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## NASAL DISCHARGE IN CHELONIANS

*Thomas H Boyer, DVM, Dipl ABVP, Reptile and Amphibian Practice*

*Pet Hospital of Penasquitos, 9888-F Carmel Mountain Road, SD, CA 92129 USA*

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### ABSTRACT

Nasal discharge is a common presenting clinical sign in tortoises and some turtles. Nasal discharge first must be distinguished from gastric reflux. Mycoplasmosis remains the most common cause of nasal discharge but the differential diagnoses also include herpes virus, ranavirus, adenovirus, intranuclear coccidiosis, chlamydiosis, reovirus, foreign bodies, and oronasal fistulas. Diagnosis and treatment of the main differentials will be discussed.

### Introduction

Nasal discharge is always abnormal in chelonians (turtles, tortoises and terrapins), even if chronic. Many clients are not aware of this, fail to seek treatment or even consider nasal discharge a problem. Clients have even speculated the coastal marine layer in San Diego causes nasal discharge; not true! There are many potential causes.

First distinguish nasal discharge from gastric reflux. Gastric reflux or regurgitation occurs with gastrointestinal obstruction or stasis and is easy to demonstrate on radiographs (DV, lateral and AP horizontal beams). Look for distended bowel loops in the lung fields on the horizontal lateral and AP views, and bowel distention, foreign bodies or masses, such as cystouroliths, on the DV view. Dyschezia and bloating may or may not be present but anorexia, constipation or obstipation, are typically present. Green to brown saliva or macerated partially digested greens may be in the oral cavity or discharging from the mouth or nares. GI impaction can result from gravel, sand, fibrous material, plastics, wire, yard trash, bladder stones, tumors, gastrointestinal infection, or hypothermia. Gastrointestinal obstruction is beyond the scope of this discussion, but be sure nasal discharge is just that, and not gastric reflux, before proceeding.

The most common cause of nasal discharge is upper respiratory tract disease, or mycoplasmosis. Several species of *Mycoplasma*, *Mycoplasma agassizii*, *Mycoplasma testudineum*, and perhaps several yet to be identified *Mycoplasma* species, are causative.<sup>3,5,6</sup> *Mycoplasma testudinus* isolated from the cloaca of a Greek tortoise, is not thought to cause disease.<sup>21</sup> Mycoplasmosis has been described in an ever widening arc of tortoise species (especially *Gopherus*, *Testudo* and *Geochelone* species) as well as box turtles, bog and spotted turtles.<sup>1,5,6,21</sup> All terrestrial turtles are likely susceptible. Mycoplasmosis is widespread in wild *Gopherus agassizii* and *Gopherus polyphemus*<sup>8,21</sup> but was not found in wild *Gopherus berlanderi*.<sup>20</sup> However 80% of 39 *G. berlanderi* were seropositive in a Texas rehabilitation facility.<sup>20</sup> In *G. polyphemus* disease incidence is higher in sexually mature adults, especially males, compared to juveniles. Contacts through courtship, mating and male agnostic behavior may be required for horizontal transmission.<sup>23</sup>

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To appreciate mycoplasmosis we must understand nasal sinus anatomy. *Mycoplasma* bacteria attach to the surface of epithelial cells in the nasal sinus and choana (internal nares) and cause loss of ciliated epithelium, mucosal hyperplasia, and infiltration of lymphocytes and histiocytes.<sup>6</sup> Normal olfactory mucosa becomes replaced with proliferating mucosal epithelial cells and proliferating basal cells, heterophils, and histiocytes.<sup>6</sup> The lesions slough large amounts of epithelial and inflammatory cells, which create the nasal discharge and over time form caseous material filling and occluding the nasal sinus.<sup>1,6,22</sup> Clinical impression is that most tortoises don't eat well because their sense of smell is affected.

Clinical signs include clear serous to tenacious mucoid bubbling to mucopurulent nasal discharge, sometimes also from the eyes, conjunctivitis and palpebral edema. Clinical signs may appear within 1-2 wk of exposure, it takes *G. polyphemus* 6-8 wk to develop an immune response.<sup>14</sup> *Mycoplasma* is spread directly or indirectly via nasal exudates and is very contagious as tortoises often greet one another nose to nose. Many tortoises are chronically infected. Infected tortoises are often inappetent, lethargic with clear nasal discharge, bubbling from the nares or have clogged nares and have lost weight or feel light. Chronic cases can have erosion of the nares and rhamphotheca; hatchlings can have enlargement and distortion of the snout (reminiscent of atrophic rhinitis in swine).

Several diagnostic options are available. Chelonian *Mycoplasma* culture is extremely difficult due to slow growth (4-6 wk for *Mycoplasma agassizii*), small size (colonies not visible without dissecting microscope) and fastidious culture requirements (lower temperature, special expensive culture media and with serum added).<sup>1,22</sup> In Jacobson, et al's, paper<sup>6</sup> less than a quarter of infected tortoises cultured positive even with a dedicated university *Mycoplasma* laboratory. *Mycoplasma* lacks a cell wall which makes life outside the host precarious at best. PCR diagnosis is also problematic because *Mycoplasma* numbers fall over time due to host response and good nasal flush samples are hard to obtain without anesthesia. Serology is the test of choice for *Mycoplasma* infections in other species and ELISA testing has been validated for desert and gopher tortoises to detect anti-*Mycoplasma* antibodies.<sup>19</sup> The disadvantage of ELISA is that it only detects antibodies which documents exposure but not necessarily infection. None-the-less this is an important screening test when importing chelonians to a closed collection. Whole blood or serum can be shipped on ice packs or dry ice via Fed Ex Priority Overnight Service sent to: *Mycoplasma* Testing Lab, University of Florida, Department of Pathobiology, 1600 SW Archer Road – BSB 350, Gainesville, FL, 32610 (contact lab beforehand, at 352-392-4700 x 3968, to request a sample submission form, samples must arrive on a weekday)<sup>14</sup> or through various commercial laboratories. Most symptomatic tortoises the author has tested are positive and clients often balk at the cost of testing in which case we proceed to treatment.

Three major classes of drugs are used to systemically treat *Mycoplasma* include fluoroquinolones, macrolides and tetracyclines.<sup>1</sup> Aminoglycosides are not indicated. Tetracyclines are not commonly used in chelonians (probably because of a lack of pharmacokinetic data) but are used in crocodylians with mycoplasmosis, Jarchow<sup>1</sup> recommended 6 mg/kg oxytetracycline IM q 24 hr x 10 – 14 days. Fluoroquinolones options include enrofloxacin (Baytril 100, 100 mg/ml, or Baytril 2.27%, 22.7 mg/ml, Bayer Corp, Shawnee Mission, KS, 5 mg/kg SC q 24 hr for 3-6 wk, vary injection site for Baytril 2.27%), or



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danofloxacin mesylate (A180, 180 mg/ml, Pfizer Animal Health, NY, NY, 6 mg/kg SC q 48 hr for 3-6 wk) or clarithromycin (Biaxin, 50 mg/ml, Abbott Labs, Abbott Park, IL, 15 mg/kg PO q 48-72 hr for 3-6 wk).<sup>24,25</sup> Palatability is horrible with clarithromycin and 2.27% enrofloxacin is irritating on injection so danofloxacin is currently the author's drug of choice. In the absence of exact data, Wellehan<sup>21</sup> recently recommended macrolides and tetracyclines as the best choices for treatment, for at least 45 – 60 days.

The nasal sinus and choanae should be flushed from the nares to choana in the sedated tortoise in dorsal recumbency with the head extended, mouth open and the glottis packed off by several cotton balls clamped to a hemostat to prevent aspiration. The author flushes 12 cc saline per nare, twice, the second flush including 0.6 mls of 2.27% enrofloxacin.<sup>1</sup> Thick mucus or solid cellular debris can be flushed out the choanae and removed from the mouth, opening up the nasal sinuses. Nasal sinus flush can be used for PCR testing. After the nasal sinus is flushed have the owner continue topical therapy daily with 0.3% Ofloxacin Ophthalmic Solution (Pack Pharmaceuticals, Buffalo Grove, IL) in the nares daily until 1 wk after cessation of nasal discharge. Drops are placed in the nares by angling the tortoise 45 – 90 degrees vertically while pushing dorsally on the gular area. After placing drops on each nare, immediately release pressure on the gular area and the drops will be drawn into the nares.

Severely underweight anorexic animals benefit from an esophagostomy tube. Most cases are still eating, or were recently eating, and in fair body weight, so an esophagostomy tube isn't indicated. Keep patients between 73° (low at night) to 86°F (high during day). Treatment is generally not curative, but most patients respond well to treatment, with improved appetite and weight gain. Untreated patients develop chronic disease with solid cellular debris eventually filling the nasal sinuses leading to weight loss and slow decline. In captivity tortoises can survive indefinitely, in the wild they may fail to make it through hibernation or die within a few years. Gopher tortoise populations on Sanibel Island, FL, declined by 25 to 50% over 10 yr and similar declines have been observed in Mojave desert tortoise populations.<sup>1,22</sup> With treatment clinical signs may resolve, in others, clinical signs persist. Relapses are common in winter, upon emergence from hibernation and after the rainy season in the Sonoran desert.<sup>1</sup> As in most animals, it not likely to eliminate *Mycoplasma* from the patient, none-the-less chelonians do far better with treatment than without. *Mycoplasma*-positive tortoises should be isolated from *Mycoplasma*-negative tortoises as the disease is extremely contagious. *Mycoplasma* survive poorly in the environment without a cell wall, cages can be rinsed and disinfected with 0.15% sodium hypochlorite. Remember nasal discharge is always abnormal and should be treated. Don't ignore it just because the owner says the tortoise has always had nasal discharge.

Several other maladies can infect tortoises, and cause nasal discharge including herpes virus, ranavirus, adenovirus, intranuclear coccidiosis, chlamydiosis, reovirus, foreign bodies, and oronasal fistulas. The difference here is that tortoises often die quickly after the owner noticing something amiss (except for the last two), which is atypical for mycoplasmosis.

Herpes virus is the next most common cause of nasal discharge. A wide variety of tortoises are affected, especially the common captive chelonians in the Gopherus, Testudo and Geochelone genera. Herpesvirus is characterized by stomatitis – rhinitis.<sup>15</sup> Stomatitis with focal to multifocal

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to coalescing, white to yellow, diphtheritic plaques, appear about 11 to 12 days after exposure.<sup>15</sup> Nasal discharge initially is serous but becomes mucopurulent later. Severe conjunctivitis and blepharodema may cause the eyes to swell shut with aqueous or white to yellow mucoid discharge. Hypersalivation, glossitis, dyspnea, dehydration and central nervous system signs, such as head tilt and circling, weight loss, cachexia, may be variably present. In the author's experience outbreaks often occur after introduction of Russian tortoises, *Testudo horsfieldi*, which may be carriers or symptomatic.

Two serotypes are known which can complicate PCR testing which can be specific for one, or both, depending on the test.<sup>15</sup> Fresh diphtheritic plaques on the tongue or caudal oral cavity are good for PCR testing and make this easy to distinguish from mycoplasmosis. ELISA serology is available to screen tortoises for herpes virus<sup>16</sup>, once exposed tortoises are likely carriers for life. Tortoises produce anti-herpes virus antibodies 4-7 wk post-infection. Infected Mediterranean tortoises had lymphocytosis, heterophilia, elevated AST and alpha globulin fractions, significantly higher than tortoises without herpesvirus.<sup>13</sup> Necropsy with an experienced reptile pathologist can confirm herpes. Impression smears of the tongue may show eosinophilic intranuclear inclusions with Giemsa or hematoxylin & eosin stains. Oral lesions with ranavirus infections can look quite similar. Keep in mind that bacterial or fungal stomatitis almost never happens in chelonians, unlike squamates.

Direct horizontal transmission is typical from virus shed in respiratory secretions, saliva or feces. Virus can persist for months in soil. Prompt aggressive treatment with acyclovir (80 mg/kg PO SID-TID x 21 days), antibiotics, fluid and nutritional support via esophagostomy tube, and broad-spectrum antibiotics are important.

Ranavirus is an iridovirus known for causing mass mortalities in fish and amphibians that may have crossed over into turtles and other reptiles<sup>9</sup> from frog virus 3.<sup>12</sup> Ranavirus has caused a rash of epizootics in chelonians since the late 1990's, especially in box turtles, aquatic turtles and tortoises.<sup>4,12</sup> It can cause death in a wide variety of chelonians from tortoises to soft shelled turtles. Clinical signs include lethargy, anorexia, dyspnea, nasal discharge, conjunctivitis, oral ulcerations, severe subcutaneous cervical edema, ulcerative stomatitis, and "red-neck disease".<sup>9,12</sup> Like herpes virus it seems to hit the respiratory and gastrointestinal tracts very hard, histologically, infected animals have hepatitis, enteritis, and pneumonia.<sup>12</sup> PCR testing is available on combined oral and cloacal swabs, as well as whole blood, as well as ELISA testing.<sup>10</sup> Transmission is poorly understood at this time but outbreaks are often associated with amphibians.

An adenovirus outbreak in over a hundred illegally imported Sulawesi tortoises, *Indotestudo forsteni*, killed the majority of them despite intensive veterinary care at multiple institutions.<sup>18</sup> Since then several other tortoise and box turtles species have been infected. Tortoises had nasal and ocular discharge, mucosal ulcers and palatine erosions. Pathologic findings in infected tortoises were multifocal hepatic necrosis, amphophilic to basophilic intranuclear inclusions and diffuse hepatic lipidosis, myeloid necrosis in bone marrow and severe necrotizing enterocolitis.<sup>12</sup> PCR testing is available for tissue, nasal flushes, cloacal swabs or serum. Virus is shed in feces and oronasal secretions and persists in the environment making fomite or keeper transfer



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Intranuclear coccidiosis can also result in high mortality in a wide variety of tortoises and box turtles and was first identified in radiated tortoises, *Geochelone radiata*, in 1990.<sup>7</sup> The causative coccidian and lifecycle have yet to be identified. Clinical signs included severe lethargy, rapid weight loss, weakness, gasping respiration, conjunctivitis, nasal discharge, oronasal fistulas and swollen erythematous vents with ulceration.<sup>17</sup> Thick choanal mucus is present on gross necropsy. The parasite is extremely contagious and seems to permeate all tissues in tortoises. Death follows onset of clinical signs within days unless treated with ponazuril or toltrazuril. Swabs of nasopharynx followed by cloaca can be tested for intranuclear coccidiosis via PCR at the University of FL.

A group of research desert tortoises were examined after unexplained morbidity and mortality. Clinical signs included nasal discharge, ocular discharge, conjunctivitis, loose feces, fecal staining around the vent and mucoid feces. Several tortoises were positive for *Mycoplasma* and *Chlamydophila*-like organisms.<sup>11</sup> In tortoises, a reovirus has only been isolated in one case from a spur-thighed tortoise, *Testudo graeca*. The tortoise was cachectic and had a necrosis of the epithelium of the tongue.<sup>12</sup>

A variety of foreign bodies can also cause nasal discharge, such as foxtails in the nasal sinus or choanae, and sticks in the roof of the mouth. One sulcata tortoise, *Geochelone sulcata*, even had a bottle cap in the roof of his mouth. One case of chronic unilateral nasal discharge in a Russian tortoise, *Testudo horsfieldi*, had an oronasal fistula discovered while flushing the nasal sinuses. Overheating, such as a hatchling tortoise flipped over in full sun, will cause nasal discharge for several hours that resolves without treatment if allowed to cool down. Vitamin A deficiency can cause blepharodema with nasal discharge as well, but tortoises rarely get hypovitaminosis A (greens being rich in  $\beta$ -carotene, a vitamin A precursor), unlike aquatic turtles and box turtles which are very susceptible.<sup>2</sup> Remember a good oral exam is always indicated with nasal discharge. Although *Mycoplasma* is the primary differential do not overlook other potential causes.

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