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PRESUMED PHOTORECEPTOR DYSPLASIAS IN PEREGRINE FALCONS (*FALCO PEREGRINUS*) AND PEREGRINE FALCON HYBRIDS

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ABSTRACT: We describe a case series of photoreceptor dysplasia with secondary retinal degeneration in juvenile peregrine falcons. Six Peregrine Falcons (*Falco peregrinus*) and three Peregrine Falcon × Prairie Falcon (*Falco mexicanus*) hybrids had early-life visual deficits. Eight birds had visual defects shortly after hatching, and one bird had visual deficits first noticed at 5 mo of age. Complete ophthalmic examinations were performed in each animal. Eight of the animals had electroretinograms, and nine of the animals had their eyes examined histologically after euthanasia. Ophthalmic examinations did not reveal consistent and potentially blinding abnormalities, including an absence of ophthalmoscopic retinal lesions. Electroretinographic findings included subnormal amplitudes (with rod responses more abnormal than cone responses), with a negative b-wave amplitude occurring in one bird. Histologically, a reduction in the number of photoreceptors was present with numerous degenerative changes to the remaining photoreceptors, including frequent blunting and disorganization of photoreceptor outer segments, decreased numbers of cells in the inner nuclear layer, decreased numbers of ganglion cells, decreased thickness of the nerve fiber layer, and decreased myelinated axons within the optic nerve. Ultrastructurally, only minor cone outer segment changes and occasional phagocytic cells were seen. Results strongly suggested a primary retinopathy, characterized by photoreceptor dysplasia and secondary retinal degeneration with loss of cellular elements throughout the retina. The presence of a similar spectrum of findings in related individuals, the early age of onset, and the relative lack of other environmental, ocular, or systemic abnormalities suggested possible heritability.

Key words: Avian, bird, cone, eye, genetic, ophthalmology, raptor, vision.

INTRODUCTION

The Peregrine Falcon (*Falco peregrinus*) was once an endangered species in the US because of widespread organochlorine pesticide use from the 1950s to 1970s (White et al. 2002). However, these birds have made a remarkable recovery since the early 1970s due to the banning of certain pesticides and strong conservation efforts (White et al. 2002). An extensive captive breeding and release program led to more than 7,000 peregrines being released by 1998 (White et al. 2002). However, captive breeding and release has the potential of becoming problematic from the introduction of heritable defects into a population. As part of appropriate conservation management, it is important to identify conditions that may have a heritable basis so that affected birds may be removed from the general breeding population. Of primary importance are particularly severe heritable conditions that would negatively affect the overall health of the population, such as conditions that would impair visual performance in such visually dependent animals.

Peregrine Falcons, widely recognized as the fastest bird on Earth, reaching speeds upward

of 230 miles per hour during stoops (Orton 1975), are highly visual animals dependent on vision while flying at high velocities and maneuvering to capture their prey midflight (Lee and Kuo 2001; White et al. 2002). Exceptional visual acuity in falcons was initially suggested through observation of wild birds in action and reports on anatomy of the eye and retina; for example, falcons have a greater cone density than humans (Walls 1942; Polyak 1957). When tested behaviorally, American Kestrel (*Falco sparverius*) visual acuity was originally thought to be between 2.4 and 2.6 times greater than that of humans (Fox et al. 1976), although this has since been challenged (Gaffney and Hodos 2003; Potier et al. 2016).

Primary retinopathies are rarely reported in birds, with reports on free-living avian species being limited to a Prairie Falcon (*Falco mexicanus*; Dukes and Fox 1983) and a Peregrine Falcon × American Kestrel hybrid (Murphy et al. 1985). A primary retinopathy occurs as a particular retinal cell begins to differentiate (e.g., photoreceptor cells in photoreceptor dysplasia) and can be early- or late-onset. Early-onset primary photoreceptor retinopathies typically cause early visual deficits, severe photoreceptor structural abnormalities, and rapid progression from further retinal degeneration. Retinas with late-onset primary photoreceptor retinopathies (e.g., some forms of progressive retinal atrophy) have developmental abnormalities but have variable onsets of visual deficits and rates of progression. In contrast, secondary retinopathies are typically characterized by photoreceptor degeneration following normal or apparently normal photoreceptor development and differentiation (e.g., trauma, toxins, systemic disease; Narfstrom and Peterson-Jones 2013; Moore et al. 2017). In contrast to primary retinopathies, there are numerous reports of secondary retinopathies across multiple avian species, including in several species of raptors (Buyukmihci et al. 1988; El-Sayyad et al. 2014).

The purpose of this study was to determine the clinical, electrophysiologic, and histologic findings in a series of nine Peregrine Falcon and Peregrine Falcon hybrids that were presented with visual deficits. Overall, diffuse retinal disease was observed. In all but one case, visual deficits were noted shortly after birth. A few individuals showed mild asymmetry in terms of disease severity, but in all birds the retinal abnormalities were bilateral. Relatedness was known in several of the birds examined, and in these cases, the condition was suspected to be heritable.

MATERIALS AND METHODS

Birds presented to the University of California Veterinary Medical Teaching Hospital (Davis, California) or the University of Wisconsin (Madison, Wisconsin) School of Veterinary Medicine underwent complete ophthalmic examinations, performed on all birds by a veterinary ophthalmologist (C.J.M., A.J.M., S.R.H.). Initially, vision was tested by observation cage-side, as well as the response to and an apparent awareness of moving objects (e.g., hands, cotton balls) placed both closely to the head and at varying distances from the bird (up to 1 m). The menace response was evaluated by use of hand motions toward the head without creating an air current that could result in a false positive response (a positive response was recorded as a blink). The pupillary light reflex (PLR) was evaluated with an open beam from a high-intensity light source. Anterior and posterior segment examinations were performed by slit-lamp biomicroscopy, and the fundus was evaluated by use of indirect ophthalmoscopy. Anterior segment, posterior segment, and fundus examinations were performed in a dark room without the use of pharmacologic dilation. Intraocular pressures were not routinely measured.

Electroretinography (ERG) was performed of both eyes without anesthesia, sedation, or pharmacologic pupil dilation on eight of the nine birds (except for one bird that died before the second eye could be tested) using a commercially available unit (BPM-100, RetinoGraphics Inc., Norwalk, Connecticut, USA). We have successfully performed ERG on other falcons and other avian species using this unit without the use of pharmacologic pupil dilation. Monopolar brass and copper wire reference and ground electrodes were placed subdermally, approximately 1 cm lateral to the lateral canthus and sagittally on the forehead, respectively. The active contact lens electrode (ERG-Jet™, Fabrinal SA, La Chaux-de-Fonds, Switzerland) was placed on the cornea after administration of a hypromellose gel coupling agent. After a calibration tracing, ERG tracings of (5, 12, 30, and 40 Hz) were obtained

after light adaptation (3, 4, 5, and 6 min) to standardize the amount of light exposure to each patient before dark adaptation, followed by ERG tracings after dark adaptation (2, 5, 10, 15, 20, and at least 30 min as a maximum). Before dark adaptation, no filter was used. During dark adaptation, a red filter (so as not to bleach photoreceptor RH1 rhodopsin continually) and a neutral density filter were used. Red and blue light responses were also tested using scotopically balanced red and blue filters, respectively.

Histologic processing and evaluation was performed in nine of 10 birds at presentation, including transmission electron microscopy (TEM) in one bird. Eyes that underwent routine paraffin processing were placed in either Bouin's fixative or glutaraldehyde, sectioned in a parasagittal plane, and stained using H&E. All eyes embedded in plastic were placed in either glutaraldehyde or Karnovsky's fixative. One eye that underwent TEM was placed in the solution of McDowell and Trump (McDowell and Trump 1976).

RESULTS

A total of six Peregrine Falcons and three Peregrine Falcon × Prairie Falcon hybrids were evaluated for vision loss (Table 1). All birds were born in captivity in California or Idaho. There was no sex predilection (five males, three females, one unknown; see Table 1). Severe visual disturbance (possibly complete blindness) was evident to owners or breeders at or shortly after birth in all but one bird, in which visual deficits were not detected until after fostering at approximately 5 mo of age (case 2). All birds were presented for evaluation from 1 mo to 4 yr of age, with a median age of 1.5 mo. Three birds from two separate clutches (cases 3–5; Table 1) were presented from the same parents (neither parent was reported to have visual deficits). This pair produced a total of 21 young, five of which were reported to have vision problems (including cases 3–5) and 16 with no evidence of visual deficits. All other birds in this study were either unrelated or had a relationship that was unknown, with no evidence of any visual deficits among clutch mates or parents. Visual deficits were reported as disorientation, difficulty locating food and perches, a lack of response to intruders within the enclosure, and a lack of response to hand motions. In one individual (case 1; Table 1), nyctalopia was observed, although photopic visual deficits were present as well.

On examination, all birds had variable aberrant visual search and absent or greatly impaired visual tracking, menace responses, and PLRs. No neurologic signs other than visual impairment were noted in any bird. Congenital incomplete cataracts were noted in both eyes of one bird (case 5), and microphakia, cataract, and subluxated lenses in both eyes in another bird (case 7). Mild alterations in fundus pigmentation were noted sporadically across different regions of the retina as linear, radiating bands of hypopigmentation, as commonly seen as a normal variant in the fundus of birds (Table 1 and Fig. 1). In many birds, areas of hyperpigmentation or pigment clumping were noted, in some cases involving the fovea. No other visible retinal or pecten oculi lesions that could be attributed to causing the apparent visual deficits were noted.

Electroretinography showed subnormal photoreceptor amplitudes (Fig. 2; case 1), with rod function being lower than cone function in several cases (cases 3, 4, 5). A negative b-wave (relative to the a-wave) was detected in one bird (case 6). Additionally, review of the flicker fusion results of case 6 at varying frequencies indicated an asymmetry between the amplitudes of right and left eyes, although the implicit times remained essentially the same.

Histologic evaluation of all birds showed a central to diffuse pattern of reduction in the number of photoreceptors and numerous degenerative changes to the remaining photoreceptors (Fig. 3; case 1). Degenerative changes included frequent blunting and disorganization of photoreceptor outer segments and a qualitatively decreased number of cells in the inner nuclear and ganglion cell layer, decreased thickness of the nerve fiber layer, and decreased myelinated axons within the optic nerve. Ultrastructurally (case 1), abnormalities were minimal and were limited to mild cone outer segment changes (outer segment discs were fractured and formed

TABLE 1. Representation of six Peregrine Falcons (*Falco peregrinus*) and three Peregrine Falcon × Prairie Falcon (*Falco mexicanus*) hybrids, several of which were related, that were evaluated for vision loss. All individuals underwent complete ophthalmic examination, including slit-lamp biomicroscopy and indirect ophthalmoscopy. Electroretinograms (ERGs) were performed in all but one individual. Diagnosis of retinal degeneration with characteristics consistent with photoreceptor dysplasia were confirmed histologically.[a]

Case	Bird	Sex	Age at first signs	Age at presentation	Relatives affected	Ophthalmic abnormalities	ERG abnormalities	Histologic diagnosis
1	Peregrine Falcon	Female	5 d	4 mo	None	Menace, PLR OU	Severely subnormal	Total retinal degeneration
2	Peregrine Falcon	Male	Postfostering	4 yr	None	PLR OU	Severely subnormal OS[b]	Photoreceptor dysplasia/degeneration
3	Peregrine Falcon	Female	Rearing	30 d	Cases 4, 5	PLR, foveal pigment OU	Severely subnormal	Total retinal degeneration
4	Peregrine Falcon	Male	Rearing	28 d	Cases 3, 5	PLR, tracking OU	Severely subnormal	Total retinal degeneration
5	Hybrid	Male	Fostering	30 d	Cases 3, 4	Cataract OU	Subnormal	Total retinal degeneration/dysplasia
6	Peregrine Falcon	Male	5 d	3 mo	None	Menace, PLR OU	Severely subnormal, negative b wave	Photoreceptor dysplasia/degeneration
7	Hybrid	Male	5 d	4 mo	None	Microphakia, cataract, lens subluxation	Severely subnormal	Total retinal degeneration
8	Peregrine Falcon	Female	Birth	48 d	Unknown	Tracking OU	Severely subnormal	Total retinal degeneration/dysplasia
9	Hybrid	Unknown	Birth	38 d	Unknown	Tracking OU	Not done	Total retinal degeneration/dysplasia

[a] OU = both eyes; OS = left eye; PLR = pupillary light response.

[b] Patient died during left eye measurement, precluding evaluation of right eye.

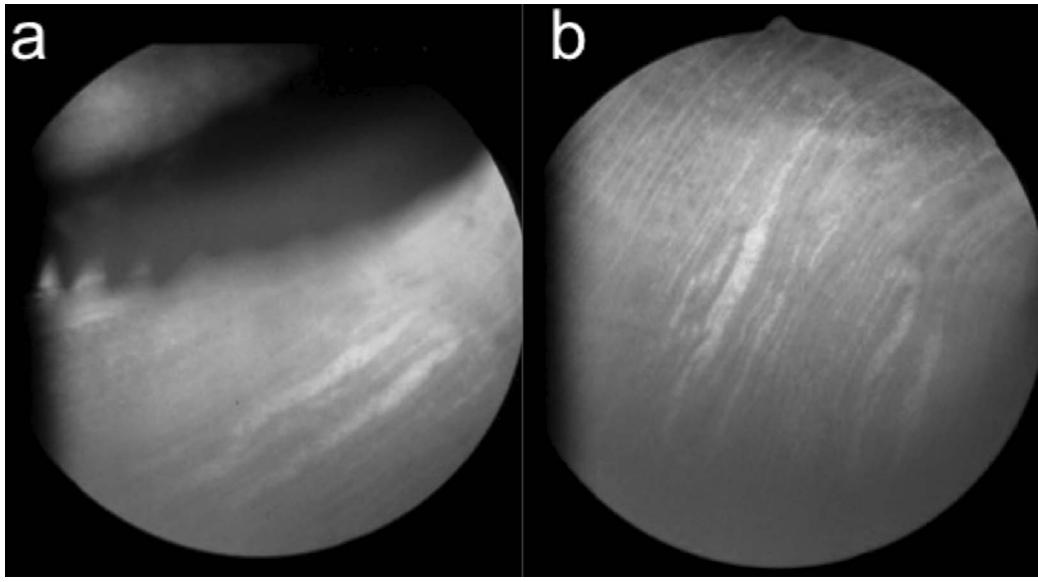

FIGURE 1. Funduscopic photographs of the left eye of case 1. There is no evidence of retinal disease. (a) A normal pecten oculi is visible as a black, wedge-shaped structure in the dorsal aspect of the plate. The linear areas that allow visualization of underlying sclera in both panels (a) and (b) are considered variations of normal. The fundus of the right eye was virtually identical.

clusters within the cell membrane of the outer segment) and occasional phagocytic cell infiltration between photoreceptor processes (Fig. 4). Retinal dysplasia characterized by several folds and rosettes consisting of a discontinuous external limiting membrane and inward displacement of photoreceptor nuclei was present in three cases (cases 5, 8, 9) and was likely to be unrelated to the other retinal abnormalities. No other significant ocular lesions were found in any bird other than the previously described lesions found on ophthalmic examination. Systemically, nephrosis and hepatitis were evident in one case (case 2).

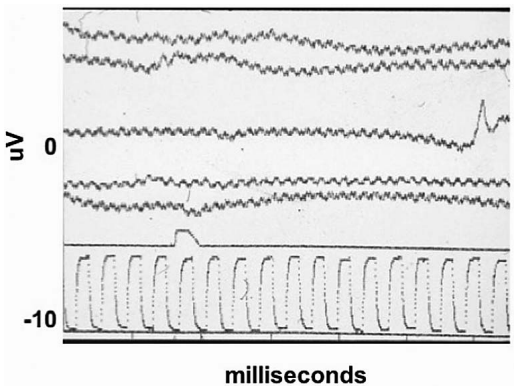

FIGURE 2. Representative electroretinogram from case 1. The electroretinogram tracings represent (from bottom to top): calibration tracing, stimulus line, and the retinal response after 3, 4, and 5 min of light adaptation and 2 and 5 min of dark adaptation. The responses seen on lines 5–6 are mixed rod/cone responses. The rod contribution begins to dominate after about 30–40 min of dark adaptation. Although there is background noise, it is not enough to obscure a normal response; thus, the electrical responses are severely diminished under all conditions.

Diet was appropriate in all cases and mostly consisted of whole quail that had been fed a balanced grain diet supplemented with vitamins and minerals. A complete blood count and fecal floatation and smear were evaluated in case 1, all of which were unremarkable. For case 6, a complete blood count was unremarkable, and adipose tissue was submitted to the New York Zoological Society (New York, New York, USA) for vitamin E and retinol analysis. Vitamin E levels were 49.58 μg/g wet weight (29.8 μg/mL serum value reported in 18 normal species of falconiform birds; Packer and Jurgen 1992), and retinol levels were 9.16 μg/g wet weight (normal concentration in adipose tissue is unknown, but the serum

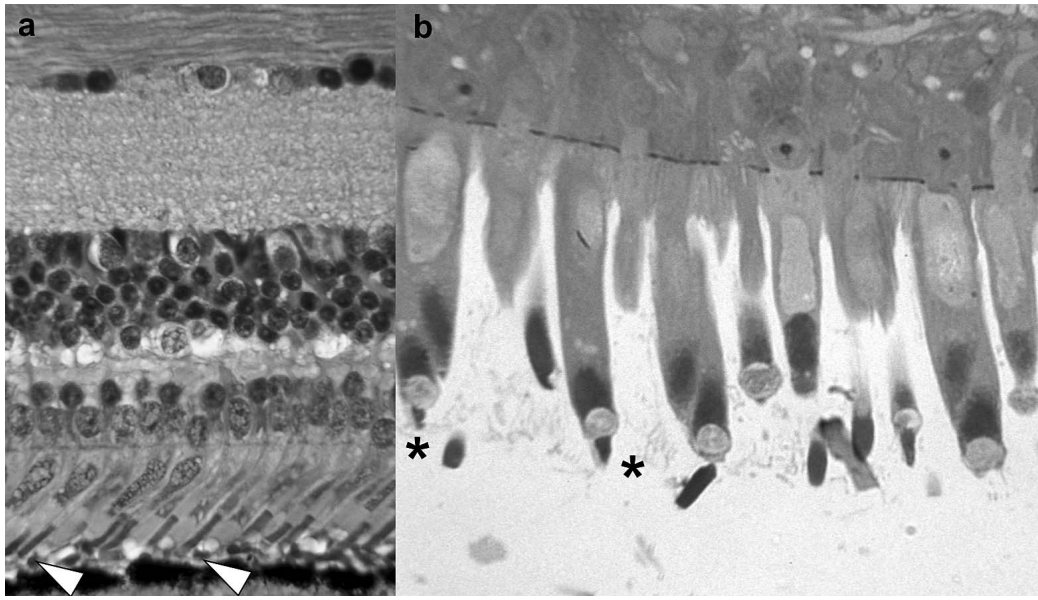

FIGURE 3. Photomicrographs of the retina and choroid of the left eye from case 1, taken near the fovea. (a) Disorganization and blunting of the outer segments and blunting of the retinal pigment epithelium (arrowheads), and a subjective decrease in the number of photoreceptors, cells in the inner nuclear layer, and ganglion cells. (b) Higher magnification showed severe blunting of photoreceptor outer segments (*).

concentration in healthy captive Peregrine Falcons was shown to be between 3.4 and 29.18 μg/mL depending on season (Dierenfeld et al. 1989).

DISCUSSION

The present case series suggested that, in some of the falcons, early onset vision loss from various forms of a primary retinopathy is more likely than other causes (e.g., nutritional, toxic, systemic disease, neoplastic, photic, traumatic). All birds had early onset visual deficits, normal siblings reared in identical conditions, good systemic health, and a general lack of other ophthalmic signs that could be associated with another cause for retinopathy. Although toxic and nutritional causes could not be conclusively excluded, they are unlikely given the systemic condition of the falcons that we examined and the lack of problems reported in their siblings (when they were known). When ancestry was known, neither the parents nor most siblings manifested ocular signs, suggesting that if heritable, the retinopathy is likely autosomal recessive.

The most documented heritable retinopathies in birds are those of the domestic chicken (*Gallus gallus domesticus*), where multiple forms are distinguished (Fite et al. 1982; Fulton et al. 1982; Ulshaffer et al. 1984; Curtis et al. 1988; Semple-Rowland et al. 1998; McKibbin et al. 2014). The scant existence of reports of primary, early onset retinopathies in raptors is probably because disease in free-living birds decreases their chances of survival, as would particularly be the case with profound visual impairment in such a visually dependent animal. Two documented heritable retinopathies in falconiform birds (Murphy et al. 1985) caused severely visual impairment from hatching with sluggish to absent PLRs. Histologic findings in the birds in that study included areas of retinal layer disorganization, rosettes, areas of retinal pigment epithelium (RPE) absence, and choroidal aplasia. In the falcons in our study, nearly all cases had distinct retinal layers without rosette formation. The RPE is important in the development of the neural

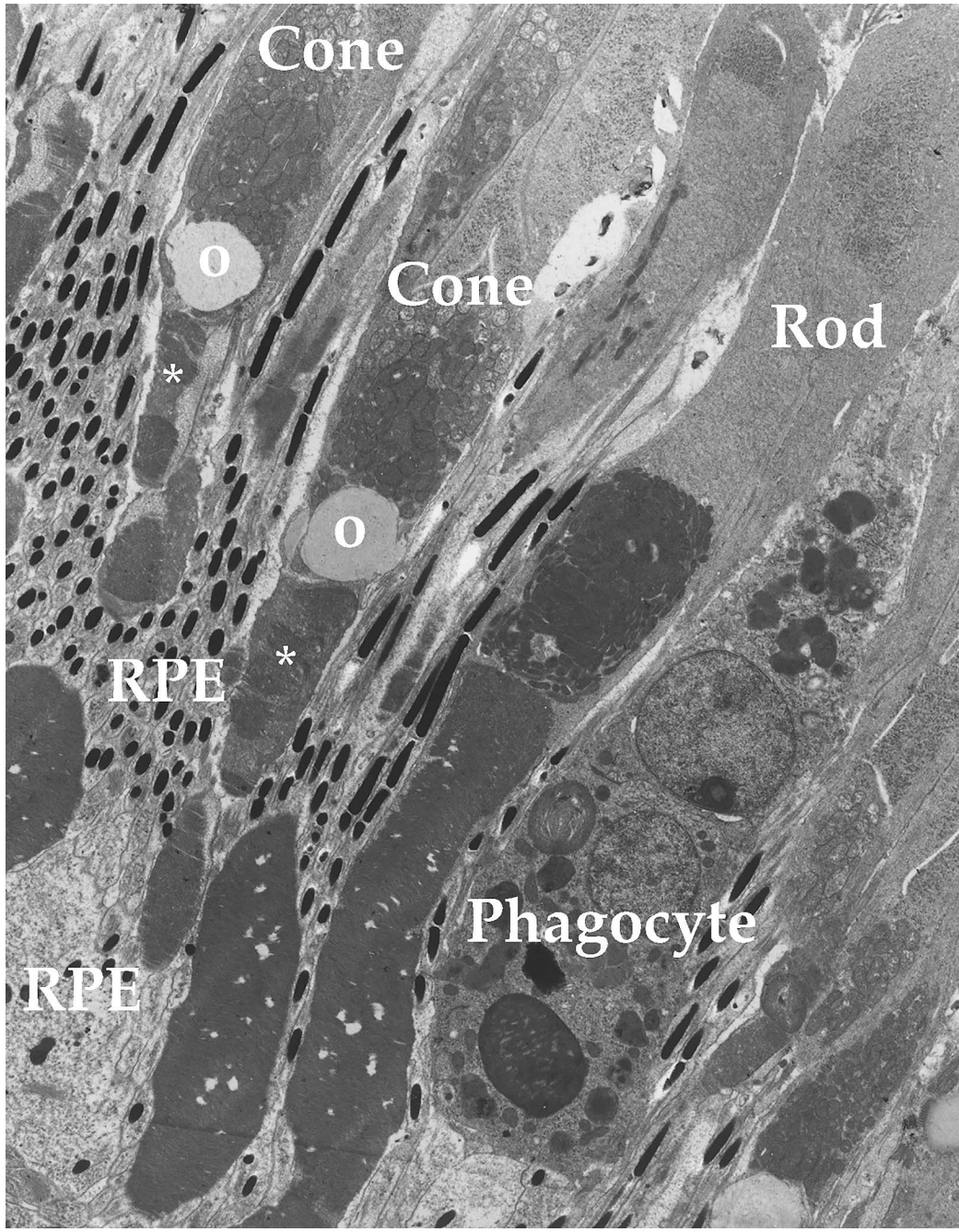

FIGURE 4. Transmission electron microscopy image of case 1. 7,800×. The image shows several photoreceptor inner and outer segments interfacing with processes of the retinal pigment epithelium (RPE). Cones are recognized by the oil droplets (o) and the short conical outer segments. Rods are characterized by the long outer segments. The cone outer segments have shattered membrane discs (*), whereas the rods sampled appear normal. The image includes a phagocytic cell squeezed between outer segments. This cell is out of place and a suggested clean-up function was underway.

retina, and the pathogenesis of generalized retinal dysplasia may involve a disruption in the normal relationship of the RPE and the neural retina (Silverstein et al. 1971). The histologic findings in the cases in Murphy et al. (1985) supported this hypothesis, but based on the histologic findings of retinal dysplasia in three cases presented here (cases 5, 8, 9), retinal dysplasia as a cause of photoreceptor or retinal degeneration is unlikely.

Among nonavian veterinary species, early onset heritable photoreceptor dysplasias and retinal degenerations have been extensively reported in the dog (Goldstein et al. 2010; Narfstrom and Peterson-Jones 2013). The canine heritable photoreceptor dysplasias in general are characterized by early onset visual deficits (typically within first 6 mo of life), usually involving nyctalopia that progresses to total blindness at various rates (Narfstrom and Peterson-Jones 2013). In the dog, maturation of the retina occurs at about 7 wk of age, and this is when visual problems are first noted in most photoreceptor dysplasias (Gum et al. 1984). Although the rate of retinal maturation of a Peregrine Falcon is not known, an earlier maturation could explain why most of the birds presented here showed visual deficits almost immediately after hatching. By the time they are blind from photoreceptor dysplasia, most dogs have funduscopic signs, but these signs are centered around visualization of a tapetum and retinal vasculature, neither of which avian fundi possess. Therefore, in a nontapetal fundus with an anangiotic retinal pattern, the signs of retinal degeneration are more difficult to identify funduscopically. The difficulty in detecting retinal degeneration funduscopically (or the lack of funduscopic lesions) emphasizes the importance of ERGs.

Electroretinography of birds with early onset visual deficits have been reported in chickens and pigeons (*Columba livia*). One study presented chickens with initially only focal retinal degeneration that primarily involved photoreceptors and ERGs at both 13 wk and 12 mo that showed a decrease in b-wave amplitude (Curtis et al. 1988). Amelanotic chickens initially have photoreceptor outer segment degenerative changes and changes to the RPE, followed by ERG diminished c-, b-, and a-wave amplitudes in decreasing severity, with dark-adapted responses being affected more than light-adapted responses (Fulton et al. 1982). In two blind Tippler pigeons (*Columba livia domestica*), blue scotopic stimuli and monocular phototopic ERGs were extinguished (Moore et al. 2004). Histologic analysis showed similar findings as the falcons in the present study, that is, reduced rod and cone numbers, degenerative changes to photoreceptor outer segments, and a decrease in the thickness of other retinal layers (Moore et al. 2004). No ERGs have been performed and reported on raptors with early onset visual defects, but several studies have presented ERGs in several different raptor species (Gaffney and Hodos 2003; Labelle et al. 2012; Kuhn et al. 2014).

Electroretinography in all of our cases showed subnormal photoreceptor amplitudes (Fig. 2). Some animals had a nearly normal PLR despite an extinguished ERG, which could be explained by the low light levels required for photoreceptors to initiate a PLR response, as well as PLR initiation by melanopsin-containing ganglion cells or horizontal cells with high-intensity light stimulation (Contin et al. 2010; Morera et al. 2016). Overall, the interpretation of these findings was complicated by a lack of a matched control or published reference values for this species of falcon (or ERGs in raptors with early onset visual defects). However, based on known positive controls for several eagle and hawk species using the same equipment and protocol, as well as normal ERGs in other raptors, including the American Kestrel (Labelle et al. 2012), the results were suggestive of a decrease in photoreceptor function in both eyes (rod function being more subnormal than cone function). In some cases, the frequency at which fusion occurred appeared to be relatively normal, suggesting that cone function was somewhat preserved.

One individual (case 6) in this series had the unique diagnostic result of a negative ERG. A negative ERG is one in which there is

selective reduction of the b-wave such that its amplitude is less than the a-wave (Koh et al. 2001). Negative ERGs reflect inner retinal dysfunction and, in humans, can be found in conditions such as congenital stationary night blindness, juvenile retinoschisis, fundus albipunctatus, Oguchi disease, diabetic retinopathy, rod-cone dysplasia, and some forms of retinal vascular disease (Nobel et al. 1990; Audo et al. 2007). Negative ERGs have also been shown to be associated with some forms of progressive retinal atrophy in dogs (Kondo et al. 2015; Somma et al. 2016). Electroretinograms dominated by the a-wave have also been described in early retinal degeneration in Norwegian elkhounds (Acland and Aguirre 1987). Although other rod-cone dysplasias have been described in a number of breeds, as discussed above, the ERG response is usually characterized by a progressive decrease in b-wave amplitude and ultimately a subnormal ERG amplitude rather than a true negative ERG (Curtis et al. 1991).

Although it has not been specifically reported in a raptor, the course, presenting signs, and histopathology of the falcons in this report would seem to parallel more closely a photoreceptor dysplasia than either a retinal dysplasia or retinal degeneration. The very early onset of visual problems in most of the birds would tend to rule out retinal degeneration, and the histopathology findings would tend to rule out retinal dysplasia as a cause of the severe, diffuse photoreceptor abnormalities. However, some variability in the cases presented here may suggest different forms of photoreceptor dysplasia. For example, a few individuals had mild asymmetry in visual deficits or ERG findings, but overall both of their retinas were severely affected and they had severely compromised vision bilaterally. Furthermore, one bird had a later onset of presenting with visual deficits; however, clinical presentation, examination findings, and histopathology were still supportive of a photoreceptor dysplasia. In another bird, a congenital abnormality of rod and cone function, as well as inner retinal dysfunction as evidenced by a negative ERG, was present. In birds with known relatedness, we strongly suspected their condition was a heritable form of photoreceptor dysplasia because of a similar progression of disease, clinical examination findings including ERG, and histologic distribution of retinal dysplasia. This condition was well exemplified in cases 3, 4, and 5, and other birds from the same breeding pair were reported to have similar visual deficits. Sequential examinations, better documentation of relatedness to track lineage, and ERGs and ultrastructural studies on normal and affected birds, is required to better understand the mechanisms and establish heritability of retinopathies in raptors.

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