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Case Report

Acute Respiratory Distress Syndrome in an Uromastix (Uromastyx acanthinura nigriventris, 1820)

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Abstract

This article describes a case of acute respiratory distress syndrome (ARDS) in a 3-year-old *Uromastyx acanthinura gravinensis*. The lizard was presented to the veterinary hospital with an 8-day history of respiratory distress. After the initial physical examination, the patient was treated for the respiratory condition for three weeks and subsequently discharged. The bearded dragon died two weeks following release from the veterinary hospital, after an episode of acute dyspnea. Acute respiratory syndrome was diagnosed following histological examination of submitted tissue samples. The authors believe the condition was caused by possible environmental exposure to volatile organic compounds.

*Key words:* Reptiles; pneumonia; ARDS; respiratory pathology; noninfectious disease
A 3-year-old adult male *Uromastyx acanthinura nigriventris* (Bell’s dab lizard), which lived in a homemade terrarium, was presented to Centro Veterinario Il Mondo Degli Animali Esotici with an 8-day history of from anorexia and abnormal respiratory sounds (“gurgling”). The owner reported that the patient had apparently been “coughing” for several months and this condition had become more prominent over time. Upon presentation the patient had dull senses (its eyes were closed during the preliminary medical examination) and was in a poor body condition (its body condition score was 1/5), with a severe dehydration and abnormal respiratory sounds, “gurgling”. The animal expelled approximately 5 ml of odorless, slightly opaque liquid from its mouth which was submitted for aerobic culture. The lizard was hospitalized for diagnostic testing and treatment. Blood was collected from the ventral tail vein and submitted for a complete blood count and plasma chemistry panel. Abnormal results of the CBC were ………. and the plasma chemistry panel ………. The abnormalities identified through the CBC and plasma chemistry results indicated that the lizard was severely dehydrated and responding to an inflammatory disease process. A cloacal wash identified flagellates in the sample. The aerobic culture of the oral discharge was positive for the presence of *Salmonella* spp. and *Neisseria* spp. *Salmonella* spp. is considered as a potential pathogen, although the reptiles are often subclinical carriers of this organism. Several articles that describe the results of treating *Salmonella* spp. in reptiles state that eliminating this organism from the reptiles is very difficult. Moreover, treatment failure may encourage the development of drug-resistant organisms.¹² *Neisseria* spp is considered normal bacterial flora of the lizard upper respiratory tract.

Following an assessment of the diagnostic test results treatment of the uromastix was initiated with enrofloxacin 5 mg/kg subcutaneously, metronidazole 80 mg/kg, orally, ringer solution for reptiles (50% Lactated Ringer’s solution and 50% of 5% glucose solution) 5 mg/kg subcutaneously, every 2 days for 4 treatments, bicomplex 0.1 ml, intramuscular, and fluimicil nebulized, every 12 hours.

The abnormal respiratory signs abated after three weeks of the previously described treatment protocol. However, the authors elected to extend the uromastix’s hospitalization in order to continue monitoring the patient. Hematologic testing was not repeated due to the animal’s increased activity and avoid unnecessary stress to the patient. While hospitalized the animal regained a normal appetite and when discharged it was considered to have a normal body condition (its body condition score was 3/5). Two
weeks after being discharged from the hospital the uromastix suffered from acute respiratory distress and subsequently died.

**Discussion**

Respiratory disease (RD) is commonly diagnosed in reptile species. Pulmonary disease conditions in reptiles are often perceived as difficult to treat that inevitably lead to significant respiratory compromise, thus the prognosis is frequently elusive. Most of the RD cases in reptiles are related to improper husbandry techniques, including temperature extremes, inadequate humidity, and poor nutrition. Essentially, these environmental and nutritional stressors cause immunosuppression with the animal being unable to adequately respond to pathogen exposure. Various noninfectious and infectious agents are also considered the primary causes of RD in reptiles, including viral, bacterial, fungal and parasitic agents. Nevertheless, most reptile RD cases have a multifactorial etiology. This case report describes an *Uromastyx acanthinura nigriventris* diagnosed with respiratory disease.

During the necropsy examination of the uromastix, the lungs had a diffuse dark pink color, were firm, edematous, and heavy (Fig. 1). The histopathologic findings from submitted tissue samples consistently showed a marked expansion of the faveolar septa (up to 5 times bigger than their normal dimension), which was due to edema, fibrin, hyperemia, and a moderate number of macrophages, melanomacrophages, lymphocytes, and heterophils (Fig. 2). The multifocal faveolar lumina were inconsistently occluded by many degenerated heterophils, occasional macrophages, and sloughed epithelial cells admixed with few cellular and karyorhectic debris. The authors observed a diffuse and severe type II hyperplasia of the pulmonary epithelia (Fig. 3). No intraepithelial, intracytoplasmic, or intranuclear inclusion bodies were observed in the tissue samples, as well as the lack of intralesional bacteria. Hepatocytes within the liver parenchyma contained a small number of multiple cytoplasmic clear vacuoles (degenerative lipidosis) without any displacement of the nuclei. The morphologic diagnosis of the disease condition affecting the uromastyx was a bronchointerstitial, subacute, diffuse, and severe pneumonia with multifocal necrosis and epithelial hyperplasia and a moderate multifocal hepatic lipidosis. All bacterial cultures that were submitted from various organs, including the lung, were negative for growth. No other histopathologic abnormalities were identified in other organ tissue. This pathological diagnosis was compatible with a diffuse and severe lung injury that both human and veterinary medicine links to an acute respiratory distress
syndrome (ARDS) or acute lung injury (ALI), which often results in acute respiratory failure.

Acute respiratory distress syndrome refers to an acute and life-threatening respiratory failure with the mortality rate in human patients ranging from 50% to 70%.\textsuperscript{8,9} Acute respiratory distress syndrome/ALI is a secondary inflammatory response to lung pathology, of which there have been several known primary and secondary contributing factors to these disease conditions that have been identified. Primary factors are directly related to lung injury (e.g., aspiration, contusion, infectious, inhalation of toxic gases).\textsuperscript{8,10} The secondary non-pulmonary factors cause a generalized inflammation that induces ARDS/ALI, including sepsis, shock, parvoviral enteritis and pancreatitis in dogs, as well as paraquat ingestion in dogs and cats.\textsuperscript{6,7} No specific risk factors for ARDS/ALI have been identified in reptile species including the \textit{Uromastyx} described in this report. The criteria for the diagnosis of ALI or ARDS have been adapted from human medicine (as is commonly implemented for dogs). Diagnosis of ARDS/ALI is determined through the patient’s exposure to known risk factors, acute onset of respiratory signs, bilateral pulmonary infiltrates, \(\text{PaO}_2:\text{FiO}_2<300\) mmHg (ALI) or \(<200\) mmHg (ARDS).\textsuperscript{11} Normally, the macroscopic examination and the histopathological findings through pathologic examination of the body following death provide the definitive diagnosis of ARDS/ALI. The pathogenesis of ARDS/ALI includes three partially overlapping morphologic phases: exudative, proliferative, and fibrotic. In humans, the first two phases last approximately 1 week following the onset of the clinical signs and are characterized by alveolar epithelial damage, pulmonary edema, formation of hyaline membranes, proliferation of type II pneumocytes, and the initial proliferation of fibroblast. The fibrotic phase is the final morphologic stage of lung injury in ALI/ARDS patients, when the deposition of collagen increases considerably within the lung parenchyma.\textsuperscript{11} As for clinical veterinary patients, little information is reported about this phase, which may be due to the high mortality rate during the initial phases.

In the uromastyx patient described in this report, the macroscopic examination, histological diagnosis, and bacterial culture results excluded the presence of any infectious agents whereas an inhalation of toxic substances was suspected. The owner was requested to provide information about the source of food administered to the patient, the mode and materials used to build the terrarium, and the products used to disinfect the terrarium and room. The owner reported that the initial respiratory signs exhibited by the
lizard started after he moved the animal into a new homemade terrarium that had recently been painted.

All the information provided by the uromastyx’s owner led to a suspected pulmonary exposure of volatile organic compounds (VOCs) contained in the paint. Volatile organic compounds are organic chemicals that have a high vapor pressure in normal and room temperature conditions and paint is a main source of VOCs.\(^{12}\) Volatile organic compounds of paint, emitted as gases, include a variety of chemicals (e.g., polycyclic aromatic hydrocarbons (PAHs), ethyl acetate, formaldehyde, methylene chloride, aliphatic compounds, glycol). These emissions of various VOCs are influenced by numerous environmental factors, including humidity, air changes, and temperature.\(^{13}\) The latter has a major impact on VOC emissions deriving from certain building materials and products.\(^{12}\)

Therefore, since proper care and maintenance of this particular species of lizard requires maintaining a high temperature in the terrarium (the daily temperature gradient is between 35-40 °C), the heat may have elicited a massive emission of the toxic substance. This possible exposure to VOCs may have caused the histological lesions identified in the uromastyx. Moreover, the patient had been exposed to the VOCs for a long period of time.

A case of respiratory failure due to a prolonged inhalation of PAHs has been reported in human medicine.\(^{14}\) The damage to the human respiratory tract as a result of formaldehyde and PAHs exposure are well known.\(^{15,16}\) The inhalation of toxic gases (e.g., smoke) is also considered as an important risk factor for ARDS/ALI in animals.\(^{8,9,10}\)

Very few case reports regarding respiratory disease in *Uromastyx acanthinura nigventris* or *Umormastix*\(^{17,18}\) have been published. A reovirus infection has been described in a spiny-tailed lizard (*Uromastyx hardwickii*) that was treated in the United Kingdom.\(^{19}\) The spiny-tailed lizard died during a disease outbreak from which macroscopic pathology was identified in the lungs of dead animals.\(^{19}\) In the *Uromastyx hardwickii* report, the histological lesions in lungs were not described and the virus was isolated from liver, spleen, and small intestine, but not lung tissue. The lung lesions observed in the case described in this report were not consistent with a viral infection.

Reptile respiratory tract disease is a common clinical presentation with both noninfectious and infectious causes having been identified as underlying causes. ARDS should be also considered in a differential diagnosis in reptiles with severe respiratory signs. Acute respiratory distress syndrome occurs when an immune response, which is triggered by a direct or indirect factor, results in pathology to lung tissue. Numerous underlying causes of ARDS/ALI are known. With this uromastyx case, the
inhalation of toxic chemical compounds was the suspected as the origin of the disease condition that ultimately resulted in the death of the animal.

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References


**Figure Legends**

**Figure 1.** Diffuse dark pink color in the edematous lung of the uromastyx.

**Figure 2.** Histological appearance of the pulmonary lesion. Severe expansion of faveolar septa due to edema, fibrin, hyperemia, and a number of macrophages, melanomacrophages, lymphocytes, and heterophils (×10, H&E).

**Figure 3.** Histological appearance of the pulmonary lesion. Severe hyperplasia of type II epithelia cells is evident (×10, H&E).