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Case Report—

Internal Papillomatosis with Intrahepatic Cholangiocarcinoma and Gastrointestinal Adenocarcinoma in a Peach-Fronted Conure (Aratinga aurea)

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SUMMARY. A 17-yr-old pet female peach-fronted conure (*Aratinga aurea*) was presented with the chief complaints of mild lethargy and weight loss with increased appetite. Antemortem diagnostics included complete blood count, plasma biochemistry, and radiography. Abnormal findings included elevated inflammatory parameters (hyperfibrinogenemia) and a space-occupying mass in the region of the liver. Histologic examination of a liver biopsy sample indicated bile duct hyperplasia leading to a presumptive diagnosis of hepatoxicosis. The bird initially showed moderate improvement with supportive care, but its condition declined 9 days after the liver biopsy. Supportive care was attempted a second time, but the bird did not improve and euthanasia was elected. Abnormal gross necropsy findings were confined to the liver, which contained multiple tan nodules that exuded yellowish fluid on cut section. Histopathologic examination revealed multicentric bile duct hyperplasia and cholangiocarcinoma as well as segmental papillary hyperplasia and adenocarcinoma in the proventriculus, ventriculus, and throughout the intestinal tract. This is the first report of concurrent internal papillomatosis, gastrointestinal adenocarcinoma, and cholangiocarcinoma in a peach-fronted conure.

RESUMEN. *Reporte de Caso*—Papilomatosis interna con colangiocarcinoma intrahepático y adenocarcinoma gastrointestinal en una lora de frente dorada (Aratinga aurea).

Una lora mascota de frente dorada (*Aratinga aurea*) de 17 años de edad fue presentada por su propietario, quien describió la presencia de letargo suave y pérdida de peso con un aumento del apetito. Los diagnósticos, antes de su muerte, incluyeron un recuento sanguíneo completo, análisis bioquímico del plasma sanguíneo y radiografía. Los hallazgos anormales incluyeron parámetros de inflamación elevados (hiperfibrinogenemia) y una masa grande en el hígado. El examen histológico de una biopsia del hígado indicó la presencia de hiperplasia de los conductos biliares, la cual condujo a un diagnóstico presuntivo de hepatotoxicosis. El ave mostró una mejoría inicial moderada al recibir tratamiento de sostén, pero su condición declinó 9 días después de la biopsia del hígado. Se ensayó el tratamiento de sostén por segunda vez, sin embargo, el ave no mostró signos de mejoría y se eligió el uso de la eutanasia. A la necropsia, los hallazgos macroscópicos anormales se restringieron al hígado, el cual presentó múltiples nódulos oscuros que al corte producían un exudado amarillento. El examen histopatológico reveló una hiperplasia multicéntrica de los conductos biliares y colangiocarcinoma, al igual que hiperplasia papilar segmentada y adenocarcinoma en el proven-

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trículo, ventrículo y en la totalidad del tracto intestinal. Este es el primer reporte de papilomatosis interna concurrente, adenocarcinoma gastrointestinal y colangiocarcinoma en una lora de frente dorada.

Key words: peach-fronted conure, *Aratinga aurea,* papillomatosis, cholangiocarcinoma, adenocarcinoma, neoplasia, biliary hyperplasia

Abbreviations: b.i.d. = twice a day; CBC = complete blood count; H&E = hematoxylin and eosin; HPF = high-power field; i.m. = intramuscularly; LRS = lactated Ringer solution; p.o. = per os; s.c. = subcutaneously; s.i.d. = once a day; VMTH = Veterinary Medical Teaching Hospital

Internal papillomatosis has been reported in 19 species of New World Psittaciformes including two conure species, *Aratinga aurea* and *Aratinga canicularis* (11,12,34,35). The histopathologic description of internal papillomatosis of parrots resembles that of papillomavirus-induced papillary lesions in the mouth, pharynx, and esophagus of cattle, dogs, and rabbits (14,17,33,36). In birds, however, papillomavirus has been demonstrated only in an African gray parrot (*Psittacus erithacus*) (21). In another report, a herpes-like virus was found in association with a cloacal papilloma in an orange-fronted conure (*A. canicularis*), although the authors believed that the finding was incidental (11). The authors of another report suggested that an underlying mechanism in the pathogenesis of internal papillomatosis might be mucosal irritation that is followed by hypertrophy and hyperplasia of the lining epithelium (34).

Hepatic cholangiocarcinoma in psittacine birds has been thoroughly documented (4,5,9, 15,18,19,20,22,24,25,29). Psittacine gastric and intestinal adenocarcinomas have been reported as well (8,13,18,22,23,31,32). Internal papillomatosis is occasionally found in association with biliary, hepatic, intestinal, and/or pancreatic carcinomas in Amazon parrots (*Amazona* spp.) and macaws (*Ara* spp.) (4,12,15,18,19). This association has not, however, been described in a peach-fronted conure (*A. aurea*). This report describes the clinical progression of antemortem signs and the postmortem diagnosis of internal papillomatosis, multicentric intrahepatic cholangiocarcinoma, gastric adenocarcinoma, and multifocal intestinal adenocarcinoma in a peach-fronted conure.

CASE REPORT

History. A 17-yr-old female peach-fronted conure was presented for a 1-mo history of decreased activity and weight loss despite increased food intake. The owner also reported increased liquid in the excrement over that time. Approximately 1 mo before presentation, another veterinarian examined the bird after a single episode of its passing dark red excrement. The owner administered a prescribed course of an oral antibiotic (drug name and dosage were not recorded), and the color of the excrement returned to normal.

The conure had been the only pet in the household for over 10 yr. It was fed a diet of commercial pelleted psittacine ration (Maintenance Pelleted Food for Seed-eating Birds [Roudybush, Inc., Paso Robles, CA] or Premium Daily Diet for Parrots [Lafeber Company, Cornell, IL]) and an equal proportion of a mixture of seeds purchased from bulk bins at a local retail pet store. The daily diet was supplemented with fruits, meats, cheeses, vegetables, and pasta. Cod-liver oil (0.5 ml) was administered twice weekly. The owner reported no previous health problems.

Upon presentation to the Veterinary Medical Teaching Hospital (VMTH), the bird was perched and quiet but responsive to stimuli. Respiratory effort was mildly increased, and the respiratory rate was 24 respirations per minute. The excrement was polyuric and diarrheic. On physical examination, pectoral muscle mass was markedly decreased, and the bird weighed 58 g. Feathers were absent in a locally extensive region adjacent to the ventral apterium. No abnormalities were noted on auscultation or palpation.

Diagnostic tests at the time of admission included a complete blood count (CBC), plasma biochemical analysis, serum bile acid concentration, fecal Gram stain, fecal Ziehl–Neelsen acid-fast stain, and full-body radiographs. Abnormal CBC findings (reference ranges in parentheses) included moderate toxic changes in

the mature heterophils, 754 band heterophils/μl (0/μl) with moderate toxic changes, and 348 lymphocytes/μl (800–5500/μl) (2). Abnormal plasma biochemistry findings (reference ranges in parentheses) included total calcium 7.8 mg/dl (8–15 mg/dl), total protein 2.0 g/dl (2.5–4.5 g/dl), albumin 0.6 g/dl (1.9–2.6 g/dl), and creatine kinase 954 IU/l (35–355 IU/l) (2). Bile acid concentration, aspartate aminotransferase activity, and lactate dehydrogenase activity were within reference ranges. Acid-fast- and Gram-stained fecal smears were considered to be normal. Radiographs demonstrated a large caudoventral coelomic soft tissue mass partially obscuring visualization of the thoracic air sac region with dilated and gas-filled intestines. An upper gastrointestinal positive contrast study (Novopaque, 60% [w/v] barium sulfate suspension, 30 ml/kg via oro-ingluvial tube; LPI Diagnostics, Yorba Linda, CA) showed that the coelomic space-occupying mass was in the region of the liver. Positive contrast fluoroscopy revealed that gastrointestinal motility was moderately decreased.

In light of the hypoalbuminemia, hypoproteinemia, and apparent hepatomegaly, the bird was prepared for surgical liver biopsy. Anesthesia was induced with 3% isoflurane (IsoFlo®; Abbot Laboratories, North Chicago, IL) in oxygen (1.5 liters/min) delivered by face mask via a nonrebreathing system. A 2.0-mm internal diameter uncuffed tracheal tube (MMJ S.A. de C.V., a Mallinckrodt Company, CD. Juarez, Chihuahua, Mexico) was inserted and anesthesia was maintained with isoflurane (1.5%–2.0%) in oxygen (1 liter/min). A 10-mm transverse skin incision was made with a no. 15 scalpel blade 3 mm caudal to the sternum. The liver was identified, and a single wedge of hepatic parenchyma measuring 2 × 2 × 1 mm was collected with iris scissors and placed into 10% neutral buffered formalin solution. Hemorrhage was controlled by the direct application of pressure with cotton tipped applicators and the placement of a small section of absorbable gelatin sponge (Gelfoam®; Pharmacia and Upjohn Co., Kalamazoo, MI). The surgical site was closed in two layers (body wall and skin) with simple interrupted 4-0 polydioxanone sutures (PDS II®; Ethicon, Inc., Somerville, NJ). The histologic diagnosis was mild multifocal biliary hyperplasia and hepatocellular anisokaryosis and anisocytosis suggestive of a toxic etiology.

The bird improved with 3 days of medical care including enrofloxacin (Baytril® 2.27% solution, 10 mg/kg per os [p.o.] twice daily [b.i.d.]; Bayer Corp., Shawnee, KS), lactulose (666 mg/ml solution, 333 mg/kg p.o. b.i.d.), fluid therapy (lactated Ringer solution [LRS], 50 ml/kg subcutaneously [s.c.] b.i.d.), and nutritional support (Critical Care®, 3–5 ml via oro-ingluvial tube b.i.d.; Lafeber Company, Cornell, IL). The conure was discharged and the owner was instructed to continue the administration of enrofloxacin and lactulose. Dietary recommendations included feeding fresh vegetables, a packaged seed mix without peanuts or corn, and a commercial formulated psittacine ration. The owner was also instructed to discontinue providing meats, cheeses, and cod-liver oil to the bird.

On the ninth day after discharge, the owner presented the bird to the VMTH after 2 days of lethargy, anorexia, and passing of undigested seeds in the excrement. The conure was perching with both eyes closed and the tail was diverted ventrally. On physical examination, the bird was markedly dehydrated, the site of the surgical biopsy was healing normally, and the body weight had decreased to 54 g. Supportive medical care was initiated with fluid therapy (LRS, 50 ml/kg s.c. b.i.d.), vitamin B complex injection (0.3 ml/kg intramuscularly [i.m.] once daily [s.i.d.]), vitamin K_1 (phytonadione 2 mg/ml injection, 2.5 mg/kg i.m. s.i.d.), and sucralfate (Carafate® 100 mg/ml suspension, 100 mg/kg p.o. b.i.d.; Aventis Pharmaceuticals, Inc., Kansas City, MO). Also, lactulose and enrofloxacin were continued as above. The owner elected to euthanatize the bird after its medical condition did not improve over the course of 2 days.

Necropsy. On gross examination the liver was adherent to the body wall at the healing surgical biopsy site. The liver was mottled brown and friable and had multiple circumscribed, tan, nodular, up to 2-mm diameter areas that exuded thick yellowish fluid on cut section. No other distinctive macroscopic lesion was present.

Histology. Sections of the following tissues were fixed in 10% neutral buffered formalin, processed routinely for TissuePrep® (Fisher Scientific, Fairlawn, NJ) embedding, and sectioned

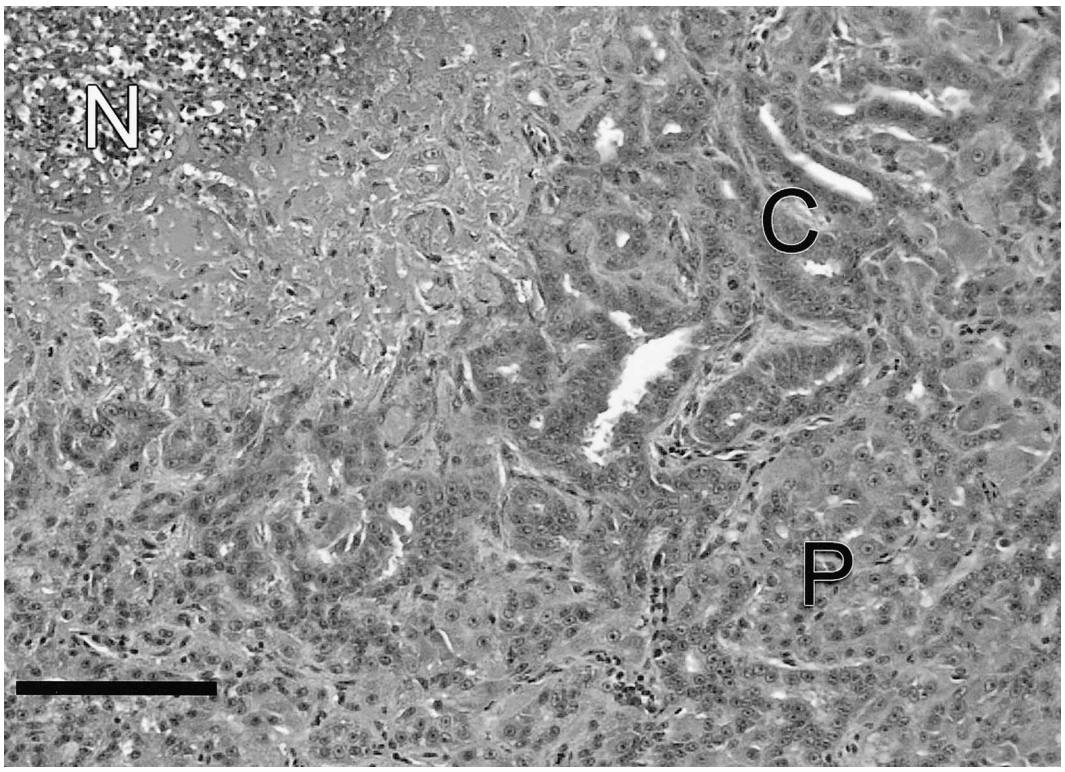

Fig. 1. Liver. Cholangiocarcinoma (C) with mild desmoplasia infiltrating liver parenchyma (P). Central necrosis with heterophilic infiltrate (N) is surrounded by hyalinized fibrous tissue. H&E. Bar = 100 μm.

at 5 μm: heart, aorta, trachea, lung, air sac, liver, ventriculus, proventriculus, duodenum, pancreas, midintestine, distal intestine, spleen, kidney, and ovary. All sections were stained with hematoxylin and eosin (H & E) for light microscopic examination, and liver sections were also stained by Gomorri silver, Brown and Brenn (modified Gram stain), Fite acid-fast, Masson trichrome, Congo red, and phosphotungstic acid–hematoxylin methods.

Throughout the liver there were numerous portal areas with bile duct hyperplasia characterized by an increased number of bile duct profiles and hypertrophy of the lining epithelial cells. Bile duct profiles were also present randomly throughout the liver lobules, occasionally continuous with hepatic cords that also exhibited anisokaryosis and anisocytosis. Multifocally there were cholangiocarcinomas characterized by ''piling up'' of epithelium and increased mitotic figures (Fig. 1). The neoplastic cells occasionally had marked anisocytosis and anisokaryosis. The mitotic rate was between zero and four per high-power field (HPF), with an average of 1.8/HPF. At the periphery of the mass, neoplastic ductules infiltrated along biliary tracts into the surrounding hepatic parenchyma. In central areas of some of the masses there was extensive coagulation necrosis associated with infiltration of heterophils surrounded by a rim of hyalinized connective tissue (Fig. 1). No organism could be demonstrated with special stains. Within sections of the proventriculus and ventriculus, particularly at the proventricular–ventricular junction (isthmus), as well as segmentally throughout the intestine there were multiple foci of mucosal papillary hyperplasia (Fig. 2) with multicentric transformation into adenocarcinoma. Invasion of the submucosa (Fig. 3) and pronounced desmoplasia were evident. The neoplastic cells showed moderate to marked anisocytosis and anisokaryosis. Mitoses were frequent, with 3–13/HPF and an average of 7/HPF. There were also areas of necrosis with associated heterophilic inflammation.

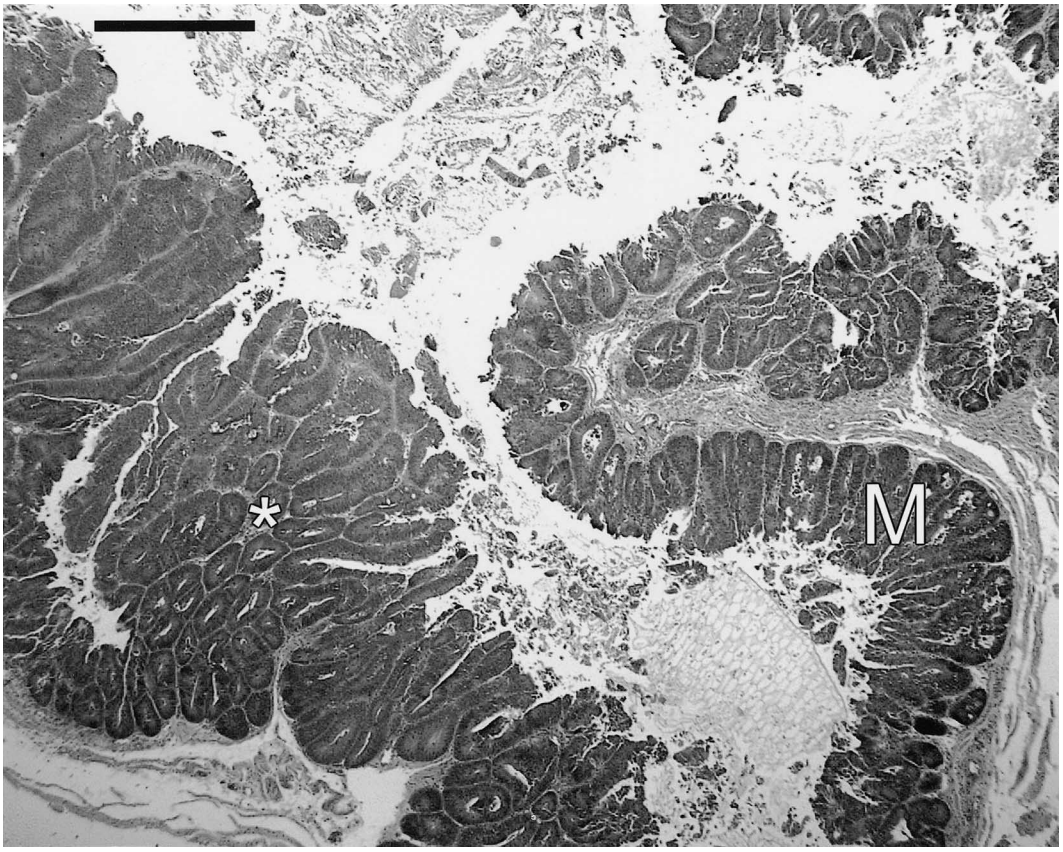

Fig. 2. Midintestine. Diffusely hyperplastic mucosa (M) and focal mucosal papillary hyperplasia (*). H&E. Bar = 400 μm.

In addition to the gastrointestinal lesions, there was depletion of lymphoid tissue in the spleen and atherosclerosis of the aorta. Unfortunately, the cloaca was not secured for histologic evaluation. Transmission electron microscopy was performed with formalin-fixed samples from the hepatic (cholangiocarcinoma) and gastrointestinal (adenocarcinoma) tissues, and no viral particles were seen. Additional diagnostics included aerobic and anaerobic bacterial cultures of lung and liver that produced small numbers of *Lactobacillus* spp.; immunofluorescent antibody staining of liver and lung impression smears that were negative for *Chlamydophila psittaci,* and *Chlamydia* enzyme-linked immunosorbent assay that was also negative.

DISCUSSION

The proliferative lesions were consistent with papillomatous lesions described in New World parrots. One potential explanation for the multicentric lesions in the liver and gastrointestinal tract is exposure to a strong carcinogen via the oral route with hepatic metabolism and biliary excretion. Bile duct hyperplasia has been reported to occur in psittacine birds with hepatotoxic diseases including aflatoxicosis (3,6,16,20,25). Aflatoxins may also be carcinogenic, and bile duct hyperplasia may precede hepatic neoplasia, although controlled studies are lacking in psittacine birds (1,3). In this case, the owner provided a seed mix purchased in small quantities from bulk bins at a local pet retailer, so chronic exposure to a mycotoxin is possible. Another possibility for the etiopathogenesis could be an infection with a transforming virus such as papillomavirus or herpesvirus, but no inclusions were seen under light microscopy and virions were not detected on electron microscopy.

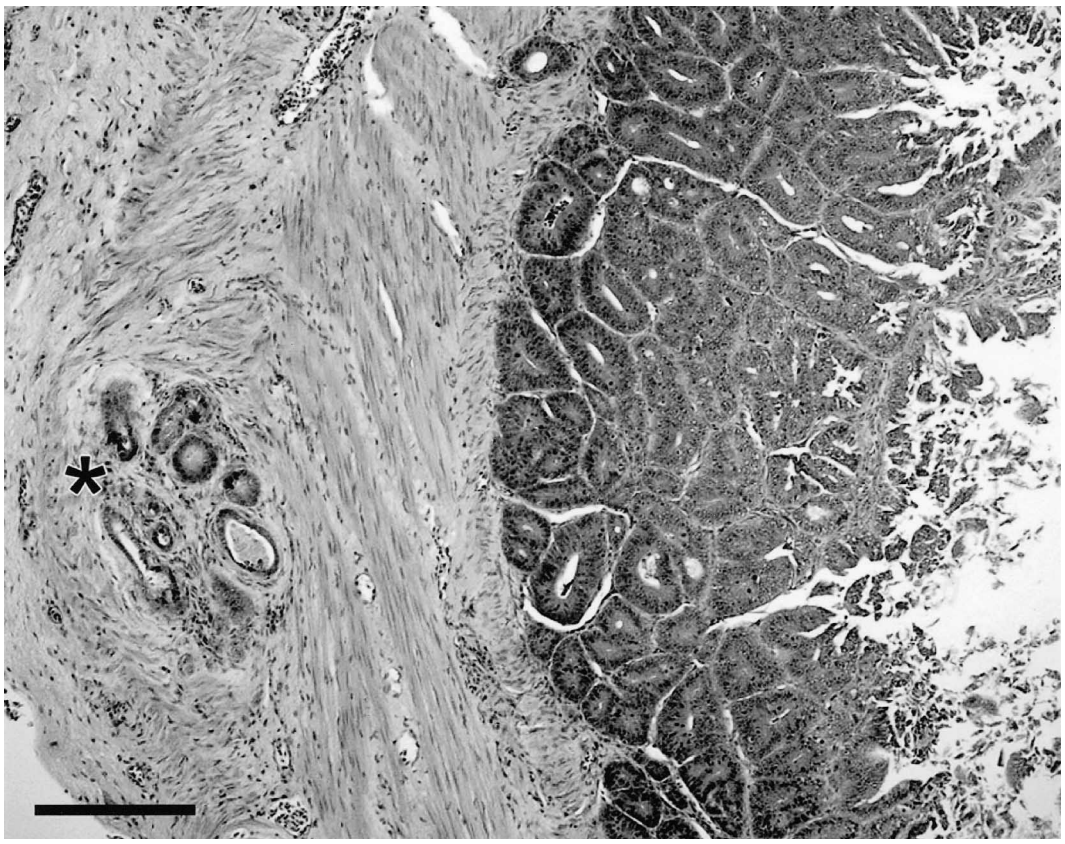

Fig. 3. Distal intestine. Adenocarcinoma infiltrating the intestinal muscular layer (*) and mucosal papillary hyperplasia. H&E. Bar = 50 μm.

Antemortem diagnosis of internal papillomatosis requires visual identification of abnormal structures and biopsy. In this case, neither the upper gastrointestinal contrast study nor the positive contrast fluoroscopic examination provided evidence of the papillomatous lesions. Endoscopic examination of the gastrointestinal tract was therefore not indicated. Similarly, no evidence of papillomatous changes was identified on the gross pathologic examination, though the cloaca was not examined clinically or at necropsy.

Treatment of internal papillomatosis depends upon the location within the gastrointestinal tract and the severity of the lesions. Surgical dissection, radiosurgery, cryosurgery, laser surgery, chemical cautery, and human interferon have all been utilized as therapies (7,10,26,27, 28,37). These treatment modalities are not consistently effective, and local recurrence is common. The use of autogenous vaccines has proven ineffective, and spontaneous regression of the papillomatous masses can occur (10,28, 30).

If internal papillomatosis had been diagnosed antemortem, treatment might have reduced associated morbidity, but given the multicentric nature of the lesions and the presence of overt malignancy, treatment would not likely have altered the outcome. This case demonstrates that, as with Amazon parrots and macaws, gastrointestinal adenocarcinoma and intrahepatic cholangiocarcinoma can occur concurrently with internal papillomatosis in peach-fronted conures.

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