

Amyloid-Producing Odontogenic Tumor in a Shih-Tzu Dog

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ABSTRACT. A 9-month-old male Shih-Tzu dog had a right mandibular tumor composed of strands, or nest-like proliferation of epithelial cells with abundant fibrous stroma characterized by spheroid to large nodular deposition of amyloid with Congo-red stain. Globule calcification was also seen throughout the tumor tissue and the spheroid depositions often had a concentrically laminated structure (Liesegang rings). The case was diagnosed as amyloid-producing odontogenic tumor in a dog.—**KEY WORDS:** amyloid, canine, odontogenic tumor.

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Odontogenic tumors are rare in animals [2], mostly arising in the dental tissues and histologically not malignant, while sometimes causing destruction of bone and displacement of teeth. Calcifying epithelial odontogenic tumor (CEOT) is rare in animals and characterized by epithelial proliferation, mineralization in the epithelium and stroma, and deposition of amyloid or amyloid-like materials [1, 4–8]. This note is to report histopathology and immunohistochemistry of CEOT in a Shih-Tzu dog.

A 9-month-old male Shih-Tzu dog was presented to a private veterinary hospital for swelling of anterior part of the right mandible in August 1995 and tentatively diagnosed as ranula. About one month later, the dog was referred to the Veterinary Teaching Hospital of Osaka Prefecture University and shown to have many cysts containing red-brown fluid in the lesion. Radiological examination revealed bone absorption in the mandible, and the lesion was removed surgically (Fig. 1). The dog was still surviving for 4 years after operation.

The removed mass was fixed in 10% neutral buffered formalin and decalcified in 5% formic acid solution. The samples were routinely processed for paraffin embedding. Sections 4 μ m in thickness were made and stained with hematoxylin and eosin (HE). Selected sections were also stained with Congo-red and Kossa's method. Some deparaffinized sections were immunostained for cytokeratin and vimentin (DAKO, prediluted and DAKO, 1:500, respectively) with the avidin-biotin peroxidase kit (DAKO). Immunoreaction was visualized by diaminobenzidine with 0.005% hydrogen peroxide.

The tumor developing within the jaw bone was poorly demarcated without fibrous capsulation. It was composed of strands or nests of proliferated epithelial cells supported by abundant fibrous stroma (Fig. 2). The epithelial cells often resembled the stellate reticulum of the enamel structure; the basal cells with a hyperchromatic nucleus often showing a cap-like arrangement (Fig. 3). In some areas, the epithelial cells varied in morphology, having a short-spindle or ovoid nucleus with lightly eosinophilic

cytoplasm varying in size. At the center of nodular growth of epithelial cells, many cells with a short-spindle nucleus proliferated. Occasionally, cellular nests with abundant eosinophilic cytoplasm were seen, indicating squamous differentiation (Fig. 4). The lesions were characterized by the presence of spheroid or nodular deposition of eosinophilic materials stained positively with Congo-red within the epithelial strands as well as the stroma (Fig. 3). Globule calcification up to 70 μ m in diameter, which were clearly demonstrated by Kossa's method, was distributed throughout the tumor tissue (Fig. 5). Spherical mineral depositions often had a concentrically laminated structure and were so-called Liesegang rings (Fig. 6). Various-sized cysts, partly lined by flattened or cuboidal epithelial cells were seen.

Immunohistochemically, the neoplastic epithelial cells were positive for cytokeratin (Fig. 7), whereas the abundant fibrous stromal cells were positive for vimentin.

The present case was diagnosed as a case of amyloid-producing odontogenic tumor [2, 4], characterized by the presence of prominent amyloid deposition and spheroidal calcification. There are two possible explanations of origin

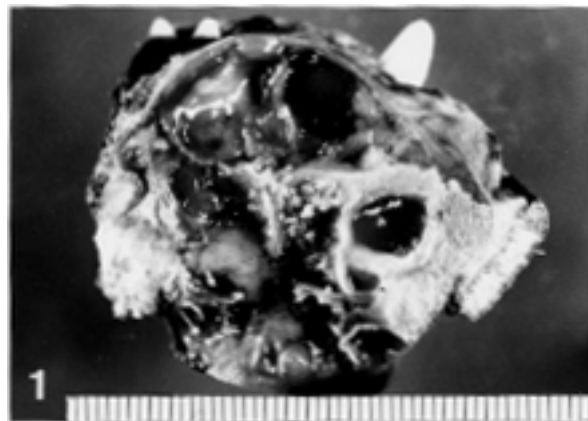


Fig. 1. Gross appearance of the mandibular tumor with many cysts.

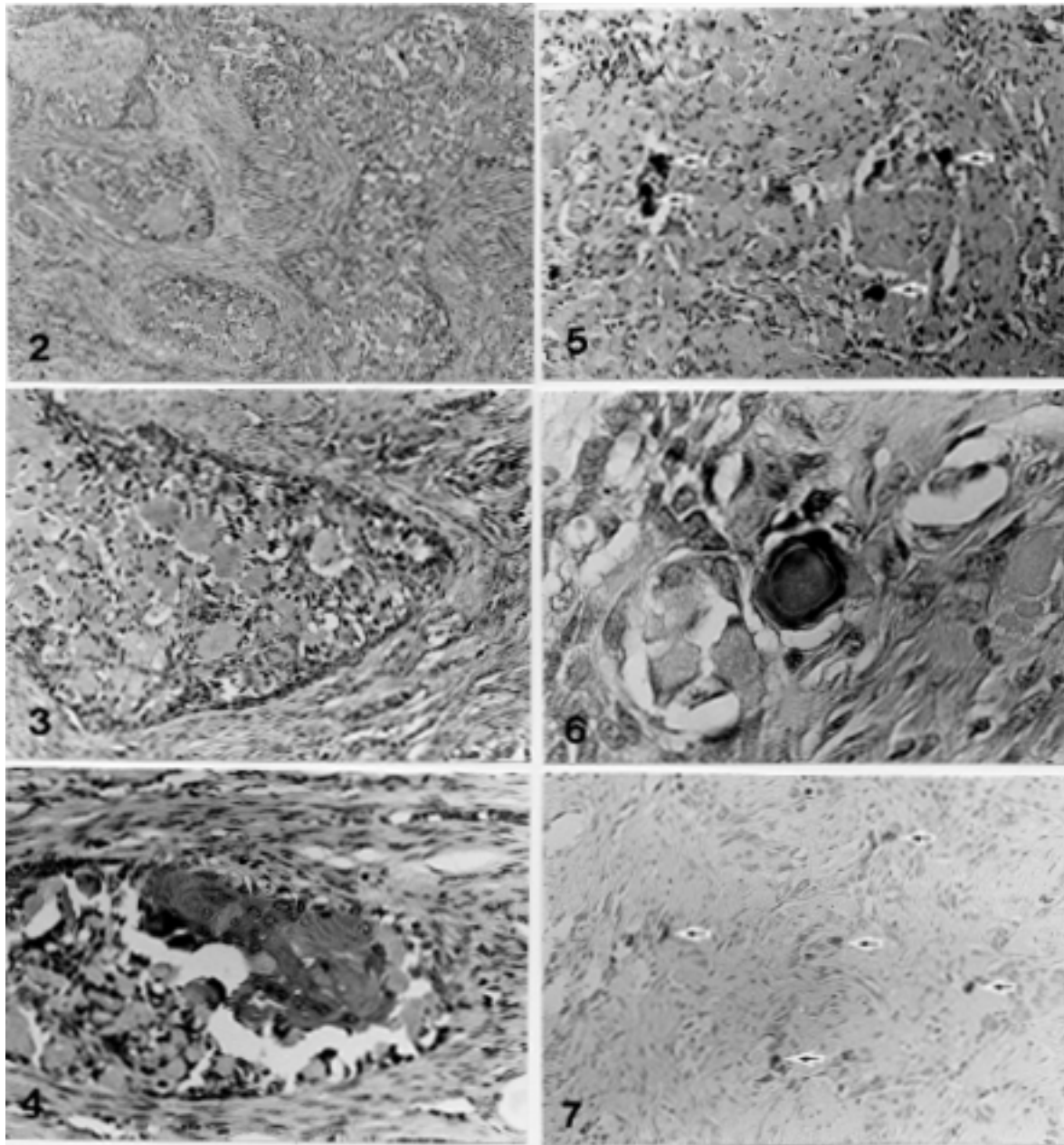


Fig. 2. Strand- or nests-like growth of epithelial cells with abundant fibrous stroma. HE.

Fig. 3. Cap-like arrangement of epithelial cells containing spheroid depositions of amyloid. HE.

Fig. 4. Epithelial cells with squamous differentiation. HE.

Fig. 5. Spheroid calcifications (arrows) in the tumor. HE.

Fig. 6. Concentrically laminated calcification (Liesegang ring) in the tumor. HE.

Fig. 7. Epithelial cells positive for keratin (arrows). Immunohistochemistry.

of the tumor; one is gingival epithelium and the other is odontogenic epithelium within the connective tissue of the gingiva or within bone [4]. Some cases of keratinizing ameloblastomas were reported to exhibit amyloid deposits in varying extents [3], and the present case showed a large amount of amyloid accumulation throughout the tumor tissue.

Four canine and 8 feline cases of CEOT have been reported in foreign countries [1, 4–8]. The canine cases were more than 8 years old, but the present case was 9 months of age. Some cases were described to recur after excision, but no metastasis was described [4]. The present case survived for 4 years after operation, indicating favorable prognosis.

There appears to be some differences in CEOTs between animals and man [4]. Human CEOTs consist of sheets of eosinophilic epithelial cells showing considerable nuclear pleomorphism and invasive growth, whereas CEOTs in animals mostly showed basal cells with hyperchromatic nuclei, arranged in palisades. The present case had both epithelial cells with nuclear pleomorphism and hyperchromatic basal cells. Because of these differences, Gardner *et al.* [4] proposed amyloid-producing odontogenic tumor as an appropriate alternative term for CEOTs in animals.

REFERENCES

1. Abbott, D.P., Walsh, K. and Ditters, R.W. 1986. *J. Comp. Pathol.* 96: 131-136.
2. Baker, I.K., Dreumel, A.A. and Palmer, N. 1993. pp. 25-27. *In: Pathology of Domestic Animals*, vol.2, 4th ed. (Jubb, K.V.F., Kennedy, P.C. and Pater, N. eds.), Academic Press, San Diego.
3. Gardner, D.G. and Dubielzig, R.R. 1993. *J. Comp. Pathol.* 109: 423-428.
4. Gardner, D.G., Dubielzig, R.R. and McGee, E.V. 1994. *J. Comp. Pathol.* 111: 221-230.
5. Langham, R.F., Bennett, R. and Koestner, A. 1984. *Vet. Pathol.* 21: 549-550.
6. Poulet, F.M., Valentine, B.A. and Summers, B.A. 1992. *Vet. Pathol.* 29: 369-380.
7. Stebbins, K.E., Morse, C.C. and Goldschmidt, M.H. 1989. *Vet. Pathol.* 26: 121-128.
8. Walsh, M., Denholm, L.J. and Cooper, B.J. 1987. *J. Comp. Pathol.* 97: 503-521.