PALPEBRAL COLOBOMA IN THREE SNOW LEOPARDS

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Since 1976, when Carl Wahlberg, (Wahlberg, 1978)[1] first described multiple ocular coloboma (MOC) in snow leopards (Panthera unica) other offspring in more than one zoo have been detected with this defect. Ocular coloboma, a congenital defect, is a malformation of any portion of the globe or adnexa, and can occur as single or multiple defects. The exact cause of MOC has not been determined but chromosomal, genetic, toxic and infectious etiologies have been examined by Wahlberg (Wahlberg, et. al., 1980)[2,3]

In the Henry Doorly Zoo, Omaha, NE, three of four snow leopard cubs in two separate litters, with the same sire (OMAHA 1) and dam (OMAHA 3) have been born with palpebral coloboma. Figure 1 shows the pedigree of these animals. All four were removed from the dam within two days after birth to hand raise because of the poor maternal care exhibited by the dam (nervous motions, repeatedly leaving the cubbing den, not allowing nursing, carrying the cubs and covering them in the bedding). All activity was observed on close-circuit television to avoid human disturbance of the dam and cubs. The decision to remove the cubs for hand rearing was made because of the lack of nursing activity and apparent weakness of the cubs.

The first litter, born 29 August 1977, contained two males (cub 1 and 2). These animals were removed for hand-rearing on the day of birth due to the dam's behavior of repeatedly carrying them in her mouth, resulting in superficial skin wounds in both offspring.

During the initial physical examination, it was noted that the eyes were open in cub 1. The upper palpebral margins, bilaterally, exhibited incomplete formation in 3 to 4 mm. defects of the midpoint of the lid margins. This defect extended dorsally, creating a fissure in the upper lids and a resulting corneal keritiitis. The cub ate and gained weight (from initial birth weight of 484 gm) for five days. During this period, body temperature fluctuated.

On day 6, the tarsal joints were swollen, food consumption decreased, body temperature remained near 101°F. The right cornea became cpaque by day 7 and the globe enlarged. Subsequently, sepsis of the right eye necessitated enucleation. His condition deteriorated leading to death on 12 September 1977.

Cub 2, weighing 453 gm, was apparently healthy and had both eyes closed; the right eye opened on day 10, the left on day 17. The right cornea exhibited keratitis upon opening; after initial antibiotic ointment and artificial tears brought about no improvement, the palpebrae were sutured closed to protect the eye. Ointment and drops were placed in the left eye as it showed mild keratitis. The right eye remained sutured for two weeks; topical ophthalmic therapy was continued for six weeks before the corneas began clearing. At eight weeks of age, this cub began exhibiting signs of anorexia, lethargy, dehydration, and diarrhea. Supportive therapy resulted in little improvement. On 4 November 1977, on day five of illness, Haemobartonella felis was confirmed on a peripheral blood smear. The cub's condition worsened despite intensive therapy and he died on 7 November 1977.

The second litter, containing male cubs 3 and 4, (weighing 431 gm and 467 gm respectively), was born 18 May 1980. Again, the dam exhibited poor maternal behavior; however, she was given thirty-six hours to care for and nurse the cubs. She continued exhibiting similar behavior as in August 1977 requiring removal of the cubs to the zoo nursery. In cub 3, the left eye opened the ninth day. At this time, a 3-4 mm defect (palpebral coloboma) was noted in the central portion of the left, upper eyelid. The right eye opened on day 10 and upon exam, a pinpoint palpebral coloboma was seen in the middle of the upper lid. On 29 May 1980, the animal exhibited hyperthermia, anorexia, and weakness. An immediate blood smear revealed Haemobartonella felis in the enythrocytes. By that evening, the animal's condition deteriorated and he died. His littermate, cub 4, had his right eye open at birth and a palpebral coloboma was diagnosed involving the middle third of the right upper eyelid margin. Treatment had to be instituted for keratitis of this eye, as the cornea was clouded slightly and extremely dry. Minute vascular loops in the anterior chamber were overlying, but not obstructing movement of, the pupil. The lens appeared clear, but was difficult to visualize due to the clouded cornea. After diagnosing H. felis in cub 3, a blood smear from this cub prepared 29 May 1980 was examined and no blood parasites were seen. He was placed on oxytetracycline prophylactically despite the apparently normal blood smear. On 4 June 1980, a second blood smear was positive for H. felis. At this time, the cub had become anorectic, had thermo-regulatory difficulties, and was lethargic. Low packed cell and low blood hemoglobin findings led to a decision to administer a whole blood transfusion, with the sire as the blood donor on 5 June 1980. No improvement was noted and the cub died on 6 June 1980.

Histopathologic examinations were performed on the tissues from all four cubs at the Veterinary Diagnostic Lab, Lincoln, NE, and the Veterinary Pathology Department, Iowa State University, Ames, IA. The findings, from the tissues examined (brain, heart, lung, liver kidney, adrenal glands, spleen, skeletal muscle, bone marrow, thymus, eyelids and globes) revealed nothing more specific than a generalized inflammatory condition. No specific etiological agent was identifiable. Only in cub 4 was there a specific fibrinous bronchopneumonia diagnosed; a pure culture of E. coli was found in this pulmonary tissue.

Early management of these cubs, as well as all hand-reared animals in the Henry Doorly Zoo nursery includes the following:

1. Day 1 - arrival in the nursery
    a. physical examination
    b. initial body weight and body temperature measurements
    c. placement in controlled temperature/humidity human infant incubator
    d. isolation from contact with any other offspring in nursery.
    e. vitamin supplementation parenterally, as well as beginning prophylactic antibiotic therapy.
    f. beginning feeding
    g. species specific blood serum given orally if infant was never observed to nurse. This is added to each feeding for the first 48-72 hours. In addition, feline offspring receive hyper-immune feline serum subcutaneously on day 1 or 2.

2. Day 2 and subsequent days

   a. blood sample taken day 2 or 3 for initial packed cell volume, hemoglobin, total serum protein, white blood cell count, and differential count, and blood smear examination.

   b. body temperature recorded every twelve hours.

   c. daily body weight measurement

   d. feeding - recording volumes offered and actually consumed

   e. vaccinations as required or additional hyperimmune serum if deemed necessary.

If the animal is apparently healthy, this routine is followed. Antibiotics will be given for five days and discontinued if no indication is seen for continuance. Any abnormalities noted in the infant results in the appropriate therapy added to the daily routine. With this procedure being standard, little opportunity is available for infants to be exposed to infectious agents. The similar deterioration of the health of all four of these cubs, with the same geneological backgrounds, indicates these animals may have had more undetermined defects than the observed coloboma. The Haemobartonella felis is felt to have been transmitted from the dam to the fetuses intrauterinally. Others have suggested this as a possible source of transmission in the domestic cat. (Hathaway 1976., Harvey, 1980).[4,5]

Since the cross-breeding of sire OMAHA 1 and dam OMAHA 3 (a father-daughter cross) resulted in two consecutive litters of cubs exhibiting palpebral coloboma and subsequently not surviving, a different breeding strategy was followed in late 1980. The same dam, OMAHA 3, was bred to a different male, namely MILWAUKEE 7, an animal totally unrelated to the Omaha female.

Additionally, it was decided to prophylactically treat the dam for H. felis with oxytetracycline therapy during the third trimester of pregnancy. On 13 April 1981, OMAHA 3 delivered two female cubs (cubs 5 and 6). It was immediately apparent that the cubs were larger than the previous four, were stronger and more active (actively seeking out the dam when she was in the cubbing den), and that the dam was exhibiting a much better maternal behavior toward the cubs. It was decided to leave the cubs with the mother as nursing was observed and she seldom left the cubbing den. The cubs were weighed on day 11, (cub 5 weighed 693 gm, cub 6 weighed 662 gm); the eyes on both cubs opened on day 15. A short examination on this day revealed normal eyelids and apparently normal corneas. On 16 July 1981, the cubs received standard feline viral vaccinations and blood samples were taken. No evidence of H. felis could be found on blood smears. The cubs have remained with the dam and to date, have grown normally. A complete opthalmologic examination is planned for each cub on the occassion either has to be immobilized in the future.

Summary of medical/behavioral findings in litters exhibiting coloboma compared with non-affected litter.

1. lower birth weights
2. decreased amount of activity, decreased "strength" of activity
3. poor maternal care toward litters exhibiting coloboma
4. presence of palpebral coloboma in three of four cubs, corneal keratitis in the one non-colobomatous cub

5. Haemobartonella felis diagnosed in three of four cubs (two with palpebral coloboma)
6. Non-survivability of all cubs from affected litters - all exhibiting similar general inflammatory conditions as seen on histopathology.

There have been no disease outbreaks in the cat complex, which houses 52 individual large felids, including the snow leopards, since its completion in 1976. Several species have delivered many normal, healthy, surviving cubs since 1976. The adult snow leopards themselves have not exhibited any disease conditions. This history, linked with the wide separation of time between the two affected snow leopard litters, (1977 and 1980), detracts from support for infectious or toxic agents being the etiology for the observed coloboma. The facts that all affected cubs were products of a father-daughter cross-breeding and that the breeding of the same dam with an unrelated male produced apparently normal offspring supports the theory that the observed coloboma may be a hereditary defect. If future eye exams reveal no ocular abnormalities in cubs 5 and 6 and future breedings between OMAHA 3 and MILWAUKEE 7 produce no more affected offspring, then the theory of a hereditary factor being the etiological agent of coloboma will be supported further. We feel that the low birth weights, "weak" cubs, poor maternal care, presence of Haemobartonella, and susceptibility to infection or inflammatory conditions are indicators that the cross-breeding of OMAHA 1 and his daughter, OMAHA 3, produced offspring deficient in many more ways than merely expressed in the palpebral coloboma.

This paper is not intended to provide definitive data to incriminate any specifid etiologic factor in coloboma in the snow leopard. It merely provides additional information to expose the fact coloboma is occuring in non-related groups of snow leopards in widely separated locations. Obviously, much investigative work has to be done in the future to clearly define specific etiologic factors to multiple ocular coloboma as it has been diagnosed in the snow leopard.

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