



Abstract

The term “epulis” was first introduced in 1979 to describe common periodontal lesions in dogs. Suggested terms for common gingival lesions with similar histopathologic appearances (background stroma resembling the periodontal ligament) were acanthomatous epulis, fibromatous epulis, and ossifying epulis. However, the term “epulis” is nondescript and does not accurately characterize the biological behavior of those common odontogenic lesions. Specifically, epulis does not account for the fact that all 3 lesions are not truly benign and that they require vastly different treatments. Updated nomenclature has been implemented and should be adopted to allow for accurate communication between pathologists, clinicians, and clients. This article describes current nomenclature, clinical and radiographic characteristics, diagnosis, and treatment for canine acanthomatous ameloblastoma (previously called acanthomatous epulis), peripheral odontogenic fibroma (POF, previously called fibromatous epulis), and POF-ossifying type (previously called ossifying epulis). Closely related lesions, including other variants of ameloblastoma and focal fibrous hyperplasia, are also briefly described.



DENTISTRY

It Is Not Called an Epulis Anymore

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The term “epulis” stems from the Greek words “epi” and “oulon,” which mean “on the gum.” Epulis is broadly defined as any nonspecific gingival overgrowth. The term gained common acceptance in reference to canine odontogenic tumors in 1979.¹⁻³ In that historical publication, the authors proposed that 3 common gingival lesions be broadly categorized as “periodontal epulides” because they all share background stroma that resembles the periodontal ligament (PDL).³ Specifically, those 3 lesions were termed acanthomatous epulis, fibromatous epulis, and ossifying epulis based on distinct histopathologic features.²⁻³

EPULIS

Using the term “epulis” is now widely discouraged because it is nondescript and suggests that the lesion is benign. Even today, the phrase “it’s just an epulis” is used in daily practice. Yet a subset of such lesions, specifically acanthomatous epulis, can be highly invasive and must be treated. Therefore, odontogenic tumors have been reclassified on the basis of their cell of origin (i.e., epithelial, mesenchymal, or mixed), similar to the odontogenic tumor classification system used in humans. The most up-to-date classification for dogs and cats can be found in the *Histological Classification of Tumors of the Alimentary System of Domestic Animals*.⁴

Take-Home Points

- The term “epulis” does not convey the actual nature of the lesion or variety of recommended treatments.
- Acanthomatous epulis should be called canine acanthomatous ameloblastoma.
- Fibromatous epulis should be called peripheral odontogenic fibroma.
- Ameloblastoma and peripheral odontogenic fibroma are the most common odontogenic tumors in dogs.
- Odontogenic tumors are rare in cats.
- Ameloblastoma is invasive and requires aggressive treatment with surgery (to achieve a neoplasia-free margin) or radiation therapy.
- Peripheral odontogenic fibroma does not invade bone and can be treated solely by removing the gingival lesion in its entirety. If the client is concerned about potential local recurrence, or the lesion involves a large portion of gingiva, the tooth of origin should also be extracted.

Although proper nomenclature for these lesions has been controversial, the current body of literature supports canine acanthomatous ameloblastoma (CAA) as the preferred term for acanthomatous epulis, peripheral odontogenic fibroma (POF) as the preferred term for fibromatous epulis, and POF-ossifying type as the preferred term for ossifying epulis. This article describes the clinical and radiographic characteristics, diagnostic workup, and treatment for ameloblastoma, including variants other than CAA, as well as POF, including ossifying and nonossifying types.

AMELOBLASTOMA

Ameloblastoma is an epithelial odontogenic tumor that is common in dogs and rare in cats.⁵⁻⁷ It often presents as an erythematous, cauliflower-like gingival mass

closely associated with the teeth. However, if the lesion arises from an intraosseous (within the jaw) location, it may appear as a gingival swelling with bony expansion (FIGURE 1). According to the tumor's histopathologic features, it may be referred to as a CAA, solid/conventional ameloblastoma, or amyloid-producing ameloblastoma (TABLE 1, FIGURE 2).

CAA, which is by far the most common variant, is diagnosed by its acantholytic pattern on pathologic evaluation. In humans, there are also distinct biologic variants, which carry different prognostic and treatment implications. The presence of biologic variants has been suggested in dogs based on distinct clinical and diagnostic imaging features,⁸ yet that suggestion remains controversial. Currently, prognosis and treatment for all variants remain the same.

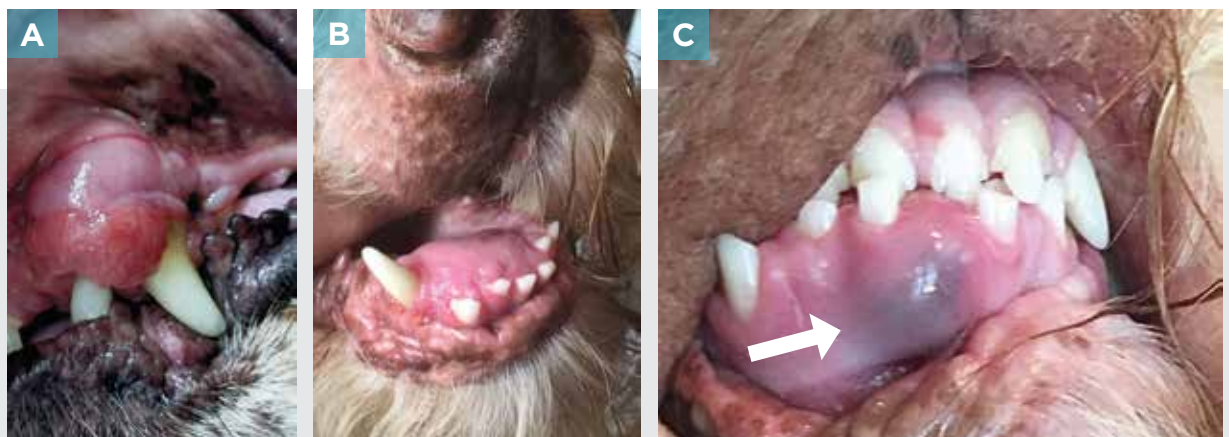


FIGURE 1. Variable clinical presentation of ameloblastoma. **(A)** Erythematous and proliferative cauliflower-like gingival mass, which is the classic appearance of ameloblastoma, especially canine acanthomatous ameloblastoma. **(B)** Gingival swelling and bony expansion from an intraosseous ameloblastoma. **(C)** A cystic component to this lesion, which often clinically appears blue (arrow).

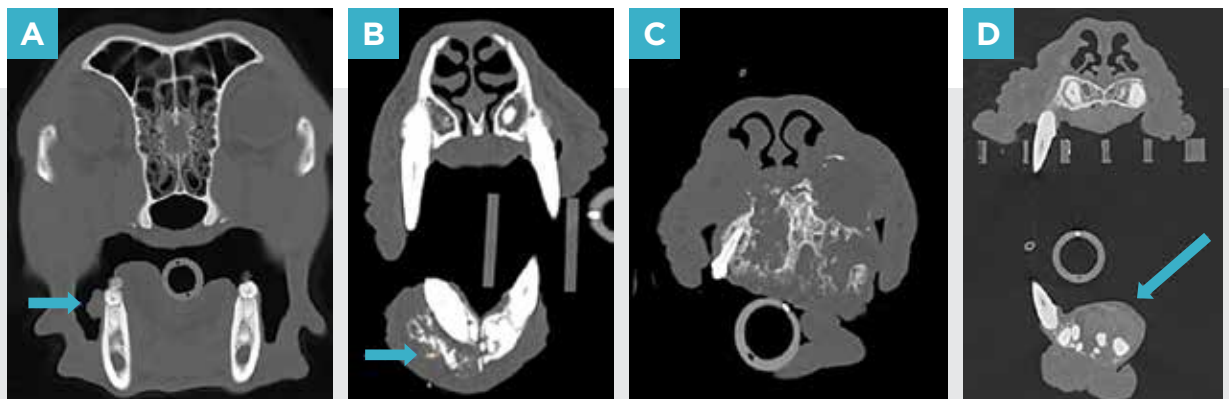


FIGURE 2. Variable diagnostic imaging (computed tomography) appearance of ameloblastoma. **(A-B)** Canine acanthomatous ameloblastoma (CAA) denoting the variability of bone lysis that can occur. **(A)** Extrasosseous CAA (arrow) with minimal to no bone lysis in the region of the soft tissue mass. **(B)** Intraosseous CAA (arrow) with severe bone lysis and expansion associated with the lesion. **(C)** Conventional ameloblastoma with classic multicentric cystic appearance. **(D)** Amyloid-producing ameloblastoma with unicystic lesion filled with amorphous material (arrow).



Of note, not all accept or use the term “amyloid-producing ameloblastoma”; rather, some refer to this specific variant as amyloid-producing odontogenic tumor (often called APOT).^{12,13} This type of tumor is relatively rare, and no large-scale clinicopathological studies have been performed in dogs or cats.

Similar to other odontogenic tumors, ameloblastomas have almost no metastatic potential. However, ameloblastomas can invade locally into the jawbone, especially when a lesion originates from an intraosseous location.^{10,11} The primary treatment recommendation for ameloblastomas is surgical intervention. Regardless of the variant, 10-mm surgical margins are recommended. As a result, ameloblastomas are most often treated with a maxillectomy or mandibulectomy, which usually results in long-term remission with very rarely reported (<5%) local recurrence.¹⁴

However, if the size of the patient or tumor preclude excision with a 10-mm surgical margin, then more conservative gross margins (5 mm) are probably acceptable. One paper revealed no local recurrence of CAA even after excision with narrow (<5 mm) and dirty pathologic margins.⁹ Thus, although removal of all abnormal bone is required to prevent local recurrence, a positive outcome may be achieved with only a very narrow margin of neoplastic-free bone around the tumor.¹⁴

Other reported treatment options for ameloblastomas include radiation and intralesional bleomycin. Ameloblastomas are radiosensitive, and long-term

control can be achieved with a definitive radiation protocol.¹⁵ Protocols vary among institutions, but the most common protocol is 3 weeks of daily radiation therapy. The author does *not* recommend use of intralesional bleomycin due to the lack of large prospective studies and safety concerns for staff and clients.¹⁶

For clients who decline definitive local treatment with surgery or radiation therapy for their pet, a discussion on quality of life and palliative care is recommended. After an ameloblastoma enters the cancellous bone of the jaw, it tends to expand rapidly, causing severe bone pain. Bone pain can be controlled with a palliative radiation protocol, bisphosphonates, and/or systemic analgesics. Clients should be advised as to expectations, and a monitoring plan to evaluate pain control should be established.

In the future, *BRAF* inhibitors, drugs that can potentially shrink or slow the growth of tumors that have a *BRAF* mutation, may also be an option for medical management of canine ameloblastomas. Recent oncogenesis literature reveals that 94% of CAAs show a mutation in the *HRAS* gene–signaling pathway, the same pathway that is most often mutated in human ameloblastomas.¹⁷ Mutations in this pathway can activate downstream pathways that promote increased tumor growth. In humans, results of initial drug trials using *BRAF* inhibitors for nonsurgical cases have been promising.¹⁸

TABLE 1 Clinical and Diagnostic Imaging Features of Ameloblastoma

TYPE OF AMELOBLASTOMA	AVERAGE AGE AT PRESENTATION	SEX PREDILECTION	BREED PREDILECTION	LOCATION PREDILECTION	DIAGNOSTIC IMAGING
Canine acanthomatous ameloblastoma ^{5,9-11}	9 years	None	Golden retrievers, Akitas, cocker spaniels, Shetland sheep dogs	Rostral mandible (51.2% reported in this location)	Originate from intraosseous or extraosseous location. Variable degree of bone lysis with intraosseous lesions exhibiting more aggressive behavior. Cysts are rare (approximately 11%).
Conventional ameloblastoma ⁸	9 years	None	None	Maxilla in canine/premolar region	95% intraosseous and cystic (often multilocular)
Amyloid-producing ameloblastoma ¹¹⁻¹³	—	—	—	—	Intraosseous and cystic (often unilocular with amorphous material within the cyst)



PERIPHERAL ODONTOGENIC FIBROMA

A POF is a mesenchymal odontogenic tumor, appearing as a smooth, broad-based gingival growth, similar to a focal fibrous hyperplasia (FFH) (**FIGURE 3**). Histopathologic evaluation is the only way to reliably differentiate a POF from an FFH. (Historically, the term “fibromatous epulis” was often used to clinically describe FFH as well.) FFH is a benign reactive lesion that occurs in response to inflammation or trauma, which distinguishes it from neoplastic-origin POF.⁵

Most commonly, POFs are in the anterior maxilla (near canine and incisor teeth) of middle-aged dogs at a mean age of 8 years. There is no known sex or breed predilection, although 1 study suggests that risk among boxers is higher.^{5,19} Overall, POFs are benign because they do not invade bone and do not metastasize. Because of their slow expansile growth, POFs may move teeth out of their normal position. They may also create bone, in which case they are called POF-ossifying type. On radiographs, an ossifying POF shows bone proliferation but no bone lysis (**FIGURE 4**). Clinically, both POF and POF-ossifying type are treated the same.

Although POFs do not invade bone, surgical treatment is still recommended because potential pseudopocketing around teeth predisposes the patient to periodontal disease. As the size of the mass increases, the patient may traumatize the lesion. Treatment is focused on removal of the entire lesion. Preventing recurrence does not require leaving a surgical margin of normal tissue. Historically, extracting the tooth of

origin has also been recommended because many believe the origin of a POF to be the PDL. Under this belief, tooth extraction is required to effectively remove the entire lesion. However, the true origin of POFs has not been definitively determined. Germinal cells in the gingiva, surface epithelium, alveolar bone, or PDL are speculative origins. In humans, POFs have been noted

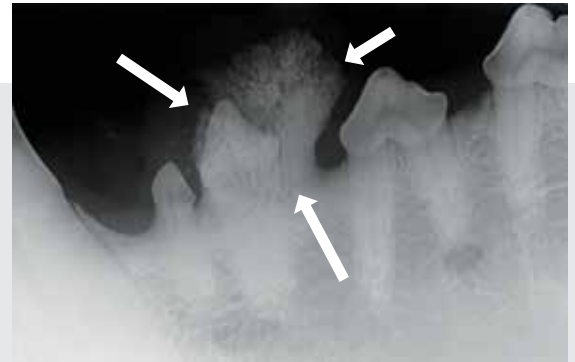


FIGURE 4. Radiographic appearance of an ossifying peripheral odontogenic fibroma at the left mandibular second premolar (306) (white arrows). Note the proliferative bone but lack of bone lysis.



FIGURE 3. Peripheral odontogenic fibroma centered at the left maxillary second premolar (206).



FIGURE 5. (A) Peripheral odontogenic fibroma on the distal aspect of the right mandibular canine (404). (B) When the lesion is entirely removed, it leaves a severe gingival defect on the distal aspect of the canine tooth. This tooth now requires either surgical extraction or an advanced periodontal procedure to replace the lost gingival tissue.



in edentulous regions, making gingiva the likely source of origin.²⁰ Consequently, a conservative approach with removal of the gingival lesion down to the periosteum, without tooth extraction, is probably appropriate.

However, if removal of a POF causes a large gingival defect, tooth extraction or another advanced periodontal procedure may be necessary (**FIGURE 5**). As a reminder, to be periodontally sound, all teeth require a minimum of 2 mm of gingiva. Furthermore, if the lesion is not completely removed, there is a risk for local recurrence. A thorough discussion with clients about risks associated with more conservative treatment without tooth extraction is essential. The exact risk for recurrence without tooth extraction has not been quantified; in the author's experience, it is rare. In addition, POFs have never been reported to malignantly transform (i.e., become a fibrosarcoma or malignant odontogenic tumor) with recurrence of the mass.

SUMMARY

Common odontogenic tumors were historically referred to as an “epulis.” However, this term is nondescript and is now highly discouraged. The most common “epulides” have been relabeled as ameloblastoma and POF. Ameloblastomas are invasive and should be treated with either surgery or radiation. POFs can cause local irritation and pseudopocketing but do not invade bone. Treatment includes removal of the entire gingival lesion; a surgical margin of normal tissue is not required. With treatment, odontogenic tumors carry an excellent prognosis. **TVP**

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