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A Case of Acanthomatous Ameloblastoma in a Anatolian Shepherd (Kangal) Dog

Bir Anadolu Çoban Köpeğinde (Kangal) Akantomatöz Ameloblastoma Olgusu

Taner ARSLAN[1*]
Mümin Gökhan ŞENOCAK[1]
Esra MODOĞLU[1]
Büşra BAYKAL[1]
Yasemin AKÇORA[1]
Çağlar ÖZKALIPÇI[1]
Selim ÇOMAKLI[2]

[1] Atatük University, Faculty of Veterinary Medicine, Department of Surgery, Erzurum, TÜRKİYE.
[2] Atatük University, Faculty of Veterinary Medicine, Department of Pathology, Erzurum, TÜRKİYE.

ABSTRACT

Canine acanthomatous ameloblastoma (CAA) is a benign but locally invasive tumor originating from odontogenic epithelium and is one of the most common oral neoplasms in dogs. This report presents a case of CAA in a 6-year-old male Anatolian shepherd (Kangal) dog, which exhibited progressive growth in the rostral region of the maxilla, accompanied by symptoms such as bleeding gums and loss of appetite. Following physical examination, radiographic evaluations, and histopathological analyses, a diagnosis of CAA was made, and lesions were excised radically. Postoperatively, the dog was treated with amoxicillin-clavulanic acid, 8.75-25 mg/kg subcutan, 0.12% oral antiseptic spray, 0.04 mg/kg subcutan meloxicam and 1.0 mg/kg per os famotidine. No signs of malignancy were found in the samples taken from around the oral masses. A follow-up examination on the 70th day after surgery showed no metastasis or recurrence. In conclusion, this case report emphasizes that CAA can also be seen in Anatolian shepherd (Kangal) dogs. Additionally, radical surgery is an effective treatment option and excision of soft tissues as well as bone tissue may reduce the risk of midterm recurrence.

Keywords: Maxilla, Odontogenic tumors, Oral, Surgical excision.

ÖZ

Köpeklerde akantomatöz ameloblastoma (CAA), odontojenik epitelden kaynaklanan, iyi huylu ancak lokal olarak invaziv bir tümördür ve köpeklerde en yaygın oral neoplazilerden biridir. Bu rapor, 6 yaşında erkek bir Anadolu çoban köpeğinde (Kangal) görülen CAA vakasını sunmaktadır. Maksillanın rostral bölgesinde ilerleyici büyüme gösteren bu tümör, diş etlerinde kanama ve iştahsızlık gibi semptomlarla birlikte ortaya çıkmıştır. Fiziksel muayene, radyografik değerlendirmeler ve histopatolojik analizler sonrasında CAA tanısı konulmuş ve lezyonlar radikal olarak çıkarılmıştır. Ameliyat sonrası dönemde köpeğe amoksisilin-klavulanik asit (8.75-25 mg/kg subkutan), %0.12'lik oral antiseptik sprey, meloksikam (0.04 mg/kg subkutan) ve famotidin (1.0 mg/kg oral) uygulanmıştır. Oral kitlelerin çevresinden alınan örneklerde malignite bulgusuna rastlanmamıştır. Ameliyattan sonraki 70. gün yapılan takip muayenesinde metastaz veya nüks izlenmemiştir. Sonuç olarak, bu olgu sunumu, CAA'nın Anadolu çoban köpeklerinde (Kangal) görülebileceğini vurgulamaktadır. Ayrıca, radikal cerrahinin etkili bir tedavi seçeneği olduğu ve yumuşak dokuların yanı sıra kemik dokusunun da çıkarılmasının, orta vadede nüks riskini azaltabileceği belirtilmektedir.

Anahtar Kelimeler: Maksilla, Odontojenik tümörler, Oral, Cerrahi eksizyon.

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Sorumlu Yazar/Corresponding author: Taner ARSLAN
E-mail: arslantaner2000@gmail.com
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INTRODUCTION

Canine acanthomatous ameloblastoma (CAA) is one of the most prevalent odontogenic neoplasms in dogs. It is a benign but locally invasive tumor originating from the odontogenic epithelium. CAA accounts for approximately 6% of all known oral tumors and 45% of all odontogenic tumors in dogs.[1] Tumors arising from odontogenic epithelial cells are classified as "ameloblastomas." The term "acanthomatous" refers to the spine-like morphology of the epithelial cells within these tumors.[2] CAA predominantly occurs in the gingival region and has the potential to invade surrounding tissues, especially the jawbone. Although it typically presents as a painless, slow-growing mass, its invasive nature necessitates surgical intervention.[3]

Histopathological analysis remains the definitive method for diagnosing CAA, with the formation of cord-like structures by ameloblastic epithelial cells serving as a hallmark feature. Ameloblastomas arise from the epithelial remnants of the gingiva, also known as Serres remnants, which manifest as irregular, exophytic gingival masses on either side of the dental arch. These tumors grow slowly and frequently display a cystic structure that destroys the alveolar bone. Ameloblastomas in humans rarely metastasize. Similarly, no cases of distant metastasis or regional lymph node involvement have been reported in dogs.[4]

CAA primarily affects dogs six years or older and is more frequently observed in brachycephalic breeds such as Boxers, Pekingese, Pugs, and Shih Tzus.[5] However, there have been no reports to date describe CAA in Anatolian Shepherd (Kangal) dogs. Diagnostic imaging, including X-ray, computed tomography (CT), or magnetic resonance imaging (MRI), is recommended before treatment to precisely assess the tumor's extent. MRI is particularly effective in delineating tumor margins compared to CT images.[5] Radical surgical resections, such as maxillectomy or mandibulectomy, is the treatment of choice for CAA. While intralesional antineoplastic drugs and radiation therapy have also demonstrated success, broad surgical excision remains the most effective treatment, especially in cases requiring long-term remission.[2]

CASE PRESENTATION

A 6-year-old, 49-kg male Anatolian Shepherd (Kangal) dog presented to the Atatürk University Faculty of Veterinary Medicine Animal Hospital with a progressive oral mass, accompanied by halitosis, gingival bleeding, and loss of appetite. Based on the owner's account, the mass had been present for approximately six months, exhibiting progressive enlargement and leading to difficulties in oral intake. Additionally, gingival bleeding was reported to occur following meals.

Physical examination revealed three firm, pink, exophytic masses with focal ulcerative regions in the rostral maxilla on both the right and left sides. The masses were palpably firm and adhered to underlying structures (Figures 1A-B). Notably, the owner reported a history of transmissible venereal tumor (TVT) in the same dog three years prior, which had been successfully treated with vincristine, leaving no residual lesions.

Radiological evaluation was performed to determine the invasion of the mass.

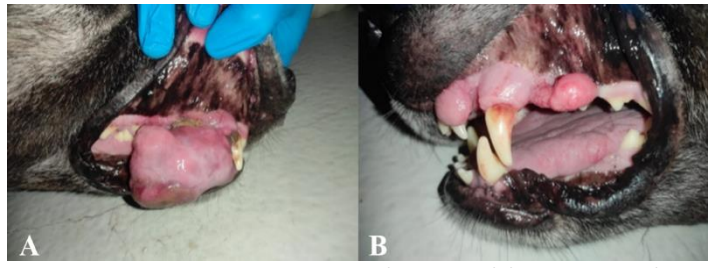

Figure 1. A. Preoperative view: Gingival mass caudal to canine tooth 104 in the oral cavity.
B. Preoperative view: Gingival mass in the oral cavity around tooth 203-205.

Radiography

After sedation with medetomidine 100 µg/kg intramuscular (Domitor, Zoetis, USA) and propofol 2 mg/kg intravenoz (Propofol %2 Fresenius Kabi, Germany) orthogonal radiographs of the maxilla were taken. Radiographic findings revealed masses extending from the distal aspect of tooth 104 to the buccal surface of tooth 108, with radiopaque and radiolucent areas around teeth 203 and 205. Canine tooth 104 was buccally displaced due to the tumor mass (Figures 2A-B).

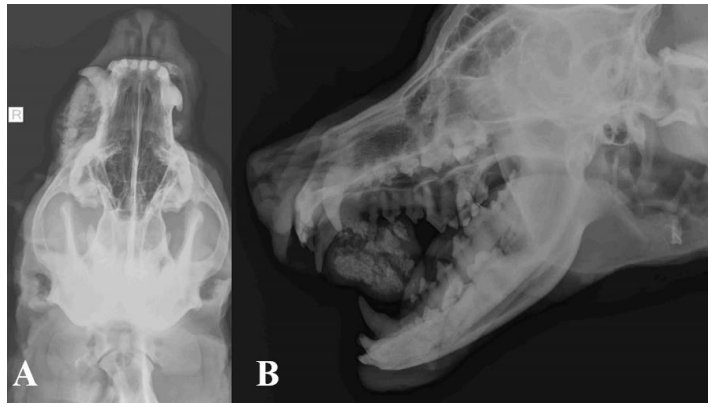

Figure 2. A. Preoperative radiographic view in ventro-dorsal direction.
B. Preoperative radiographic view in the latero-lateral direction.

Surgery

The dog was fasted for 12 hours prior to induction of anesthesia. The patient was sedated with medetomidine 100 µg/kg intramuscular (Domitor, Zoetis, USA) and induced with propofol. Infraorbital nerve block was performed with 2.5 mg/kg bupivacaine (Buvicaine, Polifarma, Turkey).[6] Oxygenation was provided using an endotracheal tube during total intravenous anesthesia (TIVA) of propofol-ketamine combinations. Ketamine 4 mg/kg (Ketasol, Interhas, Richter Pharma AG) and propofol 4 mg/kg (Propofol 2% Fresenius Kabi, Germany) were combined in a 1:2 ratio, to create a solution known as ketofol. This was administered intravenously as a bolus via the cephalic vein at a rate of 0.2 ml/kg/min. The administration continued until jaw relaxation was observed. To maintain anesthesia, additional top-ups of the same volume were given when heart rate, mean arterial pressure, and respiratory rate increased by more than 20% from baseline, with each top-up administered over 120 seconds.[7] Using the help of an oral gag and a cautery device, the masses were excised with 1 cm of healthy tissue margins. The maxillary rim was reduced using rongeurs to remove the affected bone tissue. The patient's vital parameters were monitored with a veterinary monitor (ePM 12M Vet, MINDRAY, China) during the entire procedure. Hemostasis was achieved using a monopolar electrosurgical device (EK-160, Üzümcü, Ankara, Turkey) with a power setting of 30-50 watts for cutting and 25-40 watts for coagulation. Three excised masses measured 8×5×3 cm, 4×2×2 cm, and 2×1×1 cm, respectively (Figure 3A-C). The gingiva and underlying tissues were sutured with simple interrupted sutures (Figure 4A-B).

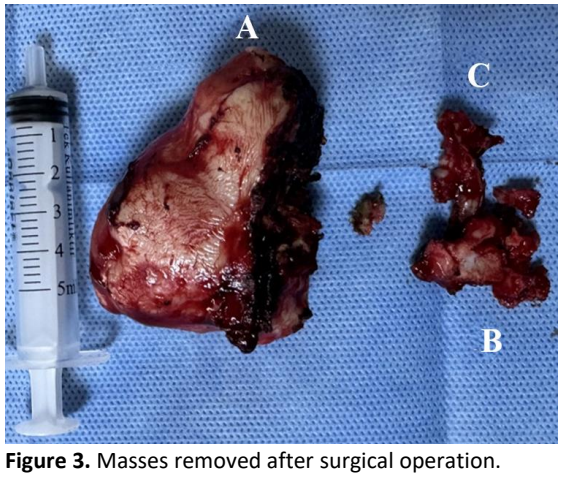

Figure 3. Masses removed after surgical operation.

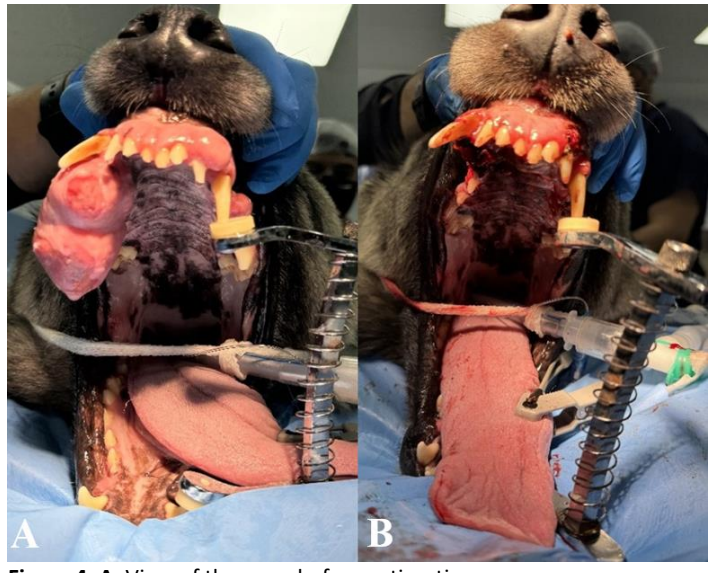

Figure 4. A. View of the mass before extirpation.
B. Jaw appearance after surgical excision of the mass.

Histopathology

The sections were placed in an oven at 57°C for one hour to thaw the paraffin. Deparaffinization was then performed in xylene I and II solutions. The sections were treated in decreasing concentrations of alcohol (100%, 100%, 96%, 90%, 70%, 50%; 3 minutes in each) and then placed in distilled water for 5 minutes for rehydration. After application of hematoxylin stain, sections were rinsed with tap water and stained in eosin solution for 15 seconds. They were then passed through increasing concentrations of alcohol before being transferred to xylene. Sections were coverslipped with Entellan and examined under a light microscope (Leica DM750, Flexicam i5). Histopathological analysis revealed characteristic features of CAA, including acanthosis, formation of ameloblastomatous islands, and uneven epithelial layering. Palisade cells in the stratum basale showed larger nucleoli, nuclear polymorphism, and an increased nuclear-to-cytoplasmic ratio. Additional findings included osseous island formation and increased vascularization (Figures 5A-D).

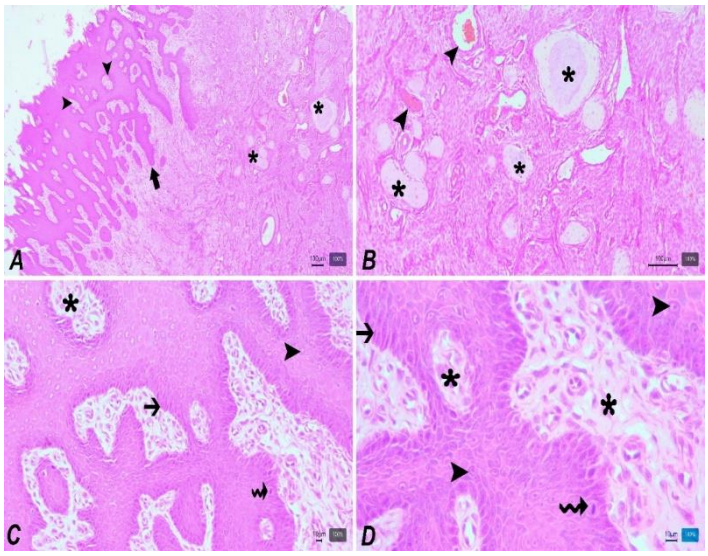

Figure 5. Canine acanthomatous ameloblastoma. **A:** Ameloblastomatous islets (arrowhead), acanthosis (thick arrow), and osseous islets in the dermis (asterisk), Magnification: 4X. **B:** Closer view of osseous islets (asterisk) and vascularisation (arrowhead), Magnification: 10X. **C:** Ameloblastomatous islets (asterisk), prickle cells (thin arrow), and mitotic figure (zigzag arrow), Magnification: 20X. **D:** Ameloblastomatous islets (asterisk), increased nuclear-to-cytoplasmic ratio (arrowhead), prickle cells (thin arrow) and mitotic figure (zigzag arrow), Magnification: 40 X. H&E.

Postoperative management

Postoperative treatment consisted of 0.12% oral chlorhexidine spray (Geraks, Kim-Pa, Istanbul, Turkey) for 15 days, 0.04 mg/kg subcutaneous meloxicam (Bavet Meloxicam, Istanbul, Turkey) for 5 days, prophylactic amoxicillin-clavulanic acid 8.75-25 mg/kg subcutaneously (Synulox, Zoetis, Istanbul, Turkey) for 7 days and famotidine 1.0 mg/kg per os (Sandoz, Istanbul, Turkey) for 5 days. An Elizabethan collar (35 cm, Pawise, İzmir, Turkey) was encircled the neck to prevent self-trauma. At 15-day intervals, the patient owner was contacted by phone to collect information about patient. In the postoperative period, antineoplastic drugs were not used because the patient owner was rejected the chemotherapy. On the 70th postoperative day, the owner reported a recovery with no signs of recurrence or metastasis (Figures 6).

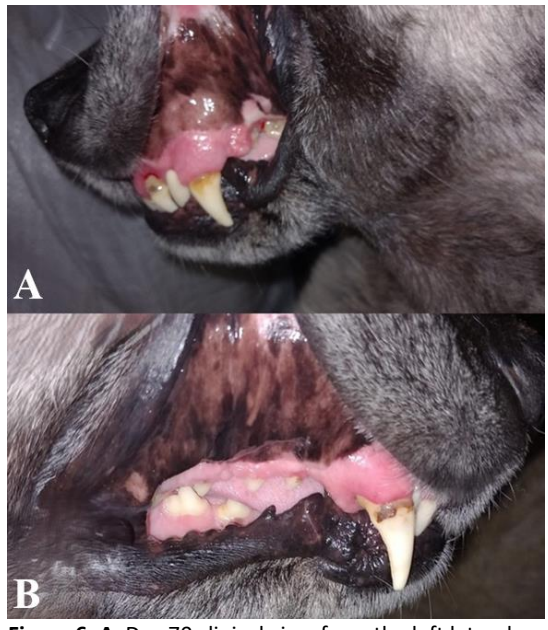

Figure 6. A. Day 70 clinical view from the left lateral aspect **B.** Day 70 clinical view from the right lateral aspect.

DISCUSSION

Canine acanthomatous ameloblastoma (CAA) is a well-defined, benign oral tumor in dogs, previously referred to as acanthomatous epulis. While this pathology has been well-documented in brachycephalic breeds[5], this represents the first reported case of CAA in an Anatolian Shepherd (Kangal) dog. Furthermore, although alternative treatments, such as intralesional bleomycin injections and radiotherapy have been reported in the literature for CAA[8], the current case demonstrates that, in the absence of chemotherapy or radiotherapy, post-surgical recurrence may not be observed even 70 days past over the case.

CAA arises from the odontogenic epithelium and is characterized by local invasion into surrounding tissues, particularly the jawbone. In dogs, it commonly presents as a painless mass and tends to infiltrate the surrounding gingival tissue.[9,10] In this case, the tumor similarly began in the gingiva, eventually spreading to the jawbone, leading to swelling, as reported by the owner.

Radiographic imaging, including X-ray, CT, and MRI, is essential for assessing the extent of these tumors.[11] In the present case, radiographs revealed areas of radiopacity and radiolucency in the alveolar bone, especially around the affected teeth. Notably, canine tooth 104 was displaced due to the tumor growth, indicating local infiltration and disruption of surrounding tissues.

Mayer et al.[2] have suggested that radiation therapy may be appropriate when surgery is not feasible due to functional or cosmetic concerns or if the tumor cannot be completely excised. Radiation therapy has proven to be a

generally safe and effective treatment, with a clinical study of 47 dogs showing good outcomes. However, the risk of recurrence increases in cases of larger tumors, particularly those over 4 cm in diameter (T3 clinical stage). Goldschmidt et al. further suggested that medical therapies may serve as viable alternatives to more invasive surgical procedures.[3] However, in the present study, radical surgical excision of large masses of soft and bone tissues was effective in restoring normal function and preventing malnutrition caused by the tumor's interference with food intake.

Stancu et al.[5] reported that the tumor exhibited a stratified epithelial-like structure and an anastomosing band and the majority of the cells were composed of epithelial cells with round nuclei. Two different cell types, fibroblast and osteoblast, were identified. In this case, it was observed that there were ameloblastomatous islands in the thickening of the stratum spinosum layer and increased vascularization with osseous island formations in the dermis layer.

In the literature, no cases were found where a dog with CAA had a previous history of transmissible venereal tumor (TVT). This raises the possibility that dogs with TVT may have an increased predisposition to developing neoplastic disorders, warranting further investigation.

In conclusion, canine acanthomatous ameloblastoma (CAA) may rarely develop in Anatolian Shepherd (Kangal) dogs and radical surgical excision of the affected soft and bone tissues is necessary when lesions cause significant functional impairments such as malnutrition. Furthermore, the potential association between CAA and transmissible venereal tumor (TVT) warrants further investigation to determine whether the presence of TVT predisposes dogs to subsequent neoplastic conditions.

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