the Vet's Corner

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Preliminary Report—Erythremic Myelosis in Conures, the "Hemorrhagic Conure Syndrome"

Summary

For many years, in clinical avian practice, the authors have noted what appears to be a syndrome in conure species (*Aratinga* and the related *Cyanoliseus patagonus*). The clinical history and/or physical examination may elucidate or suggest episodes of bleeding; hence we have coined the name "Hemorrhagic Conure Syndrome." The purpose of this report is to alert practitioners to our findings and to stimulate research on this perplexing problem.

Case I.

In December of 1983, a young blue-crowned conure (*Aratinga acuticaudata*) was rushed to our hospital for diagnosis and treatment. The bird had been purchased from a local pet store seven months previously. The patient was exhibiting apparent ataxia or profound weakness and polyuria on the day of presentation and had a history of intermittent polyuria and diarrhea. On the day of presentation, bloody fluid was noticed exuding from the nostrils. The bird died in transit to the hospital and was necropsied. Histopathologic examination of the submitted tissues revealed a large number of immature neoplastic-appearing RBCs within the hepatic sinusoids with a similar cluster also present in the pancreas. The diagnosis was Erythremic Myelosis (L.A. County Pathology Laboratory case record #111013).

Case II.

In early January of 1981, a mature golden-crowned conure (*Aratinga aurea*) was presented for diagnosis and treatment. The previous history included epistaxis, considered by the client to be a simple "nosebleed." On the day of presentation the bird was comatose and dyspneic and died shortly after being hospitalized. Histopathology examination of the submitted tissues revealed a considerable amount of hemosiderin within the lungs, suggestive of past hemorrhage. There was massive hemorrhage within the pectoral muscle, with central pseudocyst formation due to the presence of blood. Peripheral muscle fibers were degenerating secondarily. The liver sinusoids had diffuse influx of malignant hematopoietic blast cells. Moderate non-suppurative inflammation was seen in the pericardium. Focal incidental endocardial cartilage metaplasia was also seen. The bone marrow was hypercellular and populated by blasts with little evidence of significant maturation. The following diagnoses were rendered by Dr. James O. Britt, Jr., of the Los Angeles County Veterinary Pathology Laboratory (Case record #90912).

1. Erythroleukemia
2. Pericarditis, mild
3. Consolidating, multifocal pneumonia
4. Acute hemorrhage, muscle

Dr. Britt made the comment that spontaneous atrial rupture does occur in chickens and in caged birds and that it would be interesting to speculate that it and the hemorrhagic syndrome are associated with the myeloproliferative disease.

Case III.

In February of 1981, a mature half-moon conure (*Aratinga canicularis*) was presented for weakness and dyspnea. The history included previous episodes of dyspnea with nasal bleeding. The bird expired shortly after presentation and was necropsied. Lesions included hemosiderin-ladened macrophages within the lungs (around the vessels and bronchioles) and clusters of "hemosiderophages" beneath the air sacs. The hemosiderin accumulation was apparently due to previous hemorrhage into the respiratory system. The bone marrow (femoral) was populated by blasts, with early erythrocytic differentiation and leukemic blast cells seen in the hepatic sinusoids. The spleen was packed with masses of monomorphic hematopoietic cells. The diagnosis (Case record #91414) was Erythroblastosis (Erythremic Myelosis).

Case IV.

On June 23, 1983, a mature half-moon conure (*Aratinga canicularis*) was presented for diagnosis and treatment. This solo pet had been purchased at a local pet store five years previously and had been perfectly healthy. No recent exposure to other birds had occurred.

The bird was extremely weak, and exhibited an unusual trance-like posture on its perch. (The authors have often observed that conures weak from blood loss—Hemorrhagic Syndrome, injury, other causes—seem to prefer to remain perched in unusual positions, rather than lie on the cage bottom as other avian species prefer.) It was extremely thin (58.3 g) and the owner noted that bleeding episodes had occurred the previous week. The owner had assumed the bird had been injured and had suffered a "nosebleed." To facilitate diagnosis, a blood sample was collected (no appreciable stress occurred) via the nail clip technique and a gram stain was performed on the bird's feces. Tentative diagnoses included: 1) Hemorrhagic Conure Syndrome, 2) Gram Negative Septicemia, 3) Psittacosis, 4) Internal Injury, 5) Trauma, 6) other rarer conditions, 7) combinations of the above.

The laboratory work-up showed many abnormalities: the bird's PCV was 26%, there was a marked leukocytosis (27,500/mm) with a heterophilia (84%), heavy polychromasia and anisocytosis were evident, the total protein was markedly low, there was a borderline hypoglycemia and a marked hypocalcemia, and both the SGOT (SAST) and creatinine values were elevated. Many young red blood cells were seen in the peripheral blood suggestive of a response to recent blood loss. The fecal gram stain showed positive cocci and light gram negative rods.

Initial treatment consisted of i.m. injections of 5 mg of cefotaxime (Claforan - Hoechst-Roussel), .2 mg of dexamethasone, .20 cc Calphosan (Carlton), .05 cc Winstrol-V (Winthrop), and .05 cc B-complex. The bird was gently force-fed 3 cc of feeding mix three times daily with 5 mg of doxycycline (Vibramycin® - Pfizer) added to two of the feedings because of the possibility of psittacosis. Surprisingly, the bird tolerated the handling without struggling or exhibiting undue stress.

The bird showed signs of increased strength and was more alert but still critically weak two days later. It died in

the late evening of June 25, 1983, and was necropsied.

Gross pathologic findings included a hemorrhagic left lung and a slightly pale liver and heart. No other abnormalities were noted. Tissues were sent to Dr. James O. Britt, Jr. at the Los Angeles County Veterinary Pathology Laboratory for histopathology.

Dr. Britt reported that the liver had a relatively heavy influx of immature hyperchromatic cells throughout the sinusoids, most compatible with neoplastic, immature RBCs. The lung had an area of recent hemorrhage with many hemosiderin-laden macrophages. Other tissues were normal. No bone marrow, unfortunately, was submitted. The diagnosis was Erythremic Myelosis (Case #106888).

Discussion

For the past 15 years, the senior author has noted many similar cases of "bleeding conures." The condition has been so common that new technicians and veterinarians, along with clients, were always warned to "be careful when handling a sick conure as it may start coughing up blood and die." This condition was thought to be an idiosyncracy of the species involving their vascular response to respiratory inflammation. In retrospect, these may have been Erythremic Myelosis cases. Many of these cases were diagnosed as pulmonary hemorrhage of unknown cause on histopathology. We now know that many exhibit no lesions except in the bone marrow and bone marrow was not usually examined in most avian necropsies until recent times. Also, several cases showed atrial rupture as occurs in chickens. It is interesting to speculate on a possible relationship between the etiology in both species.

Much work has been done on the viral etiology of the leukosis/sarcoma disease in chickens. In these animals, the leukosis/sarcoma viruses induce a variety of transmissible benign and malignant neoplasms. Under natural conditions, by far the most common is lymphoid leukosis, but the following may also occur: Erythroblastosis, Myeloblastosis, Myelocytomatosis, Endothelioma, Nephroblastoma, Hepatocarcinoma, Fibrosarcoma, and Osteopetrosis. Table 2 summarizes the neoplasms of the leukosis/sarcoma group of viruses.

In the conures, what we are calling Erythremic Myelosis seems similar to Erythroblastosis of chickens. In fact, this term has been used several times

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Photo courtesy of San Francisco Visitors Bureau

Row upon row of crab boats and trawlers tie up at Fisherman's Wharf, a picturesque and palate-tickling corner of the San Francisco waterfront famed for its view restaurants and outdoor seafood counters.

Table 1

Date	Conure Species and No.	Lesions	B.M.	Diagnoses
9-24-80	Blue-Crowned #89220	1. Large, nucleated rbc's in liver 2. Ruptured heart	No	1. Ruptured heart 2. Regenerative anemia
1-22-81	Golden-Crowned #90912	1. Hemosiderin in lungs 2. Heterophils in lungs 3. Blast cells in liver 4. Pericardial inflammation 5. Bone marrow—many blasts	Yes	1. Erythroleukemia 2. Pericarditis, mild 3. Consolidating, multi focal pneumonia 4. Acute hemorrhage, muscle
3- 4-81	Half-Moon #91414	1. Hemosiderin in lungs 2. Bone marrow—many blasts 3. Spleen packed with monomorphic hematopoietic cells 4. Liver—leukemic blast cells	Yes	1. Erythroblastosis (erythemic myelosis)
3-10-81	Mitred #91491	1. Necrosis in liver 2. Suppurative inflammation of adrenal 3. Marrow smear—all myeloblasts and erythroblasts 4. Kidney—severe hemoglobinuria nephrosis	Yes	1. Multifocal liver necrosis 2. Severe nephrosis 3. Adrenalitis 4. Muscle hemorrhage 5. Erythremic Myelosis
1-14-82	Blue-Crowned #95685	1. Lung—massive hemorrhage hematoma with peripheral multinucleated giant cells—chronicity 2. Liver—numerous blast cells 3. Air Sacs—hemorrhage 4. Sub-acute enteritis 5. Air sac—hemorrhagic with hemosiderin pigment	No	1. Hemorrhagic syndrome 2. Erythremic Myelosis
7- 2-82	Maroon-Bellied #98805	1. Bone marrow—*autolyzed* 2. Liver—multifocal necrosis 3. Lungs—hemorrhage	Yes	1. Acute necrotizing hepatitis 2. Pulmonary hemorrhage
2-10-83	Golden-Crowned #103534	1. Bone marrow—sheet of hematopoietic blasts	Yes	1. Myeloproliferative disease, marrow (Erythremic Myelosis)
2-18-83	Half-Moon #103689	1. Bone marrow—uniform myelocytes 2. Liver—hematopoietic blasts 3. Lung—hemorrhage 4. Striated muscle—protozoal cysts	Yes	1. Myeloproliferative disease (myelogenous leukemia) 2. Pulmonary hemorrhage 3. Sarcosporidiosis
4-13-83	Half-Moon #104686	1. Liver—immature rubricytes 2. Bone marrow—75% rubriblasts	Yes	1. Erythremic Myelosis
7- 9-83	Half-Moon #106888	1. Liver—immature rbc's 2. Lung—recent hemorrhage, Hemosiderin laden macrophages (remote hemorrhage)	No	1. Erythremic Myelosis
10-17-83	Half-Moon #109095	1. Liver—immature erythroid cells 2. Bone marrow—almost all erythropoietic cells	Yes	1. Erythremic Myelosis
12-12-83	White-Eyed #110562	1. Liver—acute, coagulation Intestine—necrosis, hemorrhage 2. Lung—laminated concretions 3. Bone marrow—solid sheets of erythroblasts	Yes	1. Internal bleeding 2. Shock 3. Erythremic Myelosis
1- 5-84	Blue-Crown #111013	1. Liver—large numbers of immature neoplastic rbc's 2. Pancreas—neoplastic rbc's 3. Spleen—neoplastic rbc's 4. Parathyroid—hyperplastic or adenomatous	No	1. Erythremic Myelosis 2. Parathyroid adenoma or hyperplasia (the former if only one gland is involved)
3-23-84	Nanday #112691	1. Bone marrow—packed with blasts 2. Liver—moderate population of blasts	Yes	1. Myelocytic leukemia or erythroleukemia (Erythremic Myelosis)

by pathologists describing our submitted cases.

In chickens, two forms of Erythroblastosis have been described. The blastic or proliferative form is more commonly observed than the anemic form. It is characterized by the presence of many erythroblasts in the peripheral blood. In the anemic form, there are relatively few immature cells in the circulating blood and the predominant feature is marked anemia. Results of transmission experiments have suggested a close etiologic relationship between the two forms.

Symptoms in chickens are lethargy, weakness, and comb paleness or cyanosis. As the condition advances, the combs of the chickens with the anemic form become paler until they are light yellow to almost white. Chickens with the proliferative form may become pale or cyanotic and exhibit weakness, emaciation, diarrhea, and there may be profuse hemorrhage from one or more feather follicles. The clinical course varies from a few days to several months. The anemic form is usually of shorter duration.

The incubation period after intraabdominal innoculation of the causative RPLIZ strain virus into susceptible day-old chicks varies from 21 to 110 days. Intravenous innoculation of 11 day old embryos may produce chicks with Erythroblastosis upon hatching. Field strains and viruses passaged in cell culture induce Erythroblastosis after a longer incubation period. A similar viral strain R produces a much more rapid response with innoculated birds sometimes dying between seven and 12 days postinnoculation.

Half-moon conure in the classic weak stance of anemic conures.

In chickens, lesions of Erythroblastosis of either form include anemia, often accompanied by petechial hemorrhages in various organs such as the muscles, subcutis, and the viscera. Thrombosis, infarction, and rupture of the liver or spleen may be observed. There may be pulmonary edema, hydropericardium, ascites, and a fibrinous clot on the ventral surface of the liver. The most characteristic gross alteration in the proliferative form is diffuse enlargement of the liver and spleen and to a lesser extent the kidneys. In the anemic form there is usually an atrophy of the visceral organs, particularly the spleen. The bone marrow is pale and jelly-like and the marrow spaces may be largely replaced by spongy bone as seen in osteodystrophia fibrosa.

Photos by authors

Mitred conure with a "nosebleed" of the Conure Bleeding Syndrome which often resolves then recurs, then the bird dies.

The alterations in the visceral organs of chickens with the proliferative form of Erythroblastosis are primarily due to a hemostasis resulting in the accumulation of erythroblasts in the blood sinusoids and capillaries. In the anemic form the liver often has accumulations of small lymphocytes and granulocytes indicative of a reactive center; however, careful search will reveal localized areas of erythropoietic activity.

The syndrome in conures most closely resembles the proliferative form although the characteristic erythroblasts have not been observed in the peripheral blood. Whether the etiology of this condition is viral remains to be seen, although the similarities are striking. If the etiology is viral, is it the same virus that affects chickens or a similar related virus? Bird suppliers tell us that shipments of young conures, coming from Mexico, Central and South America are frequently housed in close proximity to chickens. Obviously, transmission studies with known leukosis/sarcoma chicken viruses are indicated. If the etiology is viral, then we must wonder how a five year old conure could contract the disease unless the prodromal phase is extremely prolonged (as in feline leukemia).

The pathogenesis of the bleeding episodes seen in these birds is an intriguing problem. The thrombocyte numbers have not been noticeably low on blood slides seen at the authors' and other laboratories. In case IV, the calcium level was low, but not low enough to be associated with bleeding. One can speculate that clotting factors must be interfered with by the action of the cancer cells in the bone marrow and in the liver. The incidence of respiratory and muscle bleeding in conures, compared to skin and visceral bleeding in chickens, is also intriguing.

Leukosis-type diseases have also been seen in other caged birds. Leukemia is seen from time to time in canaries, budgerigars, cockatiels, and rare others. The authors are studying what may be another syndrome, Heterophilic Leukemia in Brotogeris parrots. Are any of the etiologies similar? Again, much research is necessary to answer these and other questions.

The authors have seen many cases of proven Erythremic Myelosis in conure species. Unfortunately, many of our records were unretrievable without an exhaustive and prohibitive search through stacks of old files. However, several recent reports have been saved and a few were listed in the preceeding table for the benefit of those interested in this fascinating syndrome. Proven cases and two "probable" cases will be listed. "B.M." refers to whether or not bone marrow was submitted.

Of the twelve proven cases of Erythremic Myelosis, the following statements can be made. Five cases were seen in half-moons, two cases in golden-crowns, two cases in blue-crowns, and one case each in a nanday, a mitred, and white-eyed conure and the other in a maroon-bellied conure. Of the nine proven cases that had bone marrow examinations, lesions were found in all bone marrow smears. In the one "probable" case that had a bone marrow examination, the bone marrow was autolyzed. In proven cases, nine out of twelve had "blast" cells in the liver. In two cases, blast

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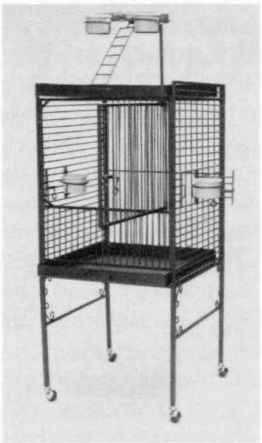

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cells were seen in the spleen, and in one case (one of those with splenic lesions) blast cells were seen in the pancreas. No other tissues produced lesions of Erythremic Myelosis. Four cases showed hemosiderin pigment in the lungs, indicating previous hemorrhage, and seven cases in all showed evidence of hemorrhage (recent or previous).

Conclusion

Erythremic Myelosis is defined as a specific neoplastic condition involving the red cell precursors in the bone marrow. It is a form of myeloproliferative disease that has been found to be a significant problem in our area among conure species. The problem has been evident for at least fifteen years, although no mention of it as seen in these species could be found in the literature.

The disease has been seen in very young conures but has also been seen in many older birds, some at least five years of age without recent bird contact. A history of a previous bleeding episode may or may not be elicited from the owner. The etiology is unknown at this time but it is tempting to speculate about possible viral causes, especially considering the similar syndromes seen in domestic chickens and their reported proximity to pet conures during holding and shipping procedures. The possible proximity of wild conures to domestic fowl may also be a factor.

The authors know of several conures which have survived the disease. Several cases have occurred which have gone into apparent remission. One half-moon is still alive after surviving two bleeding episodes the year previously. There may be several forms of the disease and its manifestations may depend on individual resistance, degree of immunity, etc. Perhaps only a certain percentage develop the classic lesions of Erythremic Myelosis. Maybe there are several other diseases that result in bleeding episodes in conures. The authors are now performing tibral bone marrow taps to aid in the diagnosis of true Erythremic Myelosis. The differential diagnosis of bleeding disorders should include bacterial septicemia with resulting clotting problems, cover disease with low vitamin K levels, hypocalcemia, trauma, toxemias, and other conditions. We have also treated conures which have had a non-fatal hemorrhagic episode and have seen their PCVs rise in a relatively short period of time only to see these birds die during a subsequent hemorrhagic episode months later. Local conure breeders have described a fatal bleeding problem in neonatal conures. To date, none of these birds have been necropsied. Also, the authors have seen profound hemorrhagic conjunctivitis with extensive circumorbital ecchymotic hemorrhage in several conure species. Are the above conditions related to the Erythremic Myelosis Syndrome? We can only speculate on the possible immune status of the population in general. Clinicians are advised of the existence of the condition and asked to cooperate with research institutions interested in the syndrome, hopefully as a result of this report.

Table 2

Neoplasms caused by viruses of leukosis/sarcoma group (Chickens)

	Neoplasm
Leukoses	
	Lymphoid leukosis
	Erythroblastosis
	Myeloblastosis
	Myelocytoma(tosis)
Connective tissue tumors	
	Fibroma tissue tumors
	Myxoma and myxosarcoma
	Histiocytic sarcoma
	Osteoma and osteogenic sarcoma
Related tumors	
	Hemangioma
	Nephroblastoma
	Hempatosarcoma
	Osteopetrosis

Hofstad, M.S.: Diseases Of Poultry — 7th Edition — Iowa State University Press, Ames, Iowa, 1978.

Table 3

Clinical Pathology Results on a Half-Moon Conure with Erythremic Myelosis (Case 4) 6/23/83

		Normals[2,3]
WBC/mm3	27,500	4,000-11,000
Hets (%)	84	40-75
Lymphs (%)	8	25-35
Monos (%)	6	0-3
Eosinos (%)		0-3
Basos (%)	2	0-5
PCV (%)	26	42-55
Blood Parasites	NPS	NPS
Anisocytosis	Heavy	Slight
Polychromasia	Heavy	Slight
Thrombos	Present	Present
Total Protein (g/dl)	1.9	2.5-4.5
Glucose (mq/dl)	180	200-350
SGOT (SAST) IU/L	1,100	125-350
Creatinine (mq/dl)	0.89	0.1-0.5
Calcium (mq/dl)	6.7	8.0-15.0
Comments:	Many young RBCs	
Fecal Gram Stain	Pos. Cocci	Pos Cocci only
	Light Neg. Rods	