Progressive Feather Dysplasia in a Juvenile Bald Eagle

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Abstract: Feather dysplasia is commonly associated with various etiologies in captive psittacine birds but is rarely reported in wild, non-psittacine avian species. Infectious causes, primarily novel circoviruses and West Nile Virus, have been identified in the vast majority of affected avian wildlife. This clinical report examines the case of a juvenile bald eagle (*Haliaeetus leucocephalus*) with a progressive feather dysplasia that demonstrated no evidence of infectious etiology. Severe feather abnormalities were found affecting all rectrices and remiges. Progression of feather abnormalities in this case was documented with photography over one year. Bloodwork, lead and viral testing, and histopathology were performed. While many gross similarities exist to the "pinching off syndrome" previously reported in white-tailed sea eagles (*Haliaeetus albicilla*) in Europe, histopathology findings do not strongly correlate. This represents the first formal report of progressive feather dysplasia in a juvenile bald eagle with no apparent evidence of infectious cause.

Keywords: bald eagle*, Haliaeetus leucocephalus*, feather dysplasia

Introduction

Feather dysplasia is generally defined as the abnormal growth or development of a feather. The condition is frequently described in cases of captive psittacine birds infected with circovirus (ie, Psittacine Beak and Feather Disease) or polyomavirus; however, reports of feather dysplasia in wild, non-psittacine avian species are limited. Novel circoviruses have been recently identified in a variety of non-psittacine species, including pigeons,[1] gulls,[2] and ravens[3]. Additionally, infection with West Nile Virus has been associated with limited cases of feather dysplasia in North American raptors.[4]

A unique case of feather dysplasia was described in 1975 in a fledgling white-tailed sea eagle (WTSE) in Germany.[5] This was then followed by over thirty similar cases in this species over the last couple decades throughout northern Europe.[6-8] The term "pinching off syndrome" was originally coined to describe the characteristic appearance of the affected feathers in these birds. Recent investigation ruled out a viral etiology in these cases, among many other possibilities, and suggested the possibility of a genetic cause in this species.[6,8]

In 2010, a juvenile bald eagle *(Haliaeetus leucocephalus*) presented to the Wildlife Care Center of the Audubon Society of Portland (Portland, OR) with severe feather dysplasia that appeared highly similar to that described in the WTSE publications. Rendered incapable of flight due to its feather condition, the eagle was housed at the Care Center for one year while feather abnormalities were closely monitored and documented with serial photography. This clinical report seeks to document the gross and histopathologic characteristics observed in this particular case and compare them to those described in

the WTSE cases. While sparse anecdotal reports of feather dysplasia in North American raptors (particularly bald ealges) exist, this is the first formal presentation of such. The goal of this presentation is to establish awareness of this condition in bald eagles and encourage collaboration on identifying future cases so that a definitive etiology may be determined.

Case Report

In July of 2010, an approximately 14-week old wild male bald eagle from southwestern Washington State presented to the Wildlife Care Center of the Audubon Society of Portland after being found on the ground below its nest. A frequent observer of the nest noted a single clutchmate had existed which had been witnessed to successfully fledge three weeks prior. On initial presentation the eagle was emaciated, dehydrated, and severely lethargic. Severe feather abnormalities were noted to affect all remiges and rectrices and were initially presumed a result of a heavy infestation of ectoparasites. Leukistic mottling of many feathers was also noted but was deemed normal variation within this species.

A complete blood cell count demonstrated a hematocrit of 30% (reference range,[9])with few immature erythrocytes and moderate polychromasia; rare leukocytozoans were noted. A mild leukopenia (9.8x 10^3 cells/uL; reference range,[9]) characterized predominantly by heterophils (7154 x 10^3/uL; reference range,[9]) was also noted. Blood chemistry revealed moderate creatinine kinase elevation (1683IU/L; reference range,[9]) and mild hypoglycemia (229mg/dL; reference range,[9]) attributed to starvation. No other abnormalities were noted. Protein electrophoresis was not performed. Following supportive care and treatment for ectoparasites with ivermectin (0.2mg/kg PO once; Ivomec 1% Injection, Merial, Duluth, GA, USA) the hematocrit increased to 44% two weeks following presentation. Initial blood lead levels performed by a commercial veterinary lab were unremarkable (0.0ug/dL). Full-body radiographs were also unremarkable.

Despite marked improvement in the eagle's mentation and body condition and resolution of feather mites, the feather abnormalities persisted. All remiges and rectrices appeared to be affected, with the primary flight feathers showing the most severe changes. Distal wing coverts also appeared to be mildly affected as time progressed. Over the course of one year the beak, talons, and contour feathers remained normal in gross appearance. Feather abnormalities were characterized by the following: longitudinal splitting of the ventral rachis of all affected feathers; weakening of the rachis resulting in easy bending and ventral curvature of some feathers; "tufts" of disorganized keratin at proximal shaft of some feathers; premature dropping of primary feathers in a symmetric fashion with replacement by feathers with similar abnormalities; "pinching off" in a subset of these dropped feathers; lack of "hooking" of barbules resulting in a disheveled appearance of the feathers. These changes were consecutively documented with digital photographs (Figures 1 and 2). Onset of the appearance of these abnormalities seemed delayed until the growing feather had partially emerged from the sheath. A flakey, yellow-brown crust developed along the ventral shaft as the feather finished growing.

During the first month of presentation, investigation into viral etiology was made given the common association of feather dysplasia and viruses in other species. PCR testing for avian polyomavirus, circovirus, and avian coronavirus were negative (Veterinary Molecular Diagnostics, Inc., Milford, OH). Additionally, testing for West Nile Virus antigen via real time PCR was also negative (Oregon State University Veterinary Diagnostic Lab, Corvallis, OR).

Nearly one year after initial presentation, the eagle entered into its "first-year" molt. New feathers were even more severely dysplastic. Given the contraindication for release and poor prognosis, euthanasia was elected.

Sections of skin and feather, active feathers, spleen and liver were collected post-mortem and submitted for histopathology (Zoo/Exotic Pathology, West Sacramento, CA). The epidermis was noted to be extremely thin, being only one cell layer thick in most sections; keratin was variable and somewhat irregular but overall normal in appearance. These findings, however, need to be validated by comparison to skin sections from the same area in a "normal" bald eagle of this age. Smaller follicles appeared underdeveloped. No specific lesions were observed in the larger follicles (Figure 3). There was no indication of any infectious or inflammatory condition. A mild lymphoplasmacytic cholangitis and mild multifocal plasmacytic splenitis were also observed. Although suggestive of a systemic inflammatory response, no specific microbes were recognized. It is not known if these latter findings related to the underlying cause of feather dysplasia.

Discussion

The wild juvenile bald eagle presented in this case demonstrated a severe and progressive feather dysplasia. Scant published reports of feather dysplasia and "pinching off" in bald eagles have previously been associated with a viral etiology (ie, West Nile Virus[4]), as have similar conditions in other North American raptors. Molecular diagnostics and histopathology, however, did not support an infectious cause in this particular case.

Between 1975 and 2006, thirty-two cases of "pinching off syndrome" were identified in WTSEs throughout northern Europe, with seventeen of these cases isolated to Germany.[6] All cases were limited to fledglings rendered flightless by the severity of the feather dysplasia, which affected all remiges and rectrices of the affected birds; no other abnormalities could be identified. Unlike those cases associated with West Nile Virus in North American raptors, all WTSE cases that were monitored over time in captivity demonstrated a progressive loss of flight feathers with replacement by similarly dysplastic feathers. The juvenile bald eagle in our case progressed similarly.

Gross characteristics of the dysplastic feathers in this case were identical to many of those described and photographically depicted in the WTSE papers.[6,7] Several prematurely dropped primary feathers demonstrated the classic "pinching off" morphology described in those papers. More striking was the severe longitudinal splitting along the ventral aspect of every single primary, secondary, and tail feather with heavy deposits of grossly disorganized keratin. Consequently, weakening of the integrity of the feather shaft resulted in the grounding of the eagle in our case as well as in the WTSE cases.

While gross appearance of the affected feathers and the pattern of involvement bared marked resemblance to the published cases in WTSEs, comparison of histopathology was less convincing. Muller et al[6] described the histopathology of 52 remiges and rectrices from 4 WTSEs affected with "pinching off syndrome". Epithelial degeneration and necrosis was noted in the epidermal collar and papilla of some feathers in all 4 of these birds, as well as similar changes in the feather sheath of 3. Necrosis and degeneration associated with vacuolar changes were noted in the cytoplasm of the pulp epithelium in 3 of 4 birds. Additionally, marked heterophil leukocytosis was noted in the pulp of growing feathers of all 4 birds. None of these findings were observed in the bald eagle. "Dyskeratosis" affecting the pulp epithelium and focal areas of the epidermis were also noted in the WTSEs. The bald eagle in this case grossly appeared to share the same type of excessive, disorganized build-up of keratin in the feather follicles; however, as no definition or illustration of the term "dyskeratosis" was provided in the WTSE papers, it cannot be determined if the same finding was present on histopathology. Lipofuscin was identified in the neurons, glial cells, and pancreas of all 4 of the WTSEs. There was no

lipofuscin detected in the samples of liver and spleen submitted from the bald eagle, but since no brain tissue was examined the possibility of neuronal accumulation cannot be ruled out.

The reason as to why only flight and tail feathers are affected in these birds is unknown. Of additional curiosity is how these marked changes in the appearance of each feather seemed to develop only after the distal third of the growing feather emerged from the sheath. Given the specificity of the feathers involved, the lack of other clinical signs, and the persistence of abnormalities despite adequate diet during captivity, it seems nutritional, toxic, and metabolic causes are unlikely, as was surmised in the WTSE studies where further testing was performed. Hypothyroidism has been associated with changes in plumage, [10] however thickening of the epidermis was noted in that case and feather loss rather than dysplasia was observed. Thyroid testing currently remains unvalidated in birds. Nonetheless, thyroid testing and histopathology of the thyroid gland should be considered in future cases of feather dysplasia given the role of thyroid hormone in feather development and skin condition. Genetic aberrations remain a plausible explanation. Cases of multiple WTSEs from the same nest affected over the years have been observed.(citation?) To our knowledge, both bald eagles in the successive clutch the following year fledged, although it is unknown if there may exist milder, subclinical forms of this feather dysplasia that would not necessarily prevent flight.

Many anecdotal reports of feather dysplasia in juvenile bald eagles can be found throughout the United States. Characteristics of the dysplasia vary among reports. While West Nile Virus is known to cause a similar "pinching off" syndrome, this does not appear to be the only cause. Strong gross similarities between the progressive feather dysplasia in our case and those thoroughly described in WTSEs exist, however the limited histopathology performed in this case is less convincing. Thyroid, trace mineral, and endoscopic evaluation have previously been performed in the WTSEs with unremarkable findings. These diagnostics were not performed in this case but should be considered in future cases, as should a full necropsy (including histopathology) of affected birds. The goal of this report is to increase awareness and encourage identification of cases of feather dysplasia in bald eagles; collaboration in compiling this data is needed to better understand the etiology and potential ramifications of this condition.

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