

Leopard Gecko Diseases and Care

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CAPTIVE CARE

Leopard geckos (*Eublepharis macularius*) are the second most common species of reptile (13% of all reptile cases seen) seen in private practice. *Eublepharis* means true eyelids; *macularius* derives from macula, Latin for spot or blemish. The leopard gecko is native to south central Asia from southern Afghanistan and perhaps Iran, throughout Pakistan and northwestern India. They are crepuscular and nocturnal, hiding under rocks or in burrows during the day. Cold winter conditions force them to brumate. They breed readily in captivity and almost all are captive born. Leopard geckos weigh 45 to 80 grams, are 20 to 25 cm in length, males being larger than females, and can live twenty, to almost thirty, years. There are many color variations and they are great beginner pets!

Leopard geckos do well in 10 or 20 gallon aquariums with built in sliding screen tops to keep out predators, such as cats. Large aquarium stones or gravel (too large to be ingested), newspaper, indoor outdoor carpeting, or reptile carpet are good substrates. Avoid sand, fine or small gravel, wood chips, crushed walnut shells or other ingestible substrates. A water bowl and small dish of calcium powder for reproducing females should be continuously available. Stacked flagstone makes excellent retreats. A combination nest/hide box can be made from small sealable plastic containers with a hole in the lid (such as a Tupperware sandwich box). This should be half filled with moist vermiculite, to boost humidity, critical to avoid stuck sheds on the toes and tail tip. An open hutch or log will not work. Shedding is nocturnal and is eaten, thus generally not observed. Juveniles shed every two weeks while adults shed monthly. Leopard geckos defecate neatly in one or two corners. Cage temperature should include a gradient from 70 to 90°F. Supplemental heat can be provided with an overhead 75 watt incandescent light bulb, for 10–12 hours of light daily, or under tank heater not attached directly to the glass. Hot rocks can result in ventral burns. A two month winter brumation with temperatures from 50 to 65° F is ideal. Males should not be housed with other males, as fighting results in serious wounds. One male can be housed with any number of females; they live in loose colonies in the wild. Sexing is possible after three months of age. Males have more prominent femoral pores and paired hemipenial bulges at the base of the tail; as adults males have a heavier build and a broader head. Leopard geckos are seasonally polyestrous. From January to September, females lay clutches of two eggs every three to four weeks for a total four to ten clutches (typically six). Females can retain sperm for one year. Eggs can be incubated at 80 to 90°F, and they hatch in about 55 to 60 days. Lethal egg temperatures are below 75°F and above 95°F. Temperature dependent sex determination produces mostly males at incubation temperatures during the first two weeks between 88 to 91°F. Females predominate above or below these temperatures. At 80°F most hatchlings will be female; at 90°F most hatchlings will be male. Females born at hotter temperatures, “hot females”, are more aggressive and often infertile. Hatchlings shed within a day or two then begin feeding within four to five days on pinhead crickets, wax worms and mealworms. Good calcium support is vital. Leopard geckos do not seem to require ultraviolet light, perhaps because they are nocturnal, provided they get vitamin D³. One study found increased shedding and erythema in leopard geckos exposed to UV light, consistent with sunburn. Hatchlings have a banded pattern which gradually gives way to the adult spotted pattern. If properly fed they should reach adult size and breed within their first year. Geckos should be fed daily to two to three times per week. Egg productivity declines after seven to eight years; some breeders retire females after they have laid 70 eggs.

CLINICAL DISEASES

Unsupplemented crickets and mealworms are an inadequate diet that leads to rampant nutritional deficiencies (chronic malnutrition, hepatic lipidosis, hypovitaminosis A, nutritional secondary hyperparathyroidism). Prey insects should be fed a calcium rich diet (usually greater than 6 to 8% Ca) for

several days, i.e., gut-loaded, and be dusted with calcium just prior to feeding. The third aspect of feeding is to offer a wide variety of insects; commonly available commercial insects (crickets, mealworms, wax worms, superworms), should be supplemented with commercially available silkworm larvae, tomato hornworms, cockroaches and wild caught moths, crickets, cicadas, grasshoppers, and sow bugs (isopod crustaceans, pill bugs or roly pollys). Owner concerns about pesticides in wild caught insects have not been a problem; fireflies are toxic and should never be fed to reptiles. Owners are surprised to learn that leopard geckos eat insects, lizards and scorpions in the wild. As adults, leopard geckos take pinky mice, especially females. Start with one to two day old live pinkies until they are comfortable eating them, no more than one per week. Eventually geckos will take thawed frozen pinkies if dangled in front of them. It is hypothesized that a high fat diet from too many pinkies may predispose females to xanthomas, although all five of Garner, *et al* case series did not feed on pinkies.

Multivitamins once or twice a month, or good insect gut loading diets, seem to be an adequate source of vitamin D₃ for leopard geckos. Multivitamins are not a good source of calcium irrespective of what the label implies. Also be aware that many reptile multivitamins contain β -carotene instead of preformed vitamin A. Recall that β -carotene comes from plants and retinyl esters (preformed vitamin A) comes from animals, so insectivorous reptiles need preformed vitamin A. Leopard geckos do not seem to be able to convert β -carotene to vitamin A. As a consequence, hypovitaminosis A is common. Human multivitamins (Centrum) are better than reptile multivitamins lacking vitamin A. Pinkies are a good source of vitamin A for adults.

Hypovitaminosis A results in decreased appetite, difficulty catching prey, retained hemipenial plugs, dysecdysis on the body, stomatitis, and eye problems, such as keratitis, conjunctivitis, blepharitis, and periocular abscesses. Chronic cases can result in corneal scarring and blindness, very similar to poorly managed keratoconjunctivitis sicca cases in dogs. Early symptoms include decreased tear production with blepharospasm and mucous buildup in the eyes. Over time the eyelids slowly swell shut and white to yellow, semi-solid to solid, keratinaceous, inflammatory cellular debris with bacteria accumulates under the eyelids. Corneal ulcers and bacterial infections may develop secondary to decreased tear production. The keratinaceous plugs should be removed under anesthesia; the eyes should be flushed and treated with broad spectrum systemic and ocular antibiotics. Usually 0.01 mL of vitamin A/D (500,000 IU/ml vitamin A) SC, repeated in two weeks, will correct the problem, in addition to correcting diet, if hepatic lipidosis has not developed secondary to anorexia. Ocular improvement takes two to four weeks.

Hemipenial casts or plugs are related to hypovitaminosis A as well and are easily expressed under surgical anesthesia (chamber induce with isoflurane, takes about 15 minutes to get no toe pinch reflex in rear legs). Left untreated hemipenial cast can abscess ventrally or laterally. Everted necrotic hemipenes should be amputated. Leopard geckos with large hemipenial casts should be anesthetized for removal.

Massive subcutaneous periocular abscesses probably involve hypovitaminosis A and periocular or lacrimal glands. Diffuse cellulitis can accompany solid cellular debris. Treatment consists of lancing and debridement under anesthesia, aerobic culture with sensitivities, antibiotics, pain medications, vitamin A supplementation and nutritional support. Occasionally a second surgery is required to completely resolve large abscesses.

Most eye problems are related to hypovitaminosis A. Some leopard geckos are born with deformed eyelids which lead to chronic ulceration and infection. Treatment is unrewarding and requires a dedicated owner to constantly flush and lubricate the eyes.

Another common consequence of poor diet, as well as disease, is hepatic lipidosis. A white liver is often visible ventrally (normal liver color is mahogany red). Treatment consists of long term nutritional support via stomach tube until the patient is eating well on its own. This may take weeks. Stomach tube 5 ml/kg LaFeber's® Emeraid™ Nutri-Support™ (LaFeber Co., Cornell, IL) BID to QID until the patient is eating well, usual six to eight weeks. Multiple small feedings are better than less frequent larger feedings. Emeraid Nutri-Support has a source of vitamin K activity and vitamin B12. Other supplements, such as L-carnitine (250 mg/kg PO q 24 hrs), methionine (50 mg/kg PO q 24 hrs), taurine, S-adenosylmethionine (30 mg/kg PO q 24 hrs), lactulose (0.5 ml/kg PO q 24 hrs), and *Silybun marianum* (extract from milk thistle, 4–15 mg/kg PO q BID to TID) can also be used, however no prospective clinical trials have been

conducted to evaluate efficacy of any of these drugs in reptiles. Consider screening for parasites, especially cryptosporidiosis.

Nutritional secondary hyperparathyroidism is unfortunately still all too common, especially in growing geckos. History typically reveals unsupplemented mealworms and/or crickets. Clinical signs include lack of growth, anorexia, difficulty catching prey, inability to raise body and tail off the ground while walking, bowed long bones (especially the radius and ulna), walking on the wrist or forearms (not plantigrade), pliable mandible and maxilla, kyphoscoliosis or lordosis. Stuck shed may be present. Radiographs and blood chemistry can be considered but aren't critical to diagnosis. Treatment consists of tape splints or cage rest, nutritional support (5–10 ml/kg LaFeber's® Emeraid™ Nutri-Support™ (LaFeber Co., Cornell, IL) or Oxbow Carnivore Care (Oxbow Animal Health, Murdock, NE) via stomach tube twice daily), calcium supplementation (1 ml/kg calcium glubionate orally BID for 1 to 3 months), vitamin D (400 IU IM, repeat in 1 week), and calcitonin injections (50 IU/kg IM, repeat in 1 week), once normocalcemic or one week after starting calcium supplementation. Once the gecko starts gaining weight, clinical signs improve rapidly; this often takes two to four weeks. For severe clinical signs such as paresis or paralysis, inability to urinate or defecate, and/or dramatic spinal deformity, consider euthanasia. Treatment generally takes months so make sure the owner is committed.

Phalangeal dysecdysis is ubiquitous and secondary to multiple retained sheds on the digits from low humidity. As retained sheds build up they progressively restrict blood circulation and avascular necrosis develops. This is easily avoided by providing a moist hide/nest box (see previously). Retained sheds can be carefully removed after soaking on damp paper towels or the toes may need to be amputated under local or general anesthesia. Systemic and topical antibiotics may be indicated.

Leopard geckos autotomize or their tail under duress through natural fracture planes with minimal blood loss. Tame geckos generally will not lose their tail, unless restraining them for a blood draw or grabbing the tail as the gecko vigorously tries to escape. For safe tail venipuncture, chamber induce with isoflurane first. Tail loss is followed by wound contraction, clot formation then epithelialization. Eight to 15 days after tail loss, regeneration begins with a central pink blastema and repigments after 25 days. The tail regrows in one month, shorter and thicker, shaped more like their head.

Intestinal impactions are common in geckos housed on sand, small gravel, wood chips or crushed walnut shells, from substrate ingested while eating prey. Calcium enriched reptile sands should not be used as substrates. Clinical signs can include lethargy, anorexia, tenesmus, and cloacal eversion. Radiographs and palpation are diagnostic. Treatment consists of nutritional support and repeated stomach tubing with a 50/50 mixture of petroleum jelly (such as Laxatone, Tomlyn Products, Division of Vetoquinol, USA, Fort Worth, TX) and water, or mineral oil, until defecating and eating normally or surgery. Cloacal or colonic prolapse are occasionally a sequelae of intestinal obstruction. Mild cases can be reduced under anesthesia; celiotomy and coelomic pull through and tacking to the body wall is advisable for severe prolapses.

Diarrhea has multiple etiologies. Leopard geckos normally have firm, solid, dry feces deposited in one or two corners of the cage. Watery, semi-formed, or smeared stools, or stools outside the normal defecation areas, are abnormal, especially with undigested insects. If the patient is emaciated consider cryptosporidiosis (see below). Protozoan colitis is extremely common, with or without cryptosporidiosis. Flagellated protozoans, such as Trichomonads, are easy to find in flocculent material at 1000x magnification on fresh colonic washes but rarely show up on fecal floatation. Treatment consists of 50 mg/kg metronidazole PO EOD x 5 to 10 treatments, or until repeat colonic washes are negative. Other alimentary parasites, much less common than protozoans, can be found on fecal floatation and direct smears. Bacteria, fungi, and very rarely viruses, can also cause diarrhea. Unidentified mobile spiral bacteria, seen on colonic wash direct smears, are also associated with diarrhea in leopard geckos and responds to metronidazole and enrofloxacin. Leopard geckos on long term oral enrofloxacin can develop filamentous fungal overgrowth which is responsive to ketoconazole, itraconazole, or fluconazole, all well tolerated by leopard geckos. Foreign bodies, especially sand or gravel, can be ruled in or out with radiographs. Coccidia are highly contagious and treated with 30 mg/kg ponazuril (Roadrunner Pharmacy, Phoenix, AZ) PO q 48 hrs for two treatments. A reovirus-associated syncytial cell enteropathy and hepatopathy was diagnosed in two to three-week-old thin leopard geckos, that died with sepsis,

gastric and hepatic necrosis, and intestinal bacterial overgrowth. Leopard geckos with weight loss and death were found to be infected with both eublepharid gecko adenovirus and *Cryptosporidium* spp.

Cryptosporidiosis remains a common disease of leopard geckos, especially in colonies with multiple deaths, and emaciated geckos less than one year. Lizard *Cryptosporidium* is not zoonotic and does not come from mice. It may be contagious to other reptiles, but is not the same as the gastric *Cryptosporidium* which infects snakes (*Cryptosporidium serpentis*). Leopard gecko *Cryptosporidium* was originally named *C. saurophilum*, but recent gene sequencing indicated that *C. saurophilum* was identical to a previously named *Cryptosporidium varanii*, which took precedence. Cryptosporidia species are resistant to disinfection, very contagious, and results in mortality of about 50%. Geckos may survive as carriers or subclinical cases and shed oocysts in their feces. Stress, such as shipping, lack of heat, overcrowding, dehydration, irregular photoperiod, excessive periods of darkness, or poor diet, may induce disease in carriers at a later time. Extreme emaciation, especially in the tail, or loss of tail, accompanies cryptosporidiosis in leopard geckos so it is also called going light syndrome, skinny tail, pencil tail, stick tail, or even dropped tail disease. Early signs are regurgitation of shed skin and decreased appetite, weight loss, then regurgitation of undigested food. Geckos become anorexic with diarrhea (cottage cheesy stools), emaciated and eventually die. Regurgitation is not always prominent in geckos or can be confused with feces. Symptomatic geckos have no fat deposits in their tail, a visible vertebral column, and sunken eyes. Cryptosporidiosis causes a lymphoplasmacytic proliferative enteritis, with hyperplasia and hypertrophy of enterocytes, which interferes with digestion and absorption. Commonly associated with cryptosporidiosis are hepatic lipidosis, hepatic granulomas and gastroenteritis. Cryptosporidia are found in all sections of the GI tract, including tongue (glossitis), esophagus, stomach, small and large intestines, but most commonly in the small intestine, and less commonly the stomach. The easiest method of diagnosis is necropsy and histopathology with an experienced reptile pathologist; save some frozen GI tissue for PCR if histopathology is negative. Newer diagnostics, such as PCR testing for *C. saurophilum*, are more sensitive and have supplanted older diagnostics such as acid fast stains, and immunoassays (copro-antigen ELISA for *Cryptosporidium* [ProSpecT®, Remel, Lenexa, Kansas, USA], or MeriFlour Crypto & Giardia Immunofluorescent Assay [Meridian Biosciences Inc., Cincinnati, OH]). Cryptosporidia are shed intermittently from the intestines so a negative fecal does not rule it out. To improve fecal shedding, force feed for two to three days then collect at least three consecutive fecal smears for testing. Many drugs have been tried in leopard geckos for cryptosporidiosis without long term success including azithromycin, spiramycin, metronidazole, nitazoxanide, paromomycin, rifaximin, hyperimmune bovine anti-cryptosporidial colostrum and herbal extracts such as thymol and creosote. At this time no treatment has proven to eliminate cryptosporidiosis in leopard geckos. Paromomycin (100 mg/kg PO q 24 hrs x 7 days, then weekly thereafter) showed promise in resolving clinical signs, but geckos start shedding Cryptosporidia six weeks after discontinuation and clinical signs recurred. Wellehan recommends 25 mg/kg nitazoxanide PO q 24 hrs x 5 days then 50 mg/kg PO q 24 hrs x 23 days. Confirmed severely emaciated cases should be euthanized because of the poor prognosis and possible source of infection to other geckos, unless the owner is extremely dedicated to lifelong treatment. All caging and cage furniture should be thrown out or disinfected with 5–10% ammonia outdoors for 30 minutes or steam cleaned. Bleach is ineffective.

Pneumonia can result from aspiration or too cool environment. Aspiration is common in anorexic geckos iatrogenically by well-intentioned owners. Bubbling from the nares and increased respiratory effort may be present. Increased cage temperatures to 82°–85°F and oral or systemic antibiotics are indicated for three weeks.

Egg retention is uncommon. Leopard geckos lay two eggs at a time, a single egg is cause for radiographs or ultrasonic examination. The soft shelled eggs can be difficult to discern radiographically. Calcium supplementation, providing a hide/nest box, and oxytocin, is recommended. Oxytocin (5–10 U/kg IM) is generally less successful in lizards than chelonians; celiotomy is indicated for egg retention greater than three weeks.

Gout (articular, visceral, and renal) is not commonly seen in leopard geckos. Gout is an end stage presentation of renal failure. Articular gout can be distinguished from pseudo gout and abscesses by biopsy. Allopurinol (25 mg/kg PO q 24 hrs, Ranbaxy Pharmaceuticals Inc., Jacksonville, FL) is indicated

if uric acid levels are elevated. Fluid therapy, debridement of urate tophi, and judicious use of meloxicam (Boehringer Ingelheim Vetmedica, Inc., St. Joseph, MO) are indicated, but keep in mind gout is not curable.

Xanthomas, single or multiple, in the brain, coelom, lungs, liver, or other tissues, were found in 11% of leopard gecko cases in a necropsy review. Xanthomas are nodular accumulations of cholesterol and lipid laden macrophages found in female geckos associated with excesses in serum cholesterol, triglycerides or lipoproteins related to dietary factors, folliculogenesis, follicular degeneration, or yolk coelomitis. Clinical signs of xanthomas in other gecko species (*Naultinus* spp., *Uroplatus* spp.) included central nervous system disorders (stargazing, torticollis, dorsal recumbency, and seizures), inappetence, weight loss, and death; ventricular xanthomas were associated with varying degrees of hydrocephalus, and coelomic xanthomas were found in most cases.

Neoplasias were found in 6% of cases in a necropsy review of leopard geckos, 77% were malignant, 23% were benign. Malignant neoplasias included a predominance of sarcomas (41% of all tumors, including soft tissue sarcomas, rhabdomyosarcomas, and fibrosarcomas).

CONCLUSION

These are the most common diseases the authors sees in leopard geckos, but keep in mind any disease is possible, such as hyperthyroidism, and many have yet to be described. Encourage owners to pursue good diagnostic workups and follow through with rechecks until the animal is healthy. The old saying what you don't look for, you won't find, definitely applies.

REFERENCES

This article was previously published and used with permission. See Boyer T, Garner M, Reavill D, Steffes Z. Common problems of leopard geckos (*Eublepharis macularius*). *Proc ARAV*. 2014:117-125, for references and full article.