Bilateral vision loss in a captive cheetah (*Acinonyx jubatus*)

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Abstract
The following case report describes a 1-year-old female cheetah (*Acinonyx jubatus*) with bilateral blindness and unresponsive pupils. For comparison, a second healthy 2.5-year-old male cheetah without visual deficits was also examined. Clinical examination of both animals included biomicroscopy, indirect ophthalmoscopy, tonometry, and electroretinography. The young female cheetah showed no menace response, no direct or indirect pupillary light reflex, and no dazzle reflex in either eye. Fundus lesions, as detected by indirect ophthalmoscopy, are described for the female animal. In both eyes, the fundus color was green/turquoise/yellow with multiple hyperpigmented linear lesions in the tapetal area around the optic nerve. The optic nerve head was dark gray and about half the normal size suggesting bilateral optic nerve hypoplasia and retinal dysplasia or differentially optic nerve atrophy and chorioretinal scarring. The ERG had low amplitudes in the right eye but appeared normal in the left eye compared with the male cheetah. Blood levels did not suggest current taurine deficiency. This is addressed to some degree in the discussion. Bilateral optic nerve hypoplasia or optic nerve atrophy is a rare anomaly in cats and has not yet been described in a cheetah.

Key Words: blindness, cheetah, optic nerve hypoplasia, retinal disease, taurine, unresponsive pupils

INTRODUCTION
Optic nerve hypoplasia (ONH) is a rare condition described in humans,1–3 horses,4 cattle,5 cats,6–8 and dogs.9,10 It is a nonprogressive, developmental anomaly of the retina and optic nerve, characterized by visual and pupillary light reflex (PLR) deficits.11 ONH is believed to result from either retinal ganglion cell (RGC) axon guidance defects or abnormal retinal/optic disc development.12 It can present as a uni- or bilateral disease and can arise in association with malformations of the central nervous system.11,11–16 In contrast, congenital absence of the optic nerve is very rare. Bilateral optic nerve aplasia has been described in a 5-week-old domestic cat,17 and in kittens exposed to weekly doses of griseofulvin.18 Optic nerve atrophy is the sequel to any process that damages the RGCs or their axons.

This report describes bilateral vision loss in a young cheetah due to an ONH or atrophy. Ophthalmologic examination and further diagnostic procedures were performed and the disease is discussed. To the authors’ knowledge this is the first report of a cheetah with visual deficits secondary to ONH or atrophy.

CASE HISTORY

History and initial clinical findings
A 1-year-old, female cheetah was presented to the Clinic for Zoo Animals, Exotic Pets and Wildlife because of suspected blindness and fixed and dilated pupils in both eyes and was then referred to the Ophthalmology Service. The animal was imported from South Africa 6 months previously and appeared to be in good general condition. The cheetah was hand reared, initially on puppy milk replacer (Esbilac®; PetAg, Hampshire, IL, USA) and then the cub was later weaned onto a diet of minced chicken (including bones) and supplement Feli-Vit (Kyron Laboratories, South Africa) at 30 days of age. After approximately 2 weeks, red meat (mainly donkey) was slowly added to and replaced the chicken. From two and a half months of age, cubs were receiving only red meat with Feli-Vit (Kyron Laboratories)
bone meal and MIRRA-COTE® (Kyron Laboratories). In this adapted situation in a wildlife range in South Africa no visual deficits could be recognized. According to the owner, first signs of impaired vision were noticed briefly after the relocation from South Africa when the cheetah was playing in her new surrounding area in Switzerland. The cheetah was kept together with four other animals in a private zoo in Switzerland and fed a regular diet of beef, raw poultry without bowels and carnivore supplement (KLIBA NAFAG, Kaiseraugst, Switzerland). The animal had been vaccinated twice with 1 mL of Fel-o-vax® (Fort Dodge Animal Health, Fort Dodge, IA, USA) against feline rhinotracheitis, feline panleucopenia, feline calicivirus, feline pneumonitis and feline leukemia, and once with 1 mL Rabisin® (Merial, Hallbergmoos, Germany) against rabies and regularly dewormed with a pyrantel palmitate (Nemex™-2; Pfizer, Exton, NY, USA).

At the same time, a clinically healthy 2.5-year-old, male cheetah from the same owner was examined. The data from this animal served as points of reference to which the female cheetah’s results are compared. The male cheetah is one of the other four animals living on the same premises and fed the same diet. He showed no vision related problems and is not related to the female cheetah.

**Ophthalmologic examination**

Both cheetahs underwent a complete physical and ophthalmologic examination. Before anesthetizing the female cheetah with a ketamine–medetomidine combination (ketamine: 3 mg/kg i.m. [Narketan®; Vétoquinol, Ittigen, Switzerland]; medetomidine: 50 µg/kg i.m. [Pfizer, New York, USA]), a limited examination including the menace response, the PLR and the dazzle reflex was performed. The healthy adult male cheetah could only be examined under anesthesia and showed no ocular abnormalities. Both eyes of the young female cheetah with visual deficits appeared normal in size and were open (Fig. 1). Menace responses, direct and indirect pupillary light reflexes, as well as dazzle reflexes were absent in both eyes. On biomicroscopy, the eyelids, conjunctiva, cornea, anterior chamber, and lens were normal. Both pupils were maximally dilated, circular in shape and unresponsive to light, although they did constrict during anesthesia. On indirect ophthalmoscopy of both eyes, the fundus appeared green/turquoise/yellow with multiple hyperpigmented linear lesions in the tapetal area around the optic nerve head that were more numerous in the ventral part of the fundus. The optic nerve head was dark gray, small and about half of the normal size. The ONH appeared recessed (Fig. 2). The peripapillary area appeared dark. A fundus photograph of the healthy cheetah is shown for comparison (Fig. 3). The fundus photographs were taken with a Kowa Genesis-D digital fundus camera (Kowa Company, Tokyo, Japan). Intraocular pressure (IOP) was measured with both animals under anesthesia using a TonoVet. The female cheetah had an IOP of 28 mmHg in the left eye (OS) and 30 mmHg in the right eye (OD). The male cheetah had an IOP of 30 mmHg OS and 32 mmHg OD.

**Results of additional diagnostic procedures**

Electroretinography was performed in a closed dark treatment room for both eyes of the two cheetahs after a 5-min period of dark adaptation. Tropicamide was instilled half an hour before the procedure in the male cheetah. An Echtest-Palst electroretinography lens was placed on the cornea. In this type of lens the reference electrode touching the palpebral conjunctiva is incorporated together with a platinum corneal electrode. A needle electrode was used as a subcutaneous ground electrode positioned at the back of the skull. Eight white light (LED) stimuli of approximately 2–3 Cd/
m²/s were averaged. The ERG of the healthy male cheetah illustrated an a-wave of 2.1 µV and a b-wave of 29.0 µV in the left eye (Fig. 4). The right eye could not be checked due to weakening of anesthesia. The ERG of the young female cheetah showed an a-wave of 9.3 µV and the b-wave measured 56.4 µV in the left eye (Fig. 5). The right eye showed an a-wave of <10 µV and lacked a b-wave (Fig. 6). A complete blood hematology and chemistry was done in both animals and revealed no abnormal findings. To rule out feline central retinal degeneration, plasma taurine levels were determined. With a concentration of 76 µmol/L in the female and 169 µmol/L in the male cheetah, these results were within the reference range known in cats (50–120 µmol/L, 31–147 µmol/L, and 70–82 µmol/L). Taurine plasma concentrations of randomly sampled zoo felids fell within reference ranges for domestic cats (80–120 µmol/L) although the ranges for cheetahs seemed to be slightly higher (158.2 ± 73.6 µmol/L). Values <30 µmol/L are considered deficient.

**DISCUSSION**

Deficits in vision in young domestic cats due to congenital abnormalities of the retina and optic disc in combination with agenesis of the eyelids, microphthalmia, and choroidal colobomas have previously been reported. Retinal dysplasia associated with intrauterine or early neonatal intraocular viral infections like feline leucopenia virus (FeLV), feline panleucopenia virus, and feline infectious peritonitis (FIP) has been described in domestic cats. Cheetahs are reported to be highly sensitive to at least one of these agents (FIP). The multiple pigmented lesions in the retina of the cheetah presented here differ from the chorioretinitis that characterizes the pathology of most of these agents. They may be characterized more as a retinal dysplasia with multiple folds. Furthermore, no clinical signs of other ocular lesions as uveitis were presented.

Dietary taurine levels above 50 µmol/L have been suggested to be necessary to prevent retinal disease in felids. Feline retinal degeneration in captive cheetahs is thought to be associated with taurine deficiency. In the present

![Figure 3](image3.png)

**Figure 3.** Normal fundus of the male cheetah (*Acinonyx jubatus*).

![Figure 4](image4.png)

**Figure 4.** ERG of the healthy male cheetah (*Acinonyx jubatus*).
case, however, plasma taurine levels were within normal range compared to references from domestic cats and a bit low but not deficient compared to a study including cheetahs. The taurine concentration of the female cheetah, however, was at least half of the blood result in the male cheetah in this study. Although no distinctive lesions of the area centralis could be identified, the reduced taurine level hypothesis argues against ONH. Another explanation for the ERG changes could be that the b-wave amplitude was significantly reduced due to an abrupt miosis observed in both eyes after about 15 min of anesthesia. Because both pupils were completely dilated and unresponsive prior to anesthesia, tropicamide was not applied in this animal. Pupil size has been shown to have a profound effect on the b-wave amplitude in dogs.\textsuperscript{31} But it cannot be completely ruled out that the female cheetah was fed a taurine-deficient diet and developed retinal lesions before she came to Switzerland. The retinal damage is irreversible, but taurine supplementation prevented further deterioration.\textsuperscript{32} In dogs, a Vitamin E deficiency may result in pathologic changes in the retina.\textsuperscript{33} Ophthalmoscopic signs of the disease develop early and were described as a mottled tapetal fundus appearance in contrast to the findings in the young female cheetah. The small and recessed ONH suggests a lack of RGC axons in the retinal nerve fiber layer and ONH. The visual deficit, absent PLR and normal ERG in at least one eye suggests a diagnosis of ONH. Similar ONH lesions are seen in cases of micropapilla. In these cases, however, PLRs are present and only subtle vision deficits would be

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recognized. In optic nerve aplasia, neither ONH nor retinal vessels would be present. This has been reported by Barnett and Grimes in a 5-week-old male black and white longhaired kitten presented with a history of total blindness. Optic nerve atrophy could have a similar appearance. Electroretinography is the recording of changes in retinal potential in response to changes in illumination. While not synonymous with vision, it provides a reasonable estimate of the functional integrity of the outer retinal layers. An abnormal ERG was found in this young cheetah, as previously described in two dogs with ONH by Gelatt and Leipold. The small a-wave and the absence of the b-wave in the right eye suggest the presence of additional retinal disease. A study about the use of medetomidine on ERG of normal dogs demonstrated a prolonged implicit time and lower amplitude of both the a- and b-waves at all flash intensities. Compared to general anesthetics as isoflurane alone or in combination with thiopental, medetomidine, medetomidine-ketamine, xylazine-ketamine and tiletamine-zolazepam affect the ERG less. Intraocular pressures appeared to be high for anesthetized animals. This could be due to the use of ketamine in both cases. For comparison a study by Ofri about IOP in lions resulted in age with ONH by Gelatt and Leipold. The small a-wave in a 12-year-old male cheetah suspected to suffer from leukoencephalopathy. This particular animal was presented with progressive hind limb ataxia and incoordination for 22 months. No neurological signs could be detected in our cheetah, however. Bilateral blindness as encountered in this female cheetah has not been seen in a number of closely related animals. This renders an inherited defect rather unlikely. In our cheetah, the disease was bilateral and the visual function markedly reduced. Apart from the fundus lesions, no other ocular malformations were observed. The cheetah was captive raised and hand reared, therefore a deep attachment to the keepers is suspected. To the author’s opinion this is a reason why the visual deficits were noted relatively late in the cheetah’s life. The cheetah is still in the Zoo in Switzerland. She is a very friendly animal, however, sometimes she react anxious to the visitors due to her impaired vision. Based on these considerations, we conclude that the case reported here represents a rare developmental disorder.

REFERENCES