Antemortem Diagnosis and Successful Long-term Management of Disseminated Intracoelomic Xanthogranulomatous Disease in an Eclectus Parrot (Eclectus roratus)

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Clinical Report

Antemortem Diagnosis and Successful Long-term Management of Disseminated Intracoelomic Xanthogranulomatous Disease in an Eclectus Parrot (*Eclectus roratus*)

Neta Ambar, Christoph Mans, and David J. Gasper

Abstract: A 12-year-old male eclectus parrot (*Eclectus roratus*) was referred for evaluation of coelomic distention. Computed tomography and blood work revealed coelomic effusion with free coelomic mineral-attenuating material and elevations in the bile acids and aspartate aminotransferase activity, respectively. Coelomic effusion was consistent with macrophagic inflammation with abundant intracellular lipids. Initial treatment with meloxicam resulted in minimal patient improvement. Disseminated xanthogranulomatous inflammation was suspected based on imaging and diagnostic laboratory results, which were consistent with those previously reported. Biopsy samples of liver tissue and intracoelomic masses confirmed this diagnosis. Treatment was initiated with prednisolone 1 mg/kg/d for 6 months, followed by 0.5 mg/kg/d for 3 months. Clinical improvement was assessed based on owner evaluation, plasma bile acid concentrations, and repeated computed tomographic scans. After 2 months of treatment, the owner reported improved behavior and appetite; this persisted throughout treatment and when the bird was reexamined 17 months following the cessation of steroid therapy. Bile acid concentrations were normal 10 months after the prednisolone therapy was discontinued. Diagnostic imaging showed minimal coelomic effusion 10 months after the last prednisolone dose was administered, with improved ventilation of the air sacs and static to improved dystrophic mineral foci. This report describes the antemortem diagnosis and treatment of disseminated coelomic xanthogranulomatous disease in a psittacine species, with an observed measurable therapeutic response.

Key words: disseminated coelomic xanthogranulomatous, avian, psittacine, eclectus, *Eclectus roratus*

CLINICAL REPORT

A 12-year-old male eclectus parrot (*Eclectus roratus*) weighing 0.4 kg was referred for evaluation of coelomic distention of 1-week duration. The owners reported the bird had a slight decrease in appetite and activity at home but otherwise exhibited normal behavior. A physical examination revealed a palpable fluid wave and coelomic distention. A computed tomographic (CT) scan and blood collection for a complete blood cell count (CBC) and plasma biochemistry profile (Vetscan Avian/Reptile Profile Plus, Zoetis, Parsippany, NJ, USA) were performed while the patient was under sedation with midazolam (2 mg/kg IM; West-Ward Pharmaceuticals, Eatontown, NJ, USA) and butorphanol (2 mg/kg IM; Torbugesic, Zoetis). The bird was fasted for 3 hours prior to sedation and blood collection. The CT scan (Fig 1A and B) revealed coelomic effusion with free coelomic mineral-attenuating material and secondary compression of the coelomic air sacs. A normal total leukocyte count ($14 \times 10^3/\mu L$, reference interval 9–$15 \times 10^3/\mu L$) with a mild heterophilia ($13 \times 10^3/\mu L$, reference interval 5.85–$8.75 \times 10^3/\mu L$) was observed.[1] Plasma bile acids were mildly elevated (73 μmol/L, reference interval 0–61 μmol/L), as was aspartate aminotransferase (AST) activity (468 U/L, reference interval 120–370 U/L).[2] Coelomic fluid was aspirated under ultrasound guidance and cytological findings were consistent with a transudate with macrophagic inflammation and abundant intracellular lipids but no infectious organisms. Meloxicam (1 mg/kg PO q24h; Metacam,

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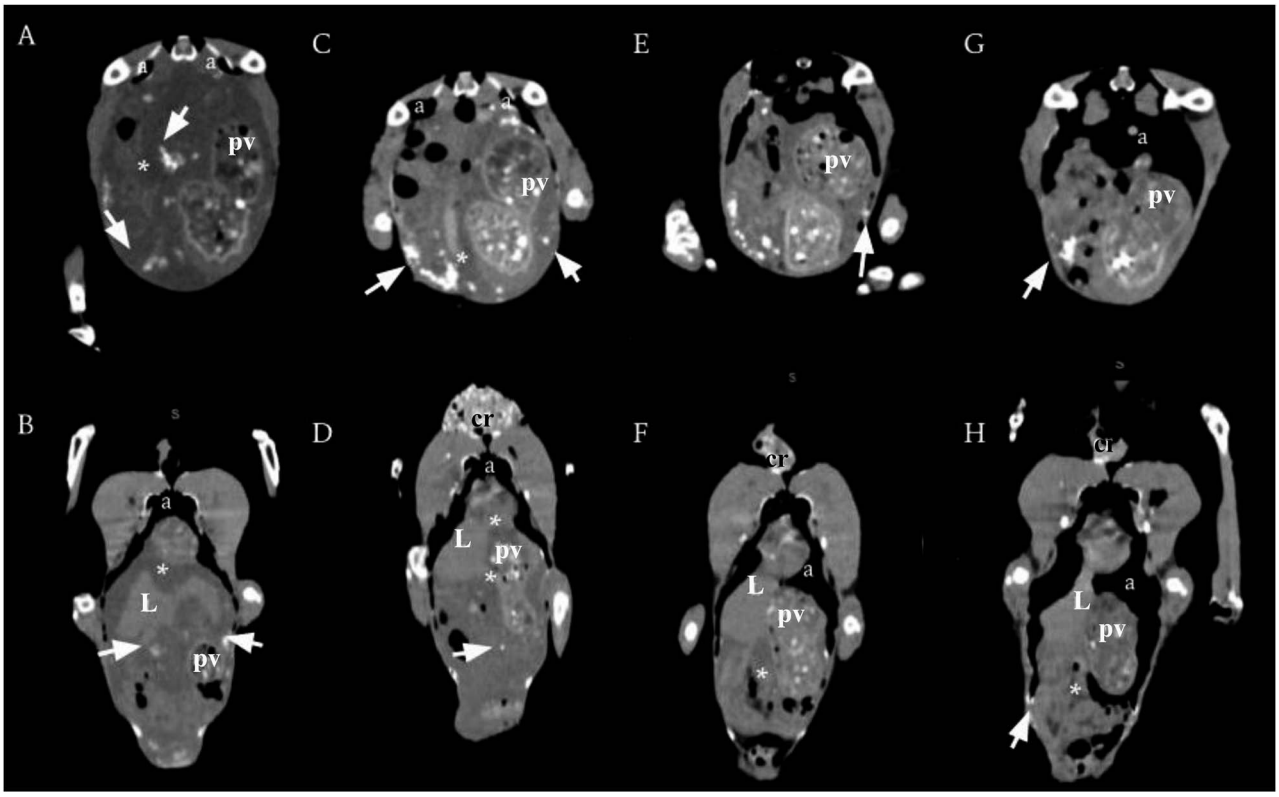

Figure 1. A transverse and dorsal multiplanar computed tomographic image of a 12-year-old male eclectus parrot (*Eclectus roratus*) with disseminated intracoelomic xanthogranulomatous disease, on initial presentation (A and B), 8 months later after treatment with meloxicam (C and D), 6 months after initiation of prednisolone (E and F), and 10 months after cessation of treatment (G and H). There is initially a large amount of coelomic effusion, with secondary compression of the coelomic air sacs (a), which progressively improved throughout the images following steroid administration. Multifocal coelomic foci of mineralization and mineralized coelomic masses (white arrows) are present in all images taken of the patient. Mineral-attenuating material is also present within the proventriculus (pv) and crop (Cr) after the initial scan, with a progressive decrease in proventricular distention. The liver (L) is normal in size and attenuation throughout.

Boehringer Ingelheim Vetmedica, St. Joseph, MO, USA) was prescribed for suspected chronic coelomitis, and dietary recommendations were made because the bird was noted to be on a suboptimal diet consisting primarily of fruits and vegetables (fresh and frozen cooked), a commercial cooked bean diet, and frequent offerings of scrambled eggs. No vitamins or supplements were provided, and owners occasionally offered nuts (primarily whole shelled almonds) and human food (eg, cheese, pizza crust) as treats. The dietary recommendations provided to the owner for the parrot included a diet of 75% pellets and 25% fresh fruits and vegetables, eliminating all human treats and seeds.

One month later, when reexamined, the coelomic distention was similar to that on the initial presentation but the parrot's appetite had improved. However, the bird also became more reluctant to move. Blood for a second CBC and plasma biochemistry profile was submitted at that time, with the results showing the bird had a mild heterophilic leukocytosis ($17.7 \times 10^3/\mu L$) but was otherwise unremarkable. The plasma bile acid concentration (68 μmol/L) and AST (517 U/L) activity were similar to what was reported in the first biochemistry profile.

When reexamined 6 months after the second hospital visit, the parrot's clinical condition had plateaued at home. A fluid wave and palpable liver margins were evident on the physical examination, consistent with hepatomegaly. Blood collected for a plasma biochemistry showed a severe elevation in bile acids (>200 μmol/L) and persistent elevation in AST (428 U/L) activity. Further evaluation of the diet revealed that although owners had eliminated eggs, cheese, and pizza, and increased the pelleted diet, the parrot was still being fed a cooked bean diet in addition to fresh fruits and vegetables. The patient was scheduled for a second CT scan, CBC, plasma biochemistry analysis, and endoscopic liver biopsy. All of the diagnostic tests were to be performed 1 month following this visit.

The patient was presented to the hospital 1 month later. At that time blood was collected and submitted for a CBC and plasma biochemistry analysis. A CT scan was performed on the eclectus parrot while under sedation as previously described. The images showed mildly improved coelomic effusion with persistent free coelomic dystrophic tissue mineralization and suspected generalized functional ileus (Fig 1C and D). The liver was normal in size and attenuation. Disseminated xanthogranulomatous inflammation was suspected based on the interpretation of the diagnostic images and CBC and plasma biochemistry results, which were consistent with those previously reported in eclectus parrots.[2] Plasma bile acids remained severely increased (280 μmol/L). The patient was premedicated with butorphanol (2 mg/kg) and midazolam (2 mg/kg) intramuscularly and mask induced with 4% sevoflurane (Dechra Pharmaceuticals, Fort Worth, TX, USA) prior to being intubated with a 3.5-mm uncuffed endotracheal tube and maintained on 3% sevoflurane throughout the procedure. A 24G intravenous catheter was placed in the left medial metatarsal vein, and a balanced electrolyte solution was administered intravenously at a rate of 3 ml/kg/h. Anesthetic monitoring of the parrot during the surgical procedure was performed by Doppler, electrocardiography, and capnography. A midline incision was performed to allow for endoscopic evaluation of the liver. However, a ~2-cm-diameter well-circumscribed yellow mass was immediately exposed and was found to adhere to the liver and surrounding tissue. The mass was bluntly dissected with care and removed from the body. On the cut surface, the mass was filled with a yellow/white liquid; this material was submitted for bacterial culture and antimicrobial sensitivity testing. Biopsy samples from the liver and the removed mass were submitted for histopathological evaluation. The anesthetic event was uneventful throughout the endoscopic procedure, with the patient having stable vital parameters and ventilation manually assisted at 10 breaths per minute. Following the endoscopic evaluation and collection of tissue samples, the patient had an uneventful recovery. The bird was prescribed meloxicam (1 mg/kg PO q24h × 7 days) and tramadol (15 mg/kg PO q12h × 5–10 days prn; compounded by the University of Wisconsin–Madison Veterinary Pharmacy) and discharged the following day. Meloxicam was only given for 3 days to allow for a washout period prior to the initiation of steroid therapy, pending biopsy results.

No growth was evident on the bacterial culture from the yellow tissue mass. On histopathology (Fig 2), the coelomic mass was composed of adipose tissue that was severely disrupted by coalescing nodular masses of eosinophilic and karyorrhectic necrotic debris; large numbers of epithelioid macrophages with discrete round cytoplasmic vacuoles (lipid) and progressively fewer multinucleated giant cells; intact and fragmented heterophils; and acicular cholesterol clefts. Occasional aggregates of mineral were scattered within the nodules, and the nodules were encircled by rims of granulation tissue and fibrosis, entrapping small clusters of plasma cells and lymphocytes. Microscopic changes in the liver were limited to a moderate diffuse increase in the number of sinusoidal macrophages that occasionally formed small periportal nodules and mild multifocal extramedullary hematopoiesis. Based on these findings, the patient was prescribed prednisolone (1 mg/kg PO q24h × 30 days; Hi-Tech Pharmacal Co Inc, Amityville, NY, USA) and placed on a diet composed of 75% pelleted diet with the remainder consisting of fresh fruits and nonstarchy uncooked vegetables.

One month later, upon reexamination, the parrot had a decrease in coelomic effusion (no palpable fluid wave), persistent coelomic distention, and no weight loss from the previous visit. The bird's appetite, activity, and energy levels had improved. Blood submitted from the patient during this visit was submitted for a CBC and plasma biochemistry analysis. The results of the CBC and plasma biochemistry analysis showed a mildly increased leukocytosis ($23.7 \times 10^3/\mu L$), characterized by a heterophilia ($19.7 \times 10^3/\mu L$), and a decreased plasma bile acid concentration (197 μmol/L). Terbinafine (15 mg/kg PO q12h 30 days; compounded at the University of Wisconsin–Madison Veterinary Pharmacy) was added to protect against secondary fungal infection.

One month later, the bird's energy levels had improved significantly, and it was vocalizing on a regular basis. When physically examined, the patient was bright, alert, and responsive and had no palpable coelomic effusion. However, its coelom remained distended. Blood collected at that time for a CBC and plasma chemistry analysis revealed the continued presence of a mild leukocytosis ($20.7 \times 10^3/\mu L$), characterized by heterophilia ($14.1 \times 10^3/\mu L$), and a further reduction in the plasma bile acid concentration (99 μmol/L).

When reexamined 4 months later (6 months after initiating prednisolone treatment), CBC and plasma biochemistry analyses submitted at that time indicated the leukocytosis had resolved ($12 \times 10^3/\mu L$) and the bile acid concentration (54 μmol/L) had further decreased. The owners reported that the bird continued to have an improved appetite, was increasingly exhibiting normal behaviors, and was becoming more

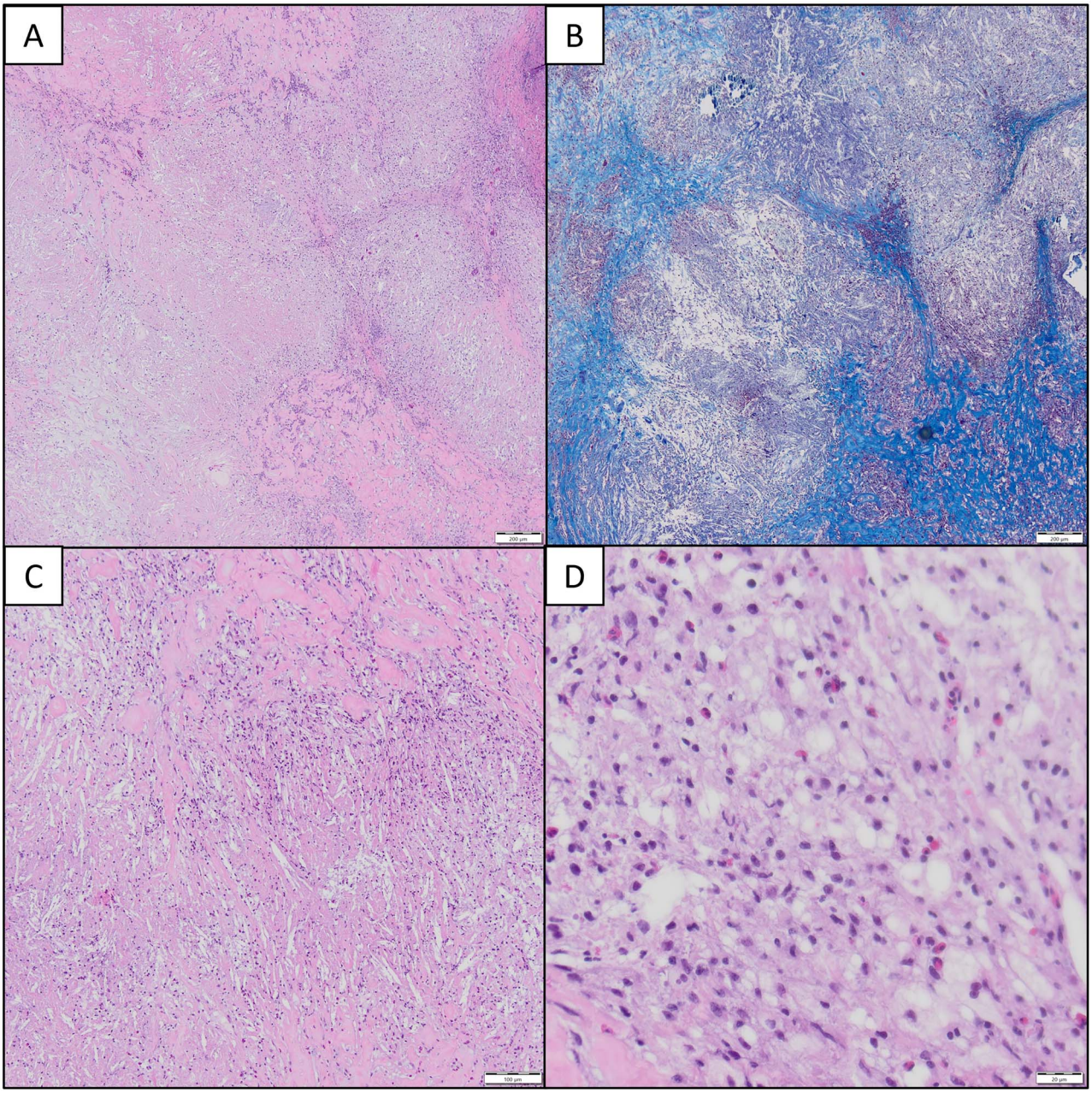

Figure 2. Histopathology panel. Coelomic connective tissue. (A and B) Coalescing nodular masses of inflammatory cells separated by collagenous tissue (hematoxylin and eosin; bar = 200 μm). (B) Collagenous tissue stains blue and outlines areas of inflammation (trichrome; bar = 200 μm). (C) Higher magnification of inflammatory cells and myriad linear cholesterol clefts (hematoxylin and eosin; bar = 100 μm). (D) Higher magnification of epithelioid macrophages with vacuolated cytoplasm and scattered heterophils (hematoxylin and eosin; bar = 20 μm).

interactive. Computed tomographic images were obtained during the visit using the sedation protocol described previously. The CT images showed marked improvement in the coelomic effusion and no change in the amount of free coelomic dystrophic tissue mineralization, consistent with disseminated xanthogranulomatous inflammation (Fig 1E and F). Atherosclerotic lesions were suspected based on a mild amount of mineral-attenuating material along the intraluminal wall of the descending aorta, brachiocephalic trunk, and subclavian arteries bilaterally. These vascular changes were not present on the 2 previous CT scans. Based on the findings of this visit, the prednisolone dose was decreased to 0.5 mg/kg PO q24h × 90 days.

Upon reexamination 3 months later, the bird remained bright and active, with a good appetite. The physical

examination results had not changed from the previous visit and the plasma biochemistry results were static (bile acids 66 μmol/L). Based on the results of this visit, all treatments were discontinued.

When reexamined 10 months following the discontinuation of all treatment, the bird was reported to have good energy levels and normal appetite and behavior. The physical examination during this visit was unremarkable, and the coelom was not distended. The plasma biochemistry results showed that the bile acid elevation (<35 μmol/L) had resolved. A CT scan was performed at this visit with the patient sedated as previously described. The CT images showed scant coelomic effusion with expansion of the air sacs, similar dystrophic mineralization, no change in the suspected atherosclerosis, and increased body condition (Fig 1G and H). Verbal communication with the owner 17 months following the discontinuation of all treatment found that the parrot continued to have improved behavior, activity levels, and appetite with no recurrence of clinical disease signs.

DISCUSSION

Xanthogranulomatous disease (XGD), also called xanthogranuloma (XG), xanthomatosis, or xanthoma, has been inconsistently defined. The unifying feature of XGD is the histological appearance of the disease, which is characterized by granulomatous inflammation and composed of foamy, lipid-laden macrophages commonly accompanied by infiltration of acute or chronic inflammatory cells.[3–9] Although XGs are generally benign, depending on the location and organ involvement, they may be of clinical concern.[7,8] Moreover, the inflammatory process initiated may in and of itself be of clinical significance.[8] Xanthogranulomatous disease is a collection of syndromes for which precise pathophysiology has not yet been elucidated.[3–9] Theories include the development of lesions due to altered systemic lipid metabolism, as a result of local cell dysfunction, or due to an underlying infectious etiology.[5,6,8,10] Alterations in lipid metabolism, including hypercholesterolemia, hyperlipidemia, hypertriglyceridemia, hyperlipoproteinemia, and hyperchylomicronemia, and lipid dysregulation have been implicated as a risk factor for XGD in both humans and animal models.[6–9,11,12] Dyslipidemic states can be idiopathic or secondary to endocrine disorders such as diabetes mellitus, feeding diets with excessively high lipid content, fat-related toxic hydrocarbons, or due to an infectious etiology.[5,11–13] Experimentally induced XGD was achieved with dietary cholesterol in both quail and rabbit models, lending support to the dietary and metabolic component of this disease process.[10,11] Additionally, there have been several reports of both cutaneous and systemic XGD in hyperlipidemic cats.[12] However, it is important to note that XGD has also been reported in normolipidemic animals and humans.[6,7,12,14] In another report, infection with psittacine adenovirus 2 was proposed as potentially eliciting a dyslipidemic state in red-crowned parakeets (*Cyanoramphus novaezalandiae*), which led to the development of XGD in some individuals.[5] An additional suggested cause of XGD development involves local trauma (cell dysfunction).[6–9,13,15] Lipid phagocytosis occurs secondary to suppuration, hemorrhage, or necrosis following the traumatic incident. Phagocytosis by the inflammatory cells (histiocytes) adjacent to the damaged sites leads to the formation of lipid-laden (foamy) xanthoma cells.[6–9] This theory more readily explains single cutaneous XG or XG in areas of high tension (ie, tendons as observed in humans) but less readily systemic or disseminated XGD. Regardless of whether the inciting cause is due to alterations in lipid metabolism or secondary to trauma, once macrophages have phagocytosed lipids, a similar inflammatory process occurs.[7–9,12] Ultimately, progressive granuloma formation, dense fibrin, collagen deposition, and replacement of the infiltrated tissue by abundant xanthoma cells lead to organ destruction, with a possible extension of the XG inflammation to the surrounding structures.[7,15,16]

Xanthogranulomas have been reported in a variety of avian species,[3–5,10,13,14,17–23] as well as in mammals,[11,24–33] reptiles,[34,35] amphibians,[36] and humans.[6–9,15,16,37–43] Although cutaneous lesions are most common, extracutaneous and systemic lesions have also been reported. Locations for these lesions include periarticular, periosseous, and mucosal sites; the nervous system; coelomic or abdominal cavities; and ocular or periocular tissues.[10–41] In humans, XGD consists of a variety of rare and diverse syndromes of unknown etiology. These syndromes are generally separated by signalment, clinical appearance, the presence or absence of hyperlipidemia, histopathological appearance, and immunohistochemical phenotype.[6,7,9,38,42] Histologically, the lesions are remarkably similar to each other and to those observed in veterinary species.[9] There have been several attempts to categorize XGD in humans, but the results of the classification efforts often lack clear distinctions and a defined clinical presentation and picture. Juvenile xanthogranulomas (JXGs) are the most common non–Langerhans cell histiocytosis that occurs in humans, and they typically affects infants. The lesions present as red-to-yellow focal and occasionally multifocal cutaneous nodules on the head, neck, and upper torso. The nodules

spontaneously resolve over 1–5 years and occur in normolipidemic states.[6,7,9,37,38] Although the cutaneous form is the most common, extracutaneous lesions and systemic JXG have also been reported.[37] Disseminated or systemic JXG may be associated with more significant morbidity, requiring aggressive treatment intervention.[7,15,37–39] In adults, XGD is rare, with multiple syndromes associated with different outcomes and spontaneous resolution being less likely.[39] Of note are hyperlipidemia-associated xanthoma, adult-onset XG, necrobiotic XG, periocular XG, and Erdheim-Chester disease.[6,7,38] Adult-onset XGs are most similar to JXG and are usually self-limiting lesions that are not associated with any systemic disease.[6,7] Xanthogranulomatous mesenteritis is characterized by chronic inflammation and fibrosis of the mesentery. The etiology is unknown but is generally associated with other idiopathic inflammatory disorders.[43] In humans, immunohistochemistry has been beneficial in categorizing lesions and targeting treatment; however, this has not been utilized in veterinary medicine.[43]

Previous reports of XGD in avian species typically describe cutaneous lesions[19,22,23] or lesions confined to a single organ system, with clinical signs associated with compromise of that organ system.[19–23] Disseminated coelomic xanthogranulomatosis has only recently been described in eclectus parrots, budgerigars, and red-crowned parakeets.[3–5] The 3 psittacine cases were characterized grossly by accumulations of irregular, soft, tan to yellow amorphous material distributed over the visceral surfaces of coelomic organs and parietal surfaces of the body wall.[3–5] Histological evaluation revealed xanthogranulomatous inflammation with phagocytized and extracellular lipid, necrosis, cholesterol clefts, fibrosis, and mineralization.[4,5] All of the birds presented with nonspecific clinical signs, including weight loss, decreased appetite, regurgitation, and difficulty breathing.[3–5] Clinical and imaging features included coelomic effusion, multifocal heterogeneous focally mineralized coelomic nodules/masses, hepatomegaly, atherosclerosis, and diffuse enteropathy.[3] Plasma biochemistry results commonly showed an elevation in bile acid concentration, hypercholesterolemia, and hypertriglyceridemia.[3,4]

The case presented here is the first report of antemortem diagnosis and treatment of disseminated XGD in an avian species. Computed tomographic imaging revealed multiple mass-like structures with dystrophic mineralization similar to that previously described in a psittacine species.[3] Mineralization, in this case, was likely due to fat necrosis, as seen in human xanthogranulomatous cholecystitis.[40] The eclectus parrot described in this report also had an elevated bile acid concentration and AST activity, which can indicate muscle or hepatic damage, including inflammation; this was present in this case.[44] A lipid panel was not performed in the current case and would have provided useful information regarding the underlying pathological process. On postmortem examination, all birds evaluated in previous publications had evidence of disseminated coelomitis characterized as xanthogranulomatous inflammation.[3,4] Fluid analysis of the coelomic effusion on initial presentation in the current case was suggestive of an active inflammatory process with primary macrophages containing abundant intracellular lipids, consistent with xanthomatous inflammation. This finding was similar to that reported in a previous case where an effusion was found to be a modified transudate with primarily vacuolated macrophages.[4] The mineralized coelomic masses were present on the initial CT along with the effusion, but only a moderate elevation in bile acids was observed at that time, suggesting that XG formation likely preceded hepatic disease. This is consistent with the suggested pathophysiology in which the inflammatory process associated with the xanthoma formation contributes to surrounding organ dysfunction.[6,7] In the previous reports, most parrots had evidence of cholangitis and cholangeohepatitis on postmortem examination.[3,4] Atherosclerosis was a common comorbidity in the previous case and was similarly identified through CT imaging in the current case, although not on initial presentation.[4]

No infectious cause could be identified with special stains for fungi and acid-fast bacteria in the current case, which is similar to previous reports.[3–5] Although a variety of factors are likely to play a role in the development of XGD, dyslipidemia and diet have been suggested previously as primary contributing factors.[3,4] As previously mentioned, diet-induced XGD has been reported in humans (hyperlipidemia-associated xanthoma) and has been experimentally induced in both rabbits and Japanese quail (*Coturnix japonica*).[6,10,11] In a strain of quail susceptible to dietary cholesterol–induced atherosclerosis, xanthomatous lesions developed after 4 weeks of exposure to a high-cholesterol diet, with over 85% of birds affected within 12 weeks. Elevations of serum cholesterol and triglycerides were observed within 4 weeks.[10] In a study involving broiler chickens, the fatty acid composition of diet directly impacted lipid metabolism, with those fed saturated fatty acids having a greater amount of abdominal fat.[45] Although avian lipid metabolism is generally similar to that of mammals, the avian liver plays a more robust role in lipogenesis, with up to 90% of synthesized fatty acids coming from the liver.[17,44] In humans, birds, and cats, low-density

lipoprotein has been implicated in the formation of XG and other diseases associated with abnormal lipid deposition, such as atherosclerosis or lipidosis.[3,4,12,44] In future clinical cases, evaluation of plasma cholesterol and triglyceride, as well as lipoprotein panels, is recommended to assess the role of lipid dysregulation in the development of this disease and evaluate its utility as a measure of response to therapy.[46] It is important to note, however, that interspecies as well as intersex and seasonal variations in cholesterol, triglyceride, and lipoproteins exist and may limit the ability to draw clinically relevant conclusions from these parameters. Species- and sex-specific reference intervals for eclectus parrots would be desirable to better manage risk factors for the development of XGD in future cases of this condition in this psittacine species.

Cutaneous or single XG surgical resection is recommended for treatment in both humans and animals.[3,6,19,38] To the authors' knowledge, no successful treatment protocols have been reported for disseminated XGD in psittacine species. Previous attempts to treat disseminated XGD in birds with diet modification and supportive care, including antimicrobial drugs, antifungal agents, hepatoprotectants, promotility agents, and nonsteroidal anti-inflammatory medication, were not successful, with survival ranging from 7 days to 1.9 years.[3,4] In humans, when surgical intervention is difficult (eg, orbital XG, disseminated/systemic disease), treatment is focused on the immune response. However, options and outcomes are variable due to the diversity of clinical syndromes. Reported immune targeting protocols that have been reported to have relative treatment success include corticosteroids, usually in combination with an immunomodulator or chemotherapy treatment, similar to that established for Langerhans cell histiocytosis.[6,7,15,37,38,40,47] Treatment is generally long-term, lasting from months to a year, depending on the syndrome and protocol used.[39,47] Corticosteroid therapy is generally continued until the resolution of inflammatory signs, with immunomodulatory or chemotherapy continued for longer at maintenance levels.[15,39] A review of systemic JXD found that the most effective treatment protocols combined the use of corticosteroids and vinca alkaloids. In that review, no cases used corticosteroid therapy alone, and protocols without corticosteroid administration had much lower success rates.[47] In another case report, JXD resolved with corticosteroid use but recurred after therapy was discontinued. An additional immunomodulator was then added for complete resolution.[36] In cases of xanthogranulomatous mesenteritis, treatment has similarly been successful with corticosteroid and immunosuppressive agents.[43]

In the current case, corticosteroid therapy was initiated as a first-line treatment because a nonsteroidal anti-inflammatory medication was not sufficient to treat the underlying inflammatory process. Birds in general are thought to be steroid-sensitive species, yet there are few reports in the peer-reviewed literature documenting this belief. Reported consequences of steroid use include immunosuppression, which leads to heightened susceptibility to bacterial and fungal infections, as well as delayed wound healing, diabetes mellitus, hepatic disease, and gastrointestinal ulceration.[48] Furthermore, there is concern that steroids could suppress the function of respiratory macrophages, which are critical in defending against fungal infections (eg, aspergillosis).[48] A case series published about blue-fronted Amazon parrots (*Amazona aestiva*) underscored the severity of the mycotic airsacculitis that can develop following prolonged high-dose (2 mg/kg) corticosteroid treatment.[49] Despite the gravity of these findings, there is a paucity of pharmacokinetic, efficacy, or safety studies on the use of steroids for the treatment of diseases in avian species. It is likely that adverse effects of corticosteroids in birds are dose related, similar to other species, and such information is crucial for the safe and appropriate use of steroids in birds. The scarcity of information necessitates that clinicians weigh the potential benefits of steroid therapy against the risks, and mitigate these potential adverse effects with close monitoring of complete blood counts and plasma biochemistry analyses, as well as the use of antibiotic and antifungal therapy, when necessary.[48] In the current case, the patient was monitored closely for signs of secondary opportunistic infections and was placed on terbinafine once long-term corticosteroid use was initiated. No adverse effects were observed with the use of prednisolone at a 1 mg/kg dose for 6 months and a 0.5 mg/kg dose for an additional 3 months.

In humans with hyperlipidemia-associated xanthoma, treatment of the underlying condition may lead to the resolution of lesions.[6] The role of diet in the current case is uncertain, as dyslipidemia was not determined. Nonetheless, prior research suggests a possible dietary factor in the manifestation of XGD.[3,4] Notably, in the previously mentioned case series, 3 out of the 5 eclectus parrots diagnosed postmortem with disseminated XGD were on a high-fat diet, including eggs.[4] Although dietary modifications were implemented to reduce fat, the study did not detail the specifics of these diet changes or their timing in relation to mortality.[4] In the current case, the parrot was initially on a nontraditional diet that was composed of bean and legumes along with high-fat items and animal products

in which cholesterol was present. Similar to previous cases, diet modifications were instituted 6 months prior to the definitive diagnosis, a period that saw a worsening clinical and biochemical profile. A lipid panel could have been useful in gauging the effects of eliminating fats from the diet but was not performed. Despite the diet modifications made, beans and legumes remained a staple in the parrot's diet throughout the 6 months before the bird was diagnosed with XGD. Unfortunately, there are limited data on the capacity of eclectus parrots to digest beans and legumes. Upon diagnosis, the bird easily transitioned to a fully pelleted diet supplemented with low-sugar fruit and vegetables with no starch.

Eclectus parrots are considered frugivores in the wild, with their diet primarily consisting of fruit pulp.[50] This has led to a multitude of diet recommendations on the internet, steering many pet owners towards unconventional or homemade meals. Such trends may inadvertently subject the species to nontraditional or high-fat diets. Given the increased prevalence of this disease in eclectus parrots, more research into dietary requirements, lipid metabolism, and genetics is necessary to gain further knowledge regarding the pathophysiology and highlight preventative measures for XGD. Current findings suggest a significant dietary component in the development of this disease process. However, as with human syndromes, the emphasis of treatment usually lies in modulating the immune response and reducing inflammation, which proved to be a vital component of the treatment's success in this case.

Disseminated coelomic xanthogranulomatosis is a rare condition in avian species, and it appears that eclectus parrots may be predisposed to developing this condition.[3,4] Further research is required to better understand the pathophysiology, patient predisposition, diagnosis, and treatment options for XGD. In addition, a better understanding of the nutritional requirements and lipid metabolism of psittacine species, specifically eclectus parrots, could be beneficial in optimizing husbandry and patient care. To the authors' knowledge, this is the first report describing an antemortem diagnosis and treatment of disseminated coelomic XGD in a psittacine species, with a measurable response to therapy observed.

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