Differential Diagnosis of the Suddenly Ill Mature Pet Bird

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The U.S. pet bird market has changed markedly in the last several years from an import-based supply to avicultural-based supply. The pet bird as a product has changed. New nutritional problems have emerged, adding to classic nutritional diseases. Mature pet birds of today share the wild bird tendency to mask clinical signs of disease. These birds share some of the disorders found in imports, in addition to some new syndromes.

Many syndromes of acutely ill mature pet birds are chronic in nature. Acute manifestations of this chronic disease will induce the owner to present the patient for treatment. The clinician should develop differential diagnosis schemes, based on species, age, clinical history, physical examination, and the initial diagnostic assessment.

Nutrition-Associated and Metabolic Disorders

While most everyone agrees that a pet bird should have good nutrition, there continues to be a wide disparity of information and practices among veterinarians, retailers, and bird owners. A seed predominant psittacine diet will typically provide a ration with excess fat calories vs aerobic exercise levels, incomplete amino acids, and inadequate calcium, zinc, Vitamin A, Vitamin D, and Vitamin K. Supplementation with North American fruits typically provides little enhancement to the imbalances mentioned here. Low-level, long-term exposure to seed-laden mycotoxins probably contributes to hepatic insults.

Clinical effects of nutritional disease in the mature pet bird will be apparent in months to years. Obesity can contribute to reduced reproductive success, difficult defecation, reduction of air sac tidal volume, and be associated with hepatic lipidosis. Vitamin A deficiencies can present with classic metaplastic changes of the oral cavity, but it is important to realize the numerous long-term effects due to absence of this nutrient. These effects include renal tubular degeneration, thought to be associated with budgerigar renal disease and gout. Vitamin A enhances the viability of epithelial linings. Lack of this nutrient will result in skin changes, changes in the airway epithelium, and alterations in alimentary tract linings.

Calcium, which is absent from most seeds and grains, can be regularly ingested from supplemental sources. Calcium will not be absorbed unless either adequate exposure to unfiltered solar radiation occurs or adequate Vitamin D3 is included in a formulated diet or vitamin supplement. The African Grey parrot cannot apparently mobilize bone calcium with the same ease of many other birds. Clinical effects include hypocalcemia and neurological signs (seizures, weakness). Hypocalcemic tetany can occur in other psittacine species, but the duration of malnutrition in non-ovulating birds may extend for years before clinical signs occur. Pet birds who are laying eggs regularly without D3/calcium supplementation, are very susceptible to eggbinding and related problems. Pathologic fractures may be evident upon physical exam or radiography.This speaker has seen pet laying cockatiels present with acute lameness showing fractures in three limbs.

Mineralization of soft tissues of mature pet birds, particularly kidneys presents more frequently due to the widespread usage of formulated diets. While these types of products have much merit in a feeding program, some formulations fed exclusively to some species and individuals, result in renal tubular mineralization. The pathologist's perspective suggests excessive Vitamin D3 in the ration, but controlled psittacine studies don't exist. Unpublished trials with commercial formulas containing very low D3 content were associated with renal mineralization in fledgling macaws. Other clinicians feel that excessive protein will result in these changes. Poultry studies employing high protein intake are divided on how such a diet effects renal pathology. Unfortunately renal mineralization cases frequently are found dead or exhibit minimal clinical signs until severe pathology is present. Psittacine birds presenting ante-mortem with developing renal mineralization will typically exhibit marked polyuria and polydipsia. Plasma uric acid levels typically show a moderate elevation. Effects

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aiding recovery include replacing the formulated product with carbohydrates. Allopurinol may provide further assistance. Until these products are known by the clinician to have a satisfactory track record in a with a particular species and set of circumstances, it is prudent to provide up to 20% of the caloric content in non-formulated diet. Carbohydrates are most satisfactory. Many clients will feed fruits and vegetables in addition to a formulated diet. It must be realized that the caloric density of these products, relative to moisture content, is low. Additionally, the high phosphorus content of fruits may contribute to further imbalance.

Excess lipids in the pet bird diet are often associated with medical problems. Birds are well-designed physiologically to use fat aerobically as a sustained fuel during flight. Most pet-birds, though, could be characterized as "couch potatoes," lacking either the interest or opportunity for safe exercise. The target organ for this excess lipid appears to be the liver. Seeds are the most common source of excess lipid, but nuts, animal fats, junk food, fast food and other high-fat human source products can contribute. There has been a recent trend among some formulated diet companies to increase the fat content of their products, presumably to increase caloric density and palatability. On a dry matter basis, these products are still fat limited compared to seeds and nuts.

Hepatic lipidosis, diagnosed by antemortem liver biopsy or postmortem histopathology, is characterized by loss of parenchyma, replaced by fat. Plasma bile acids will elevate from slight to moderate to severe in a non-linear correlation to histopathologic severity. Poultry studies have demonstrated that a lack of lipotrophic agents contribute to hepatic lipidosis. This speaker's long-term clinical experience suggests that lipotrophic agents are useful for pre-terminal therapy. Currently, selection of a maintenance or therapeutic formulated diet provides for inclusion of these lipotrophic agents. This speaker has also used L-thyroxine to "turn up the thermostat" on these cases. While thyroxine therapy was discredited for human lipidosis therapy some time ago, poultry research suggests such therapy has merit in birds.

Other chronic liver pathology is common, particularly in cockatiels and Amazon parrots. Histologic changes include fibrosis, cholangitis, bile duct proliferation, hemosiderosis, and fatty change. Enzyme elevation is absent except in cases where current hepatocellular damage and leakage is underway. Plasma bile acids are often helpful in identifying clinically normal birds who need a liver biopsy. Sadly, pet birds aged 5-15 years, present frequently with terminal (histological) liver disease. These disorders have often been developing for years, without any obvious indication to the owner. The clinician attending such a case may have recently pronounced the bird "healthy" based on a recent physical and checkup laboratory work. Clinical features of chronic liver disease include long history of poor diet, obesity, poor feather, abnormal beak growth, feather color change. Acute manifestations of chronic liver disease include somnolence, anorexia, yellow urates, head tilt, weakness, and anemia. Leukograms and enzymatic profiles in crisis cases are variable from normal to elevated.

Atherosclerosis presents as a problem particularly in slightly older parrots of the Amazona genus. Clinical signs can include "sudden death." Pathology findings may include complete occlusion of the internal carotid arteries. Cardiac vessel atherosclerosis is not a common problem. Vessel changes may be evident radiographically or via ultrasonography. No controlled studies in pet birds correlate plasma cholesterol and atherosclerosis. Treatment regimens which may help promote reversal of this pathology are similar to those for hepatic lipidosis.

Gout presents in birds in two not entirely distinct syndromes: visceral and articular. Articular gout is most common in older budgies and is thought to be associated with renal tubular changes due to Vitamin A deficiencies. High plasma uric acid levels and urate joint tophi render a very guarded prognosis, even with the use of anti-gout medications. Visceral gout is characterized by large amount of urates deposited in serosal areas, particularly the pericardium and peritoneum. Uric acid levels are frequently normal with this condition, with a common history of "sudden death." Nephrotoxicity, including Vitamin D3 can contribute to cause this condition.

Toxicoses

Lead toxicosis is common in most parts of the country, more so in long-settled urban areas containing more exposure to old lead paint. Modern homes, though, also contain a variety of lead-containing materials, including costume jewelry, grout, putty, stained glass art, hunting and fishing materials, and curtain weights. Amazons and cockatiels are particularly predisposed to lead ingestion. History is very important in regards to confinement and exposures of the patient. Clinical signs can include acute illness, neurological signs, and change in droppings.

Hemoglobinuria is most common in Amazons, while hematochezia is infrequently reported in pet birds. The most common droppings change is green to tan change in the urates and a change in the greenish color of the feces. A markedly responsive anemia, with erythrocytic ballooning is evident in a majority of the cases. Radiography is not consistently diagnostic: toxicosis can occur in the absence of metal particles and the finding of metal-dense particles is common in the absence of lead toxicosis. Blood level levels are diagnostic. Other assay methods (ALAD etc) are used in wildlife medicine, but may not be available to the practitioner. Lead toxicosis is often chosen as incorrect rule-in, based on clinical signs alone. This disorder is also missed because inadequate history collection. In California, the most common differential for a mature pet bird with neurological signs is liver disease.

Zinc toxicosis has been increasingly recognized as a problem. Housing in galvanized cages does not assure toxicosis, but poultry studies find that zinc deficiencies are prevented. New cages and pennies are commonly incriminated as sources of toxicity. Hematologic changes and clinical signs (lethargy, PD/PU, diarrhea, weakness) are vague. The target organ for damage and death is the pancreas. Blood zinc levels are diagnostic.

Rarely is there an indication for pesticide application to pet birds. The mostly unnecessary mite and lice products containing primarily pyrethrins. Clients, goaded into believing they have a mite problem, have been known to use potent organophosphate pesticides on their bird, resulting in classic toxicity. Cholinesterase testing can be performed; history is most important. Atropine, supportive care and specific antidotes are indicated.

Iatrogenic toxicosis can occur due to improper drug usage. Clients get their hands on products and try them. Older aminoglycosides, including gentamicin, are contraindicated, due to a narrow therapeutic index and the high likelihood of renal tubular necrosis. Corticosteroid usage should be used judiciously: avoid topical products in small birds. Systemic corticosteroids should be limited to acute trauma, hypovolemic “shock-like” conditions, and suspected endotoxemia. Routine, indiscriminate use of corticosteroid is dangerous, due to immunosuppression, secondary infection and death.

Neoplasia

Budgerigars are well-known “tumor factories.” Recent epidemiologic evidence in the wild and retroviral DNA assays point to a viral basis for some budgerigar tumors. Tumor incidence peaks at 3-4 years of age and again at 7-9 years. A classic presentation is the renal carcinoma or variant. The budgie will present with unilateral non-weight bearing lameness, due to vertebral nerve root invasion. Abdominal masses can be seen in this and other budgie tumor types. In addition, fibrosarcomas and squamous cell carcinomas are quite common. The challenge to the clinician is to differentiate generally untreatable, malignant tumors from treatable alimentary, reproductive, and other multi-organ disorders. A viral basis for neoplasia in other pet bird species has not been uncovered.

Reproductive Tract Disorders

Unwanted egg-laying in unwanted cockatiels, budgies, and lovebirds is a very common presentation. Malnutrition and co-existing medical conditions exacerbate this condition. Physiologically, normal clinical changes in the egg-laying pet bird include radiographic endosteal new bone formation, lipemia, “relaxed” abdominal tissue, hyperproteinemia, nesting behavior, and changes in droppings quantity or size.

Reproductive tract-associated problems include egg-binding, yolk peritonitis, pathologic fractures, and hypocalcemia. Under-calcified eggs may not be visible radiographically, but can be imaged by ultrasound or confirmed with adominocentesis. Yolk peritonitis is characterized by a leukocytosis, variable abdominal distension and fluid, and variable cytologic changes. Cytologic infiltrates range from primarily mesothelial cells to a mixed inflammatory response with visible bacterial organisms. One consistent cytologic change is a “fatty” background on the smear, viewed microscopically.

Respiratory Tract Disease

Cockatiels have a predilection for millet seed aspiration, resulting in acute, noisy dyspnea. Differential diagnosis includes bacterial/fungal bronchopneumonia, fluid air sacculitis from yolk peritonitis leakage. Any pet bird with severe dyspnea benefits from the installation of an air sac breathing tube and oxygen therapy. Removal of millet seed is difficult and involves, passing, under anesthesia a tracheal tube to the syringeal area for aspiration.

When airway distress is not due to a foreign body, tracheal aspiration is a valuable diagnostic tool for cytology and culture. When possible, this procedure should be preceded by endoscopic examination.

Respiratory hypersensitivity syndrome is common in *Ara* macaws particularly when exposed to powder-down producing parrots such as cockatoos and African Grey parrots. Acute therapy involves oxygen and steroids. Chronic therapy involves air quality management and drugs which can safely minimize the allergic response. The disease tends to be progressive, resulting in pulmonary interstitial fibrosis.

Pinpoint aspiration pneumonia can carry-over from the weaned to the mature pet bird. In the clinically normal bird, only leukocytosis will be evident. A bacterial granuloma or subsequent development of an aspergilloma may occur.

"Corncob disease" is a result of long-term exposure of the pet bird to organic, fermenting cage litters including corn cob, walnut shell, leaves, and wood shavings. Unfortunately, some retailers enable the lazy bird owner by encouraging non-changeout of these products for extended periods. Massive fungal spore exposure can induce systemic mycoses in even the immunocompetent patient. Susceptibility to infection is greatly enhanced by reduced defenses, including pre-existing infections and Vitamin A deficiency. Prognosis for recovery to acute spore-overdose aspergillosis is poor. Successful aspergillosis therapy usually includes identification and debulking of the fungal lesion(s), followed by topical and systemic drugs.

Disparity of opinion exists as to the clinical significance of pharyngeal/choanal bacterial isolates, particularly in the absence of clinical signs. This speaker feels that visibly inflamed oral cavities, associated with leukocytosis provides sufficient indication for treatment of specific gram-negative opportunists. The dilemma presented is whether or not to treat the Klebsiella or E coli pharyngeal isolate. Will it become a problem in the bird with less than adequate defenses? How do we know the pathogenicity of that particular strain? We don't have answers to these questions. Conversely when the acutely ill pet bird presents and yields a gram negative isolate, the clinician must be diligent to ascertain whether or not this isolate has ANYTHING at all to do with the illness.

A notable exception to the above: this speaker feels that Pseudomonas (and usually Aeromonas) isolates should be treated with fear and respect. While possibly recoverable by culture for months, untreated pseudmonads will eventually finish off the bird with a septic state.

Viral Diseases

The leading viral problems in psittacines resulting in mortality primarily in neonatal and pediatric patients. A history of no exposure to any birds for several years greatly lessens but do not inconclusively rule out viral disease.

Polyoma virus infections occasionally result in adult bird mortality. Herpesvirus hepatitis (Pacheco's) can strike without warning in a closed household collection with very little signs before "sudden death." Vaccination is not widely practiced and no satisfactory ante-mortem test is available. Adenovirus infections occasionally show up in multiple-bird collections.

Psittacine circovirus continues to be a significant threat to exposed mature psittacines, particularly Old World birds. While immune response has been measured in challenge studies, resulting in "street immunity" or the promise of future vaccines, adults can linger with a pediatric-contracted infection or can receive primary exposure as an adult.

While cockatoo producers appear to have exerted some control over this problem, African parrots continue to be victims. The pediatric African Grey parrot who is persistently infected with psittacine circovirus, will probably not survive to maturity.

A significant reservoir for infection and exposure exist in certain species, particularly the lovebird (Agapornis). Colony breeding, lack of veterinary consultation, and reduced lovebird circovirus awareness are probably factors which can contribute to the apparent high incidence in this genus. Appropriate screening (DNA-PCR, ARA Labs, Ohio) and biosecurity measures should be employed.

Chlamydial Infections

Acute chlamydiosis is generally a disease of young or newly exposed birds. An unknown percentage of exposed birds become exposed, infected and chronic carriers, without acuity. That is, they are not obviously ill. Shedding will persist for up to several weeks post-infection and typically taper off. Shedding in the mature bird may not occur, be infrequent, or occur in times of great physiologic/immunologic stress, including the injudicious use of corticosteroids. Currently available antigen and antibody detection methods fail to demonstrate carrier states in the mature bird.

Vague illnesses, characterized by occasional depression anemia and variable leukocytosis, may occur, improving in response to treatment of "doxycycline" deficiency. Many psittacine liver histopathology examinations will show a inflammatory component, such as cholangitis, in the absence of visible or recoverable (bacterial) organisms. Again, clinical improvement with doxycycline is a frequent finding.

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