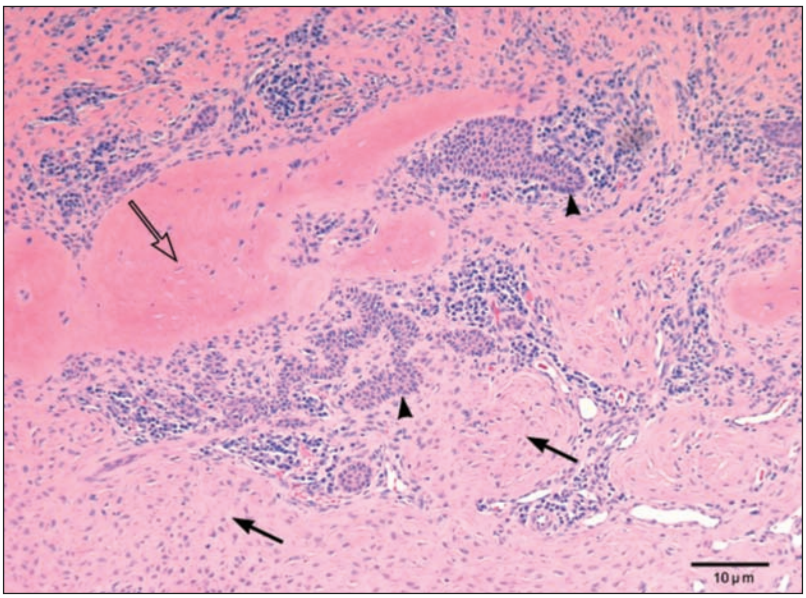

Figure 2—Representative photomicrograph of tissue from a POF in the mouth of a dog. A mass of cellular, fibroblastic connective tissue (arrows) appears separated from the surface epithelium by a zone of histologically normal fibrous connective tissue. Rests of odontogenic epithelium (arrowheads), located in the connective tissue, are present but vary in number. Mineralized tissue (open arrow) may also be present. H&E stain; bar = 10 µm.

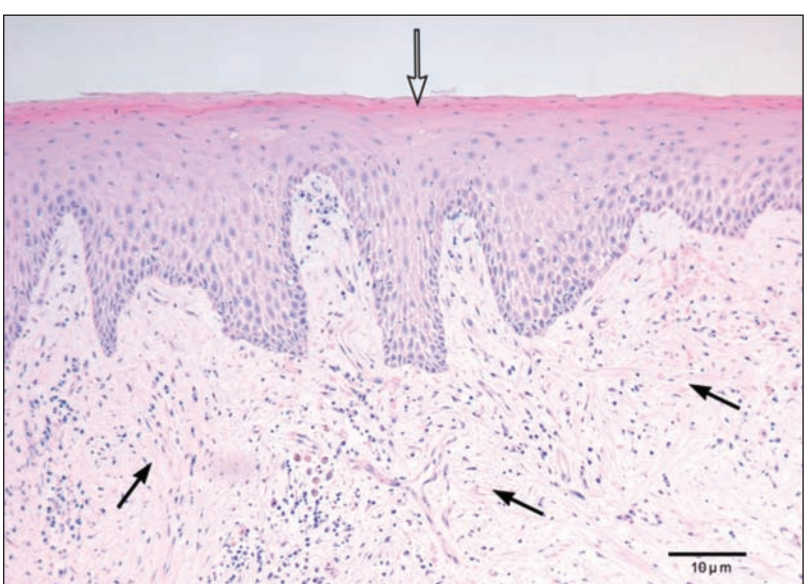

Figure 3—Representative photomicrograph of tissue from an FFH in the mouth of a dog. Dense, fibrous connective tissue is evident (arrows) below the hyperplastic layer of surface epithelium (open arrow). Notice the lack of odontogenic epithelium. H&E stain; bar = 10 µm.

Results

One hundred fifty-two dogs with oral tumors of possible odontogenic origin were included in the study. The 3 most common types of epulides in these dogs were CAA (n = 68 dogs; 45%), POF (47; 31%), and FFH (24; 16%). The remaining 13 (9%) dogs had various other tumor types (APOT [5], ameloblastic carcinoma [2], complex odontoma [2], and 1 each of compound odontoma, desmoplastic ameloblastoma, odontogenic carcinoma, and odontoameloblastoma), but none of these types was of sufficient quantity to include in meaningful statistical comparisons. Ages of dogs with an APOT ranged from 1 to 8 years.

CAA—The mean ± SD age of the 68 dogs with CAA at first evaluation was 8.8 ± 2.6 years. The distribution of these dogs by reproductive status differed significantly ($P = 0.044$) from that of the hospital population. Sexually intact males and females were underrepresented among dogs with CAA, whereas the proportions of neutered males and females were close to the expected value (**Table 1**). The female-to-male ratio was 1.03:1. There was no significant correlation between age and reproductive status of dogs with CAA.

A significant ($P < 0.001$) difference was detected in the distribution of CAAs within various regions of the oral cavity (**Figure 4**), with the highest proportion present in the rostral aspect of the mandible. The ratio of mandibular to maxillary lesions was 2.3:1. There was no association between dog reproductive status and tumor location. On the other hand, the distribution of breed among the dogs

Table 1—Observed versus expected distribution (%) of dogs evaluated for the 2 most common odontogenic tumors (CAA [n = 68] and POF [47]) and FFH (24) by reproductive status, as compared with the distribution among the general population of dogs (n = 66,442) in a veterinary teaching hospital from 1995 to 2005.

	CAA		POF		FFH	
Reproductive status	Observed	Expected	Observed	Expected	Observed	Expected
Sexually intact female	3	11	4	11	0	11
Spayed female	47	40	30	40	33	40
Sexually intact male	9	15	11	16	8	15
Castrated male	41	33	55	33	58	33

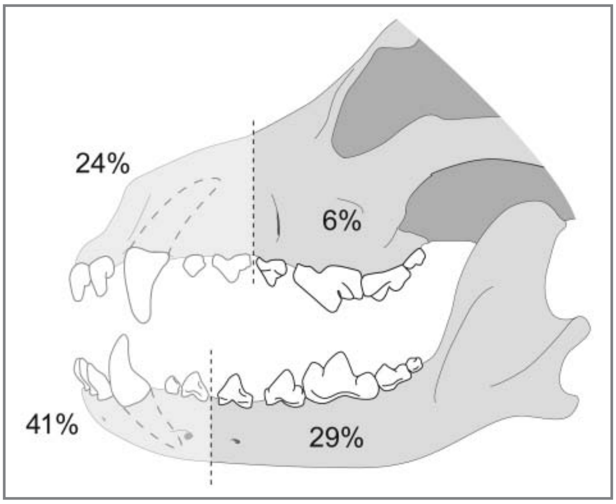

Figure 4—Distribution of CAA in the oral cavities of dogs (n = 68) evaluated for oral tumors of possible odontogenic origin at a veterinary teaching hospital from 1995 through 2005.

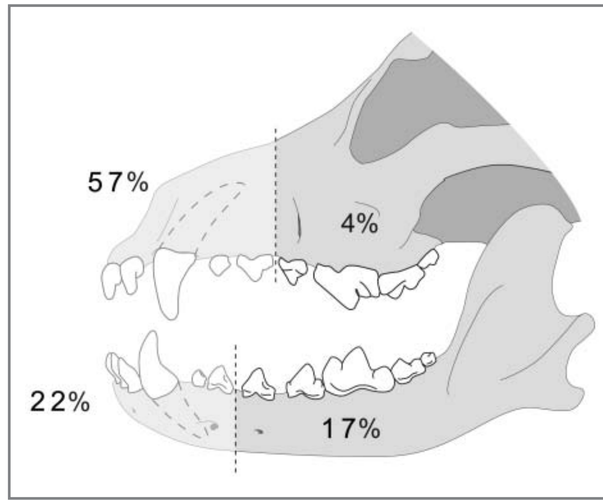

Figure 6—Distribution of FFH in the oral cavities of dogs (n = 24) evaluated for oral tumors of possible odontogenic origin at a veterinary teaching hospital from 1995 through 2005.

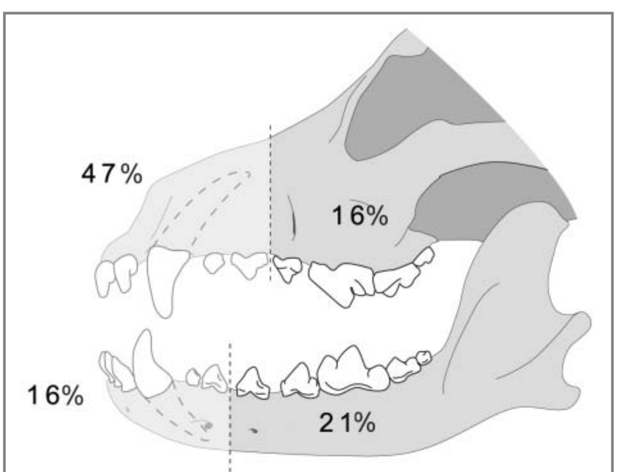

Figure 5—Distribution of POF in the oral cavities of dogs (n = 47) evaluated for oral tumors of possible odontogenic origin at a veterinary teaching hospital from 1995 through 2005.

with CAA differed significantly from that of the general hospital population, with Golden Retrievers (n = 13), Akitas (4), Cocker Spaniels (6), and Shetland Sheepdogs (5) overrepresented among the dogs with CAA.

POF—The mean age of 47 dogs with POF at first evaluation was 8.5 ± 2.9 years. The difference in the distribution of dogs by reproductive status when compared with that of the hospital population was significant (*P* = 0.013), with castrated males overrepresented and sexually intact females underrepresented (Table 1). The male-to-female ratio was 1.8:1. There was no significant correlation between age and reproductive status of dogs with POF.

Differences existed in the distribution of POFs within various regions of the oral cavity (*P* = 0.014), with the highest proportion present in the rostral aspect of the maxilla **(Figure 5)**. The ratio of maxillary to mandibular lesions was 1.7:1. There was no association between dog reproductive status and tumor location. Breed distribution in the dogs with POF did not differ significantly from that of the general hospital population; however, there were more Golden Retrievers among dogs with POF than the expected number for that breed (observed value, 6; expected value, 3).

FFH—The mean age of 24 dogs with FFH at first evaluation was 9.0 ± 3.0 years. When compared with the distribution of reproductive status among dogs in the general hospital population, castrated males were overrepresented and sexually intact males and females were underrepresented among dogs with FFH (*P* = 0.041). The ratio of males to females was 1:1. There was no significant correlation between dog age and reproductive status of dogs with FFH.

Differences existed in the distribution of FFH within various regions of the oral cavity (*P* = 0.003), with the highest proportion present in the rostral aspect of the maxilla **(Figure 6)**. There was no association between dog reproductive status and tumor location. There was also no specific breed predisposition; however, when purebred dogs and mixed-breed dogs were grouped separately, the proportion of mixed-breed dogs was higher than the expected value as determined from the proportion in the general hospital population.

Discussion

In the present study of dogs with suspected odontogenic tumors, CAA was the most common type of tumor identified (45% of lesions). This is contrary to findings of other similarly sized studies. For example, a study[6] found that 44% of 154 epulides were FFH, whereas another[22] found that 57% of 189 epulides were fibromatous, making FFH the most common type. The difference between those findings and ours may be a direct result of an evolved understanding of the histologic and clinical nature of these lesions since the earlier studies were performed. It is also likely a reflection of the types of lesions seen on a referral basis given the aggressive or destructive nature of some oral tumors (eg, CAA).

As previously mentioned, FFH is a reactive lesion, believed to result from irritation caused by plaque and calculus.[5,11] In our study, although FFH was the third most common lesion, it represented only 16% of lesions. Our lower number may have been a direct result of the increasing awareness of pet oral hygiene among veterinarians and owners. The second most common tumor type in our study was POF (31%), with various odontogenic tumors comprising the remaining 9% of lesions. Five of these remaining tumors were APOTs, previously known as calcifying epithelial odontogenic tumors.[14]

The mean age of dogs with CAA in our study was consistent with previously reported findings.[5,12,22–24] In one of the aforementioned studies,[22] fibromatous and ossifying epulides were treated as 2 entities rather than the currently accepted view that both are variations of the same tumor.[5,9,22] The associated findings are therefore difficult to interpret; however, it appears the ages of dogs with such types of epulides are similar to our findings. Dogs with FFH had a mean age that was marginally older than that of dogs with CAA or POF. The prevalence of periodontal disease in dogs increases with age[25]; therefore, it would not be surprising if the proportion of dogs with FFH did indeed increase with age. Regarding APOTs, another group of investigators found that the limited number of dogs with an APOT in their research ranged from 8 to 13 years.[5,14] Our findings indicated that APOT can also develop in much younger dogs, with ages ranging from 1 to 8 years.

The suggestion has been made that CAA is more common in female dogs than in males.[23,24] However, our findings indicated that males and females were equally represented, which is also true for humans with ameloblastoma.[1,26] On the other hand, sexually intact males and females in our study were underrepresented when compared with their distribution in the hospital population. A noteworthy difference in age at presentation exists between men and women with ameloblastoma (with men being older than women)[26]; however, no such finding was identified in our study.

When compared with the general hospital population in our study, castrated male dogs with POF were overrepresented, whereas sexually intact females were underrepresented. Indeed, the male-to-female ratio was 1.8:1. In another study,[22] the proportion of male dogs with fibromatous and ossifying epulides was greater than the proportion of female dogs; however, the reproductive status of those dogs was not indicated. Peripheral odontogenic fibroma is considered an uncommon tumor in humans, and a greater proportion of females than males are reportedly affected.[1,27]

With regard to FFH, castrated male dogs in the present study were overrepresented, compared with their distribution in the general hospital population, whereas sexually intact males and females were underrepresented. It is difficult to draw conclusions based on these findings given that FFH is a reactive and not a neoplastic lesion. In humans, FFH is common but does not appear to have a gender predilection.[16] Among the 5 study dogs with APOT, 4 were female, of which 3 were spayed. Thus, sexually intact females appeared to be least commonly affected by this type of neoplastic odontogenic lesion, which may suggest a hormonal role in the pathogenesis of these tumors.

Breed dispositions for development of odontogenic tumors were also investigated in the present study. Other reports[22,24] include lists of various dog breeds in which CAA was detected; however, no statistical analyses were performed to identify whether certain breeds were more susceptible than others. In our study, Golden Retrievers, Akitas, Cocker Spaniels, and Shetland Sheepdogs were overrepresented among dogs with CAA. On the other hand, no breeds were overrepresented among dogs with POF, although Golden Retrievers were more numerous than expected. The lack of a significant effect of breed may simply reflect limited statistical power due to the small number of dogs with POF. Focal fibrous hyperplasia was more common in mixed-breed dogs than in purebreds. Again, small numbers of dogs limited the power to detect whether brachycephalic breeds are more susceptible than other breeds, as has been suggested elsewhere.[11]

To evaluate whether certain locations in the oral cavity were more susceptible than others to tumor development, the oral cavity was divided into 4 anatomic regions on the basis of oncological surgical principles for mandibulectomies and maxillectomies.[28,29] By these principles, the rostral aspect of the maxilla and mandible are defined as extending from the first incisor to the level of the second premolar tooth. This is because an osteotomy between the canine tooth and the first premolar or between the first and second premolar teeth would transect the alveolus of the canine tooth. We defined the caudal aspect of the mandible and maxilla as the dentate region caudal to the second premolar tooth. A predilection of CAA for the rostral aspect of the mandible in dogs has been reported,[5,13,22,23] and our findings supported this supposition. The ratio of mandibular to maxillary lesions (2.3:1) suggested a vulnerability of the mandible in general to developing CAA. In humans, ameloblastoma also develops more commonly in the mandible (80% of all oral ameloblastomas vs 20%

in the maxilla); however, there is a marked predilection for the posterior region.[1,26] Given that CAA is the most common and also one of the most locally infiltrative odontogenic tumors, its predisposition for the rostral aspect of the mandible makes complete excision a realistic goal.

Peripheral odontogenic fibromas in our study were most common in the rostral aspect of the maxilla (47%) and the caudal aspect of the mandible (21%), with a maxillary-to-mandibular lesion ratio of 1.7:1. In humans, the mandibular premolar and the maxillary anterior regions are also the most commonly affected.[27] Furthermore, a similar maxillary-to-mandibular lesion ratio exists.[1] Focal fibrous hyperplasia does not appear to have a site predilection in the human oral cavity.[16] However, we identified a predilection of FFH for the rostral aspect of the maxilla (57%), whereas the caudal aspect of the maxilla was affected in only 4% of dogs with FFH. It is possible that the rostral aspect of the maxilla is more predisposed to periodontal disease than the caudal region; however, because the study was retrospective in nature, we were unable to ascertain whether this was true. It is also possible that the more rostral lesions were simply more easily noticeable during physical examination and that caudal lesions may have been missed. Interestingly, APOTs were most numerous in the mandible, particularly the rostral aspect. Previous reports provide little information with regard to location of this type of tumor in dogs.

As with any retrospective study, limitations existed in our study that could have affected the findings. The relative proportion of lesions in studies such as this one does not necessarily reflect the true distribution of lesions in the general canine population.[30] Indeed, the more aggressive types of tumors were probably overrepresented in our study because affected dogs were more likely to be referred by general practitioners for invasive surgical resection. Another limitation is the low number of odontogenic tumors in the canine population, making clinicopathologic conclusions difficult to draw. Regardless of these limitations, we were able to show that the 2 most common odontogenic tumors and FFH have distinct clinical manifestations of which veterinary clinicians and pathologists should be aware.

a. StatXact-8, Cytel Software Corp, Cambridge, Mass.

References

1. Barnes L, Eveson JW, Reichart P, et al. *World Health Organization classification of tumours—pathology and genetics of head and neck tumours.* Lyon, France: IARC Press, 2005.
2. Poulet FM, Valentine BA, Summers BA. A survey of epithelial odontogenic tumors and cysts in dogs and cats. *Vet Pathol* 1992;29:369–380.
3. Dubielzig RR, Goldschmidt MH, Brodey RS. The nomenclature of periodontal epulides in dogs. *Vet Pathol* 1979;16:209–214.
4. Gardner DG. An orderly approach to the study of odontogenic tumours in animals. *J Comp Pathol* 1992;107:427–438.
5. Gardner DG. Epulides in the dog: a review. *J Oral Pathol Med* 1996;25:32–37.
6. Verstraete FJM, Ligthelm AJ, Weber A. The histological nature of epulides in dogs. *J Comp Pathol* 1992;106:169–182.
7. Verstraete FJM. Oral pathology. In: Slatter DH, ed. *Textbook of small animal surgery*. 3rd ed. Philadelphia: WB Saunders Co, 2003;2638–2651.
8. Barker IK, Van Dreumel AA. The alimentary system. In: Jubb KVF, Kennedy PC, Palmer N, eds. *Pathology of domestic animals*. 3rd ed. Orlando, Fla: Academic Press Inc, 1985;1–237.
9. Gardner DG, Baker DC. Fibromatous epulis in dogs and peripheral odontogenic fibroma in human beings: two equivalent lesions. *Oral Surg Oral Med Oral Pathol* 1991;71:317–321.
10. Gardner DG. The peripheral odontogenic fibroma: an attempt at clarification. *Oral Surg Oral Med Oral Pathol* 1982;54:40–48.
11. Dubielzig RR. Proliferative dental and gingival diseases of dogs and cats. *J Am Anim Hosp Assoc* 1982;18:577–584.
12. Gardner DG, Baker DC. The relationship of the canine acanthomatous epulis to ameloblastoma. *J Comp Pathol* 1993;108:47–55.
13. Gardner DG. Canine acanthomatous epulis. The only common spontaneous ameloblastoma in animals. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1995;79:612–615.
14. Gardner DG, Dubielzig RR, McGee EV. The so-called calcifying epithelial odontogenic tumour in dogs and cats (amyloid-producing odontogenic tumour). *J Comp Pathol* 1994;111:221–230.
15. Head KW, Cullen JM, Dubielzig RR, et al. Histological classification of tumors of odontogenic origin of domestic animals. In: Head KW, Cullen JM, Dubielzig RR, et al, eds. *Histological classification of tumors of the alimentary system of domestic animals (WHO)*. Washington, DC: Armed Forces Institute of Pathology, 2007;46–57.
16. Regezi JA, Sciubba JJ, Jordan RC. Connective tissue lesions. In: Regezi JA, Sciubba JJ, Jordan RC, eds. *Oral pathology clinical pathological correlations*. 5th ed. St Louis: Saunders, 2008;155–177.
17. Breuer W, Geisel O, Linke RP, et al. Light microscopic, ultrastructural, and immunohistochemical examinations of two calcifying epithelial odontogenic tumors (CEOT) in a dog and a cat. *Vet Pathol* 1994;31:415–420.
18. Benlyazid A, Laxroix-Triki M, Aziza R, et al. Ameloblastic carcinoma of the maxilla: case report and review of the literature. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2007;104:e17–e24.
19. Gardner DG. Some current concepts on the pathology of ameloblastomas. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996;82:660–669.
20. Sun ZJ, Wu YR, Cheng N, et al. Desmoplastic ameloblastoma—a review. *Oral Oncol* 2009;45:752–759.
21. Mosqueda-Taylor A, Carlos-Bregni R, Ramírez-Amadora V, et al. Odontoameloblastoma. Clinico-pathologic study of three cases and a critical review of the literature. *Oral Oncol* 2002;38:800–805.
22. Yoshida K, Yanai T, Iwasaki T, et al. Clinicopathological study of canine oral epulides. *J Vet Med Sci* 1999;61:897–902.
23. Thrall DE. Orthovoltage radiotherapy of acanthomatous epulides in 39 dogs. *J Am Vet Med Assoc* 1984;184:826–829.
24. Langham RF, Mostosky UV, Shirmer RG. X-ray therapy of selected odontogenic neoplasms in the dog. *J Am Vet Med Assoc* 1977;170:820–822.
25. Kortegaard HE, Eriksen T, Baelum V. Periodontal disease in research beagle dogs—an epidemiological study. *J Small Anim Pract* 2008;49:610–616.
26. Reichart PA, Philipsen HP, Sonner S. Ameloblastoma: biological profile of 3677 cases. *Eur J Cancer B Oral Oncol* 1995;31:86–99.
27. Daley TD, Wysocki GP. Peripheral odontogenic fibroma. *Oral Surg Oral Med Oral Pathol* 1994;78:329–336.
28. Verstraete FJ. Mandibulectomy and maxillectomy. *Vet Clin North Am Small Anim Pract* 2005;35:1009–1039.
29. Salisbury SK. Maxillectomy and mandibulectomy. In: Slatter DH, ed. *Textbook of small animal surgery*. 3rd ed. Philadelphia: WB Saunders Co, 2003;561–572.
30. Bostock DE, White RAS. Classification and behaviour after surgery of canine 'epulides.' *J Comp Pathol* 1987;97:197–206.