

Canine acanthomatous ameloblastoma in a golden retriever mix dog

Citra Yudeska, Palagan Senopati Sewoyo*

Department of Pathobiology, Faculty of Veterinary Medicine, Udayana University, Bali, Indonesia 80234.

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*Correspondence:

Corresponding author: Palagan Senopati Sewoyo
E-mail address: senopati.sewoyo@unud.ac.id

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ABSTRACT

Canine acanthomatous ameloblastoma (CAA) is a benign but locally invasive oral tumor that arises from the remnants of tooth-forming (odontogenic) epithelium within the submucosa and periodontal ligament. Although non-metastatic, CAA exhibits aggressive behaviour, particularly invading and destroying surrounding bone and dental structures. This case involved a 9-year-old female Golden Retriever mix, weighing 28.5 kg, presenting with a solid mass on the mandibular gingiva between canine tooth 304 and 404, approximately $2.5 \times 2 \times 1$ cm in size. The tumor had been growing for five months. Diagnostic imaging, including dental radiography, along with cytological and histopathological examinations, was performed to establish the diagnosis. Histopathology revealed a thickened mucosal layer, acanthosis, with the presence of ameloblastoma island. These findings were consistent with the characteristic of acanthomatous ameloblastoma. Rim excision was chosen to remove the tumor mass. Postoperative care included analgesia with tramadol and antibiotic therapy with enrofloxacin and metronidazole. The dog demonstrated a successful recovery, with normal clinical status observed within a week post-surgery. A one-year postoperative examination confirmed no signs of tumor recurrence.

Introduction

The oral cavity is a common site for the development of various tumors, both benign and malignant. In dogs, oral tumors account for approximately 6% of all tumor cases (Liptak, 2020). Despite their low prevalence rate, oral tumors can have a significant impact on a dog's quality of life. In general, symptoms that may arise due to the appearance of oral tumors in the early stages include decreased appetite, bleeding in the mouth, and swelling in the facial area (Tipirneni *et al.*, 2024). These tumors are classified as either odontogenic or non-odontogenic (Arzi and Verstraete, 2020). According to Wingo (2018), approximately 37% of oral tumors in dogs are malignant, while the remainder are benign. Among benign oral tumors, the most common types include acanthomatous ameloblastoma (43.22%), peripheral odontogenic fibroma (31.31%), plasma cell tumor (8.47%), histiocytoma (5.08%), fibroma and osteoma (2.54%), granulosa cell tumor, hemangioma, and schwannoma (1.69%), as well as lymphangioma and papilloma (0.85%) (Saththathum *et al.*, 2023).

Canine acanthomatous ameloblastoma (CAA), the most frequently diagnosed benign oral tumor in dogs, was formerly known as acanthomatous epulis or adamantinoma. It originates from remnants of tooth-forming (odontogenic) epithelium within the submucosa and periodontal ligament and exhibits a slow growth rate. However, CAA is locally aggressive, often invading the mandible or maxilla (Fiani *et al.*, 2011; Palić *et al.*, 2022). Despite its invasive nature, it does not metastasize (Malmberg *et al.*, 2017). The most common sites of occurrence are the rostral mandible, followed by the caudal mandible, rostral maxilla, and caudal maxilla (Goldschmidt *et al.*, 2017).

Diagnosis of CAA is typically confirmed through radiographic and histopathological examination (Malmberg *et al.*, 2017). Histopathological findings characteristic of CAA includes islands of proliferating odontogenic cells within the gingival mucosa, bordered by ameloblastic cells (Das *et al.*, 2013). Various treatment options for CAA have been reported, including radiation therapy (Feest, 2017), intralesional chemotherapy with bleomycin (Kelly *et al.*, 2010), systemic chemotherapy (Stancu *et al.*, 2012), cryosurgery (Guimarães *et al.*, 2013), radical surgical excision

(Goldschmidt *et al.*, 2017; Malmberg *et al.*, 2017), rim excision (Murray *et al.*, 2010), and maxillectomy or mandibulectomy in cases with bone invasion (Malmberg *et al.*, 2017). This case report described CAA in a golden retriever mix dog, the clinicopathological aspect and its management.

Materials and methods

Clinical presentation

A 9-year-old female golden retriever mix, weighing 28.5 kg, was brought to the clinic by its owner due to a firm, marble-like mass on the lower gingiva, which had progressively enlarged over the past five months. Prior to this visit, the dog had not received any treatment or medication. The owner reported no signs of decreased appetite or weight loss. The dog's defecation and urination remained normal.

Clinical examination of the oral cavity revealed a solid, red, marble-like mass, and slightly ulcerated approximately $2.5 \times 2 \times 1$ cm in size, located on the rostral mandibular gingiva, positioned between lower canine teeth (Fig. 1). No other abnormalities were detected upon general clinical assessment. The respiration, heart rate, capillary refill time, and rectal temperature were in normal range. A fine-needle aspiration (FNA) biopsy was performed, and cytological evaluation revealed a collection of red blood cells and inflammatory cells (Fig. 2).



Fig. 1. (a) Marble-like mass on the mandibular gingiva (b) The mass located between canine tooth 304 and 404 on the rostral mandibular gingiva.

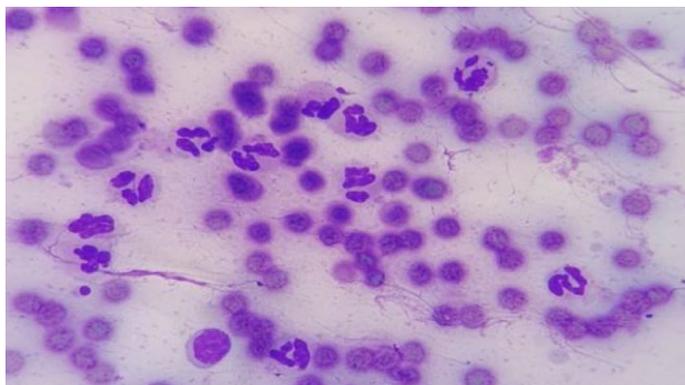


Fig. 2. Fine needle aspiration cytology of the mass revealing neutrophilic and lymphocytic inflammatory cells (Diff-Quik stain, 1000 \times magnification).

Hematological and blood biochemistry examination

Hematology and blood biochemistry analyses were conducted to assess the dog's overall health status. The blood was collected from the cephalic vein and then placed into two types of tubes: EDTA tubes and plain tubes without anti-coagulant. The first tube was directly assessed for hematology using hematology analyzer (Vetscan HM5, Zoetis), while the second tube was kept until the serum became visible. Once the serum was visible, it was collected and analyzed using veterinary chemistry analyzer (CatalystOne, IDEXX) to assess the blood biochemistry profile (Catalyst Chem 17 CLIP, IDEXX). The analysis was carried out twice: during the first visit and the second visit prior to surgery.

Dental Radiography

A dental radiographic examination was performed to assess the extent and demarcation of the mass. The animal was positioned in dorsal recumbency with the head extended.

Histopathology Examination

Since cytological evaluation alone is often insufficient for a definitive diagnosis and given the mass characteristics suggestive of a tumor, a histopathological examination was performed for further confirmation. A biopsy of the mass was performed, and the tissue sample was fixed in 10% neutral-buffered formalin at a 1:10 ratio. The sample was subsequently processed for histological examination and stained with hematoxylin and

eosin (HE). It was then submitted to a veterinary pathologist for diagnostic confirmation.

Results

The initial hematology results revealed leukocytosis, lymphocytosis, neutrophilia, and microcytic normochromic anemia (Table 1). Blood biochemistry analysis indicated an elevated lipase level (Table 2).

The radiological imaging revealed a well-defined, round mass attached to the gingiva between the mandibular first, second, and third incisors, leading to tooth displacement. The mass exhibited radiopaque characteristics (Fig. 3).

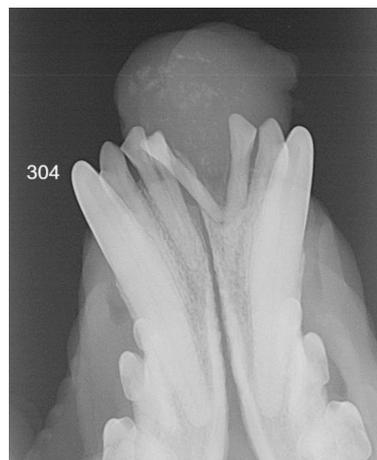


Fig. 3. Dental radiograph in the mandibular occlusal view showing the presence of a tumor mass and displacement of the first, second, and third incisors.

Histopathological analysis revealed a thickened mucosal layer with hyperplasia of epithelial cells in the stratum spinosum. The epithelium exhibited a connecting layer of non-keratinized odontogenic cells with peripheral palisading and prominent acanthocyte formation derived from the stellate reticulum. The mesenchymal tissue of the periodontal ligament was occasionally indistinct, with stellate fibroblasts situated behind dense fibrillar collagen in an organized arrangement, accompanied by neovascularization. Scattered neutrophilic infiltration was observed in small numbers (Fig. 4).

Based on the histopathological findings, the mass was diagnosed as CAA. Given this diagnosis, surgical intervention was selected as the preferred treatment. Prior to surgery, the animal's condition was stabi-

Table 1. Complete blood count (CBC) examination results.

| Parameter | First examination | Second examination | Normal range |
|--|-------------------|--------------------|--------------|
| WBC ($\times 10^3/\mu\text{L}$) | 22.67** | 10.4 | 6-17 |
| Lymphocyte ($\times 10^3/\mu\text{L}$) | 1.78** | 1.4 | 1-4.8 |
| Monocyte ($\times 10^3/\mu\text{L}$) | 1.17 | 0.42 | 0.2-1.5 |
| Neutrophil ($\times 10^3/\mu\text{L}$) | 19.30** | 8.49 | 3-12 |
| Eosinophil ($\times 10^3/\mu\text{L}$) | 0.29 | 0.07 | 0-0.8 |
| Basophil ($\times 10^3/\mu\text{L}$) | 0.13 | 0.02 | 0.0-0.40 |
| RBC ($\times 10^6/\mu\text{L}$) | 7.85 | 7.58 | 5.5-8.5 |
| Hb (g/dL) | 10.4* | 17.5 | 12-18 |
| HCT (%) | 32.54* | 51.53 | 37-55 |
| MCV (fL) | 41* | 68 | 60-77 |
| MCH (pg) | 13.3* | 23.1 | 19.5-24.5 |
| MCHC g/dL | 32 | 34 | 31-39 |
| Platelet ($\times 10^3/\mu\text{L}$) | 365 | 241 | 165-500 |
| MPV (fL) | 10.2 | 10.7 | 3.9-11.1 |

Note: *Below normal range **Above normal range. WBC=White blood cell; RBC=Red blood cell; Hb=Hemoglobin; HCT=Hematocrit; MCV=Mean Corpuscular Volume; MCH=Mean Corpuscular Hemoglobin; MCHC=Mean Corpuscular Hemoglobin Concentration; MPV=Mean Platelet Volume. Normal range according to veterinary hematology analyzer manufacturer (Vetscan HM5, Zoetis). First CBC performed during first visit. Second CBC performed prior to surgery after stabilization.

lized, as the initial hematological evaluation (Table 1) indicated suboptimal parameters. The preoperative treatment regimen included amoxicillin-clavulanate (18 mg/kg BW, PO, b.i.d.) and prednisolone (0.35 mg/kg BW, PO, q.d.) for 10 days. A follow-up hematological assessment post-therapy confirmed normalization of all parameters, indicating the animal was stable for surgery.

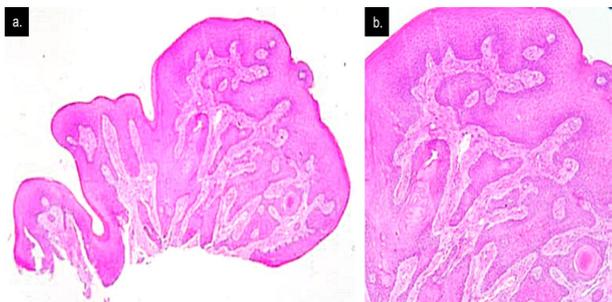


Fig. 4. Photomicrographs of a histologic section of the gingival mass in a golden retriever-mix dog. (a) Marked thickening of the stratum spinosum, characterized by acanthosis, with the presence of ameloblastic epithelium islands. (b) Non-keratinized odontogenic epithelium exhibiting a palisading arrangement of acanthocytes and ameloblast cells, with subepithelial tissue displaying nuclear variation (HE, 100x & 400x).

The chosen surgical approach was rim excision, performed under iso-flurane inhalation anesthesia. The procedure involved tumor excision with a 1 cm margin of surrounding tissue and extraction of the mandibular incisor teeth (Fig. 5). Postoperative management included tramadol (1.8 mg/kg BW, PO, q.d.) for analgesia over two days, along with antibiotic therapy consisting of enrofloxacin (5.5 mg/kg BW, PO, q.d. for eight days) and metronidazole (28 mg/kg BW, PO, b.i.d. for seven days). One-week post-surgery, the surgical site had healed well, and the dog exhibited normal activity levels with a good appetite and drinking behavior. A one-year follow-up revealed no evidence of tumor recurrence.

Discussion

Although CAA is classified as a benign tumor, it exhibits locally aggressive behavior, frequently invading the alveolar bone and carrying a risk of recurrence following surgical excision (Palić *et al.*, 2022). To date, this tumor has only been reported in dogs (Fiani and Peralta, 2019; Fer-

reira *et al.*, 2024). CAA typically arises in the rostral mandible (Bolek *et al.*, 2024), a location often associated with extensive bone destruction and osteolysis (Goldschmidt *et al.*, 2020). However, in this case, no radiographic evidence of bone destruction was observed. The incidence of CAA in the mandible is reported to be 70–72% compared to the maxilla (Fiani *et al.*, 2011; Goldschmidt *et al.*, 2017).

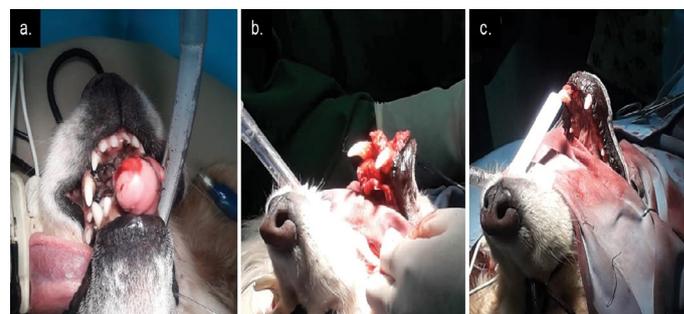


Fig. 5. Surgical procedure for CAA mass (a) The dog positioned dorsal recumbency. (b) Suturing process following the rim excision procedure. (c) Final postoperative outcome after suturing.

In human ameloblastoma, tumorigenesis is associated with mutations in the BRAF, HRAS, NRAS, and KRAS genes, leading to activation of the MAPK signaling pathway. A study by Peralta *et al.* (2019) identified similarities in mutation patterns between human and canine ameloblastomas, reporting that 94% of CAA cases harbored somatic HRAS p.Q61R mutations, while no oncogenic mutations were detected in BRAF, KRAS, NRAS, FGFR2, or SMO genes. In contrast, a study by Saffari *et al.* (2019) reported HRAS mutations in 63% of cases, along with a lower frequency of BRAF mutations (8%). Dysregulation of RAS signaling is known to activate the epithelial-to-mesenchymal transition (EMT) program, a key mechanism underlying the invasive potential of CAA (Peralta *et al.*, 2021).

Surgical resection with a 1–2 cm margin is recommended to minimize local recurrence. Previous studies have reported that surgical excision results in a recurrence rate of less than 5% (Kosovsky *et al.*, 1991; Murray *et al.*, 2010; Goldschmidt *et al.*, 2017; Sarowitz *et al.*, 2017). Thus, surgery remains the primary treatment of choice for CAA (Fiani and Peralta, 2019; Tsugawa *et al.*, 2022). A breed predisposition has been suggested, as CAA

Table 2. Blood biochemistry examination results.

| Parameter | First examination | Second examination | Normal range |
|-------------------------|-------------------|--------------------|--------------|
| Glucose (mg/dL) | 102 | 103 | 74-148 |
| Creatinine (mg/dL) | 0.9 | 1 | 0.5-1.8 |
| BUN (mg/dL) | 14 | 13 | 7-27 |
| BUN/Creatinine | 15 | 13 | |
| Phosphor (mg/dL) | 5 | 4.2 | 2.5-6.8 |
| Calcium (mg/dL) | 10.1 | 9.9 | 7.9-12.0 |
| Total protein (g/dL) | 7.1 | 6.9 | 4.8-7.2 |
| Albumin (g/dL) | 3.3 | 3.2 | 5.2-8.2 |
| Globulin (g/dL) | 3.7 | 3.7 | 2.5-4.5 |
| Albumin/Globulin | 0.9 | 0.9 | |
| ALT (U/L) | 64 | 58 | 10-125 |
| ALP (U/L) | 128 | 139 | 23-212 |
| GGT (U/L) | 0 | 0 | 0-11 |
| Total bilirubin (mg/dL) | 0.2 | 0.3 | 0.0-0.9 |
| Cholesterol (mg/dL) | 207 | 200 | 110-320 |
| Amylase (U/L) | 1046 | 820 | 500-1500 |
| Lipase (U/L) | 5464** | 4968** | 200-1800 |

Note: *Below normal range **Above normal range. ALT=Alanine Transaminase; ALP=Alkaline Phosphatase; BUN=Blood Urea Nitrogen; GGT=Gamma-Glutamyl Transpeptidase. Normal range according to veterinary chemistry analyzer manufacturer (CatalystOne, IDEXX). First CBC performed during first visit. Second CBC performed prior to surgery after stabilization.

occurs more frequently in Shetland Sheepdogs, Cocker Spaniels, Akitas, and Golden Retrievers, though no gender predisposition has been reported (Sampaio *et al.*, 2025). In this case, a 1 cm excision margin was utilized, which was confirmed to be effective, as no tumor recurrence was observed at the one-year follow-up.

Histopathological findings in this case were consistent with previous reports, reinforcing the role of histopathology as the definitive diagnostic method for CAA (Ferreira *et al.*, 2024), as cytology alone may be insufficient for definitive diagnosis, as seen by our FNA findings that did not lead to CAA. The hallmark histopathologic features of CAA cannot be observed on cytology due to lack of architecture (Palić *et al.*, 2022). Additionally, radiographic examinations are useful for assessing the extent of tumor invasion (Arslan *et al.*, 2024). Histomorphological variations can sometimes complicate diagnosis, as CAA closely resembles fibromatous epulis of periodontal ligament origin (Kamdi *et al.*, 2024). Furthermore, CAA and oral squamous cell carcinoma (OSCC) share similar clinical, radiological, and histopathological features (Soukup *et al.*, 2013). In cases where differentiation from OSCC is challenging, RAS Q61R immunohistochemical staining can aid in diagnosis (Peralta *et al.*, 2023). Additionally, CAA can be differentiated from other oral tumors using cytokeratin and vimentin immunohistochemically, as CAA typically exhibits negative staining for both markers. While histopathological examination may reveal a high degree of cellular atypia, including pronounced pleomorphism and frequent mitotic figures, these features do not correlate with metastatic potential or a poor prognosis (Malmberg *et al.*, 2017).

Hematological analysis in this case revealed leukocytosis, likely due to a secondary bacterial infection on the tumor surface. Antibiotic therapy effectively reduced the infection, as evidenced by the normalization of hematological parameters following treatment. Elevated lipase levels were also noted but were not associated with CAA. Increased lipase levels can indicate pancreatitis, gastrointestinal inflammation, renal dysfunction, or urinary tract obstruction. However, as no clinical signs of these conditions were observed and clinical examination findings were unremarkable, the elevation was considered clinically insignificant.

Conclusion

Examination of the oral gingival mass confirmed a diagnosis of canine acanthomatous ameloblastoma. This case highlights the importance of early detection and surgical management of CAA. Rim excision with a 1 cm margin was effective in preventing recurrence, supporting its use as a primary treatment modality.

Conflict of interest

The authors have no conflict of interest to declare.

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