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Authors

Loukopoulos, Panayiotis
Okuni, Julius Boniface
Micco, Teresa
et al.

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Author(s): Panayiotis Loukopoulos, D.V.M., PG.Dip.V.St., Ph.D., Julius Boniface Okuni, B.V.M., M.Sc., Ph.D., Teresa Micco, D.V.M., Jorge P. Garcia, D.V.M., Francisco A. Uzal, D.V.M., M.Sc., Ph.D., Dipl. A.C.V.P., and Santiago S. Diab, D.V.M., Dipl. A.C.V.P.

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AIR SAC ADENOCARCINOMA OF THE STERNUM IN A QUAKER PARROT (*MYIOPSITTA MONACHUS*)

Panayiotis Loukopoulos, D.V.M., PG.Dip.V.St., Ph.D., Julius Boniface Okuni, B.V.M., M.Sc., Ph.D., Teresa Micco, D.V.M., Jorge P. Garcia, D.V.M., Francisco A. Uzal, D.V.M., M.Sc., Ph.D., Dipl. A.C.V.P., and Santiago S. Diab, D.V.M., Dipl. A.C.V.P.

Abstract: Respiratory neoplasia is rarely reported in avian species. A 17-yr-old Quaker parrot (*Myiopsitta monachus*) was admitted with a 2-wk history of anorexia, depression, and respiratory distress. Clinical examination revealed a large, firm mass in the left pectoral muscle. Radiology showed a mass silhouetting the heart and the liver. Supportive treatment was provided, but the bird died during the seventh weekly visit to drain thoracic cavity fluid. Necropsy showed a white, 3 × 3 × 2–cm, hard, gritty sternal mass. Histology showed a nonencapsulated, moderately differentiated air sac carcinoma of the sternum. Immunohistochemically the neoplasm was cytokeratin positive and vimentin and calretinin negative. This is the first report of an air sac neoplasia in a Quaker parrot and one of few respiratory tumors in psittacines.

Key words: Adenocarcinoma, air sac, bone tumor, psittacine, Quaker parrot, sternum.

BRIEF COMMUNICATION

Reports of tumors in general, and of respiratory carcinomas in particular, are scarce in psittacines and other avian species. Tumor incidence represented 1.9% ($n = 5,957$) and ~3.8% ($n > 10,000$) of all avian cases in two studies from California[4] and Australia,[12] respectively. Only one and two respiratory tumors were diagnosed in each of those studies, respectively.[4] Reports of respiratory and thoraco-brachial tumors in Psittaciformes include two cystadenocarcinomas of the right humerus in moluccan cockatoos (*Cacatua moluccensis*),[8,9] a cystadenocarcinoma of the left humerus of a sulphur crested cockatoo (*Cacatua galerita*),[10] an adenocarcinoma of the left axilla in three galahs (*Eolophus roseicapillus*),[10] air sac adenocarcinomas of the left humerus and axilla of an African grey parrot (*Psittacus erithacus*),[1] a bronchiolar adenoma in a parrot,[12] a pulmonary carcinoma with metastases in a moluccan cockatoo,[7] and a pulmonary adenocarcinoma in a blue and gold macaw (*Ara ararauna*).[5] Respiratory tumors in other avian orders included two pulmonary neoplasms in zoo birds,[11] a pulmonary carcinoma metastatic to the air sacs and humerus in a great horned owl (*Bubo virginianus*),[13] and a bronchial carcinoma in a red-shouldered hawk (*Buteo lineatus*).[6] To the authors' knowledge, published reports of tumors in Quaker parrots (*Myiopsitta monachus*) are limited to two liposarcomas.[14]

A 17-yr-old female Quaker parrot was presented to a clinic (day 0) with a history of nonproductive sneezing, inability to fly, depression, and anorexia of 2 wk duration. On physical examination, the bird was in fair nutritional condition and was unable to fly and in respiratory distress characterized by labored breathing, a clicking respiratory sound, sneezing, and tail-bobbing. The right pectoral muscle showed marked atrophy, while the left had a large, firm, round swelling, measuring approximately 2.5 × 2 × 1.5 cm.

The bird was treated with supportive therapy (day 0), which included oxygen and fluid administration, gavage feeding, analgesics, and antibiotics. Radiologic examination on day 3 revealed a mass described as an opaque to hazy radio density in the left pectoral muscle and cranial abdomen invading the thoracic wall and silhouetting the cranial left border of the liver and the caudal left border of the heart. The thoracic air sacs appeared underinflated, although the bronchial air sac was not affected. The ventriculus was severely

From the California Animal Health and Food Safety Laboratory System, School of Veterinary Medicine, University of California, Davis, 105 West Central Avenue, San Bernardino, California 92408, USA (Loukopoulos, Garcia, Uzal, Diab); the College of Veterinary Medicine, Animal Resources and Biosecurity, P.O. Box 7062, Makerere University, Kampala, Uganda (Okuni); and the Point Vicente Animal Hospital, 31270 Palos Verdes Drive West, Rancho Palos Verdes, California 90275, USA (Micco). Present addresses (Diab): California Animal Health and Food Safety Laboratory System, School of Veterinary Medicine, University of California, Davis, West Health Sciences Drive, Davis, California 95616, USA; (Loukopoulos): the Veterinary Diagnostic Laboratory, Building 295, School of Animal and Veterinary Sciences, Charles Sturt University, Nathan Cobb Drive, Wagga Wagga, New South Wales 2650, Australia. Correspondence should be directed to Dr. Loukopoulos (ploukopoulos@csu.edu.au).

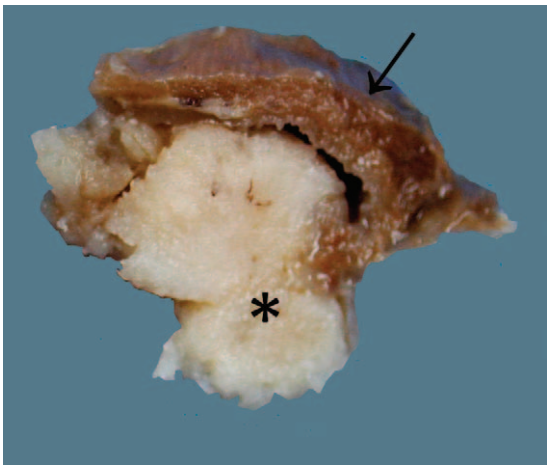

Figure 1. Formalin-fixed mass showing a white, oval, irregular, well-defined tumor (asterisk) of the sternum (arrow).

displaced dorsocaudally. Fine needle aspiration on day 8 yielded brown opaque fluid, with a specific gravity of 1.010, 306 white blood cells/μl, 27,000 red blood cells/μl, and <2.0 g protein/dl. Cytologic examination of direct and concentrated preparations of the fluid removed from the left pectoral muscle showed moderate hemodilution, large numbers of lysed cells, and a low number of intact macrophages, some with engulfed material, suggestive of chronic inflammation. After 12 days in the clinic (day 12), the patient regained the ability to eat on its own and was discharged, despite the still-guarded prognosis. Five days after discharge (day 17), the bird was brought back to the clinic, and 9 ml of serosanguineous fluid was drained from the mass. Drainage of the mass was repeated on day 20 and then at weekly intervals for 6 wk. The patient died on day 69 while en route to the clinic for the routine procedure. A field necropsy was performed by the attending veterinarian. Grossly, the sternum and left pectoral musculature were severely destroyed and distorted by a hard, gritty mass. Formalin-fixed tissues, including a portion of the mass with the keel, lungs, intestines, and liver, were submitted to the California Animal Health and Food Safety Laboratory in San Bernardino for histopathology.

Grossly, the sternal mass was white, roughly oval, hard, and gritty and measured approximately 3 × 3 × 2 cm (Fig. 1). Hard tissue segments were decalcified. All tissues were processed by standard histologic techniques for the production of 4-μm–thick hematoxylin and eosin sections. The sternal mass was composed of a neoplasm that completely effaced normal bone and air sac architecture and destroyed or severely distorted sternal bone spicules, often resulting in significant bone remodeling. The sternal mass occupied and expanded most of the intertrabecular space and invaded and expanded adjacent tissues, including the alveolar space of the adjacent nonosseous air sac (Fig. 2). Neoplastic cells lined the bony trabeculae, at places gradually transitioning from normal respiratory epithelium of the sternal air sac, and were arranged in variably sized and irregularly shaped, often tortuous, tubules and acini that often piled in layers or formed papillary projections and were multifocally supported by moderate amounts of fibrous stroma (Fig. 2). Tumor cells were tall and cuboidal and had an occasional brush border, abundant eosinophilic cytoplasm, and basilar or centrally located vesicular nuclei. There was moderate anisocytosis and anisokaryosis and marked apoptosis. The mitotic index was high (average 3 mitotic figures per high-power field [×400]). The lumen of many tubules contained apoptotic neoplastic cells. A moderate number of multinucleated giant cells and osteoclasts were observed. The lungs were moderately and diffusely congested. The parabronchial epithelium was focally markedly hyperplastic and in places showed mild dysplasia. In several foci, the parabronchi and respiratory bronchi were narrowed by the abundant presence of foamy macrophages in the respiratory atria and parabronchial epithelium. There was no evidence of tumor expansion to the lung tissue or of metastases in any the organs examined. No epithelia, other than those of the air sac, showed evidence of tumor involvement.

The immunohistochemical expression of cytokeratin (BioCare, Concord, California, 94520 USA), vimentin (DAKO, Carpinteria, California, 93013 USA) , calretinin (Zymed/Life Technologies, New York, New York 14072 USA), CD3 (Leukocyte Antigen Biology Laboratory, Peter F. Moore, University of California–Davis, Davis, California, 95616 USA), and CD79a (AbDSerotec, Raleigh, North Carolina, 27609 USA) by the neoplastic cells was examined on 4-μm–thick, formalin-fixed, paraffin-embedded tissue sections, with either heat-induced epitope retrieval using the DAKO Target Retrieval Solution (in a microwave oven set at 25% power) or enzymatic digestion with DAKO Proteinase K, per the manufacturer's instructions (Table 1). All incubations were performed in a humidity chamber at room temperature. Sections were counterstained

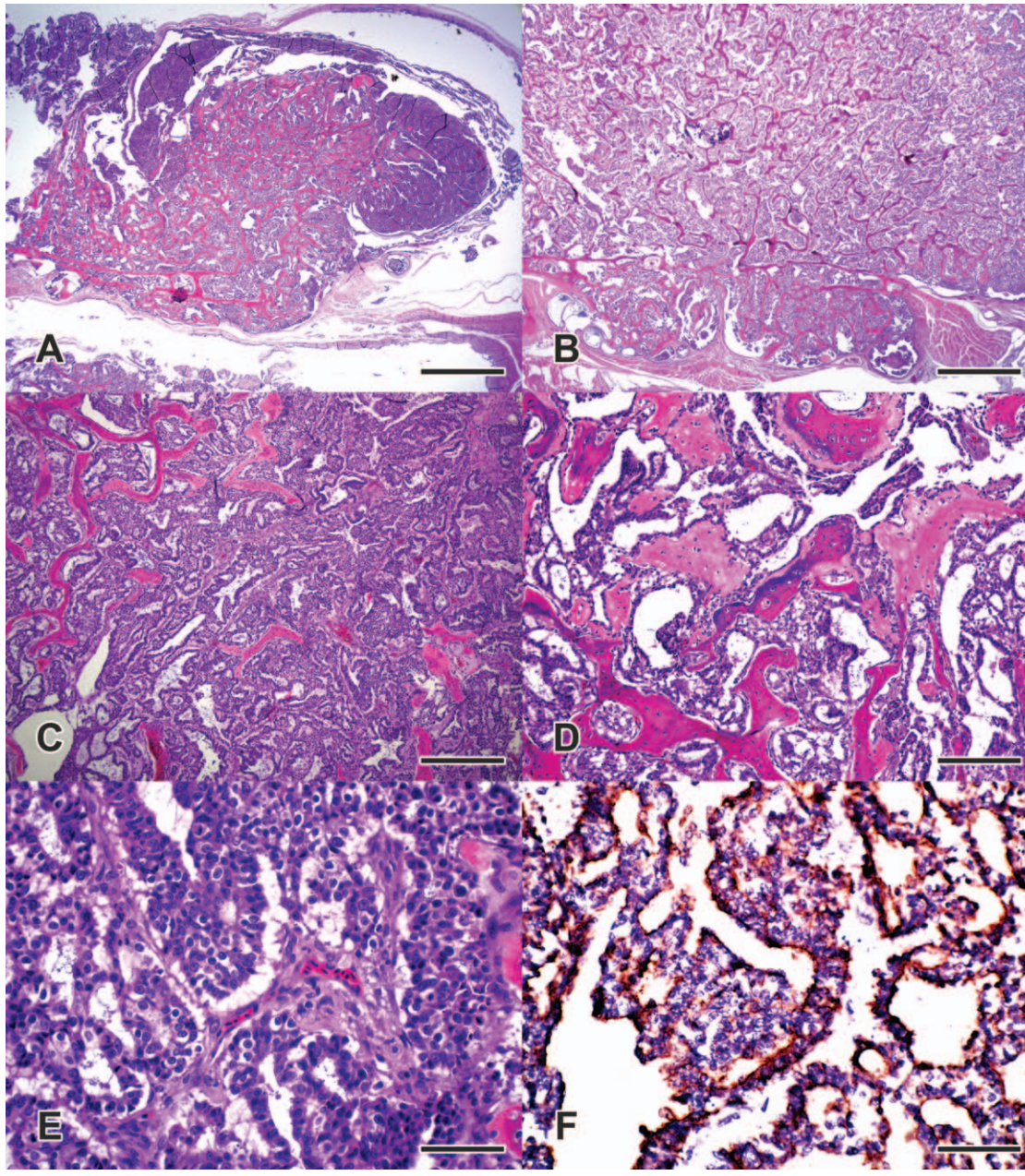

Figure 2. Air sac adenocarcinoma of the sternum. The neoplasm occupied and expanded most of the intertrabecular space (**A–D**) and invaded adjacent soft tissues (**A**). The tumor formed variably sized and shaped, tortuous acini and tubules (**B**); effaced normal bone architecture (**C**); and destroyed or severely distorted sternal bone spicules, often resulting in significant bone remodeling (**D**). Tubules were often multilayered (**E**). Tumor cells showed intense cytoplasmic cytokeratin staining (**F**). **A–E:** Hematoxylin and eosin; **F:** streptavidin–biotin, Meyer hematoxylin counterstain. (**A**) Bar = 1,900 μm, (**B**) Bar = 950 μm, (**C**) Bar = 380 μm, (**D**) Bar = 190 μm, (**E**) Bar = 63 μm, and (**F**) Bar = 190 μm.

with Mayer hematoxylin. Nonspecific background was evaluated with duplicate sections receiving diluent in place of the primary antibody. Positive controls were suitable tissues from other psittacines. Nontumorous mesenchymal and epithelial tissues in the sections from the Quaker parrot subjected to immunohistochemistry provided excellent internal positive controls for

Table 1. Immunohistochemical reagents and protocols used and results obtained on the air sac carcinoma.[a]

Antibody	Clone	Company	Origin	Dilution	ER	Result
Pan-cytokeratin	LU5	BioCare	Mouse	1 : 100	HIER	Positive
Vimentin	3B4	Dako	Mouse	1 : 300	ED 7 min	Negative
Calretinin	DC8	Zymed	Mouse	1 : 200	HIER	Negative
CD3	CD3-12	LABL	Rat	1 : 10	HIER	Negative
CD79a	HM57	AbDSerotec	Mouse	1 : 100	HIER	Negative

[a] All antibodies were monoclonal. ED indicates enzymatic digestion (DAKO Proteinase K, Dako Corp.); ER, epitope retrieval; HIER, heat-induced epitope retrieval (DAKO Target Retrieval Solution, Dako Corp.); LABL, Leukocyte Antigen Biology Laboratory, Peter F. Moore, University of California, Davis, California, USA.

vimentin and cytokeratin, respectively. Neoplastic cells showed diffuse, strong cytoplasmic cytokeratin expression (Fig. 2F) and did not express vimentin, calretinin, CD3, and CD79a, indicating that the neoplastic cells were of epithelial, rather than mesenchymal, mesothelial or lymphoid origin. A diagnosis of air sac adenocarcinoma of the sternum was established, based on the grossly and histologically evident destruction and distortion of the sternum by the mass; the histologic evidence of the presence of features overall denoting epithelial malignancy, including the invasion of the air sac space, expansion into adjacent tissues, formation of irregularly shaped, tortuous tubules, high mitotic index, high apoptotic rate, a range of features denoting an overall moderate degree of cellular anaplasia; and the malignant biologic behavior, ultimately resulting in the animal's death.

The respiratory signs exhibited by the parrot were nonspecific and can be produced by several etiologies, including viral, bacterial, and mycotic pulmonary infections.[3,6] The radiologic and cytologic findings could also suggest a chronic respiratory disease such as tuberculosis or mucormycosis.[3] Supportive treatment of the tumor appeared to help only at the initial stages. It is not clear whether the fluid drained was due to vascular compromise or was produced by the glandular neoplasm.

Adenocarcinoma of the air sacs within the bony trabeculae of the sternum is an exceedingly unusual location. The sternum is a pneumatic bone, and its trabeculae are lined by respiratory epithelium, constructing an intraosseous air sac that is continuous with the nonosseous (interclavicular, thoracic, and abdominal) air sacs. Surgical debulking of the tumor was not attempted.

The bird was housed indoors in an apartment built with asbestos-containing materials, and the owner was a heavy smoker and frequent incense user. Tobacco smoke contains thousands of chemicals, many of which are known carcinogens. Prolonged exposure to it is a well-established cause of lung cancer in humans and is thought to account for nearly one third of all cancer deaths in people. The possibility that this respiratory tumor was the result of passive smoking cannot therefore be ruled out. Exposure to asbestos in building sites or in the household has been associated with changes in the respiratory epithelium and increased risk for the development of mesothelioma and other respiratory pathology.[2] It is highly unlikely, however, that the bird in the present case had been exposed to the asbestos, and therefore any association between prior exposure to asbestos and the development of the neoplasm in the present case cannot be established.

Parrots are popular pets that live relatively long lives; however, reports of neoplasms in psittacines birds are scarce. This report is one of few on tumors of the Quaker parrot and one of few of air sac neoplasia in any species. Air sac adenocarcinoma should be included in the differential diagnosis of respiratory diseases in psittacines.

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