GOUT AND UREMIA SYNDROME ON CAPTIVE KOMODO (*VARANUS KOMODOENSIS*): CASE REPORT

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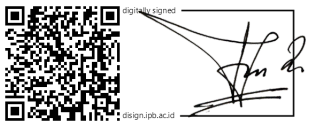

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Gout and Uremia Syndrome on Captive Komodo (*Varanus komodoensis*): Case report

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ABSTRACT

Visceral gout is an important consequence of chronic renal disease in reptiles but has rarely been documented in the endangered Komodo dragon (*Varanus komodoensis*). This report describes the gross and histopathological findings of generalized visceral gout in a 10-year-old captive female Komodo dragon that died following progressive anorexia and weakness. Complete necropsy revealed extensive white chalky deposits on the kidneys, pericardium, lungs, gastrointestinal tract, and ovaries. Representative tissue samples from the heart, lungs, stomach, intestines, liver, kidneys, spleen, and ovaries were examined histologically using hematoxylin and eosin, Periodic acid-Schiff, and methenamine silver stains. Microscopic examination demonstrated severe chronic granulomatous urate nephropathy characterized by diffuse interstitial fibrosis, glomerular atrophy, tubular degeneration and necrosis, and extensive intratubular urate deposition. Additional lesions included granulomatous urate pericarditis with myocardial degeneration and necrosis, pulmonary congestion, hemorrhage, emphysema, metastatic mineralization, ulcerative hemorrhagic gastritis with intralesional candidiasis, granulomatous splenitis, and hemorrhagic oophoritis. The distribution and severity of lesions indicate that chronic renal failure resulted in persistent hyperuricemia, systemic urate crystal deposition, and multisystemic uremic syndrome. This case expands the limited pathological literature on naturally occurring visceral gout in *V. komodoensis* and emphasizes the importance of early recognition of renal disease and husbandry-related risk factors in the health management of captive Komodo dragons.

Keywords: *Varanus komodoensis*; visceral gout; chronic kidney disease; urate nephropathy; uremic syndrome; pathology.

INTRODUCTION

The Komodo dragon (*Varanus komodoensis*) is the largest living lizard and an iconic apex predator endemic to the Lesser Sunda Islands of Indonesia. Because of its restricted distribution, habitat fragmentation, climate-related environmental changes, and increasing human activities, the species is currently classified as Endangered by the International Union for Conservation of Nature (IUCN) and is protected under Appendix I of the Convention on International Trade in Endangered Species (CITES). The growing role of zoological institutions in ex situ conservation has increased the need for comprehensive knowledge of diseases affecting captive Komodo dragons, particularly disorders that compromise long-term survival and reproductive success (Koch et al., 2013; Jessop et al., 2018; IUCN, 2021).

Renal disease is among the most important health problems reported in captive reptiles and frequently culminates in visceral gout. Unlike mammals, reptiles are uricotelic and excrete nitrogenous waste primarily as uric acid. Consequently, impaired renal function reduces urate excretion, resulting in persistent hyperuricemia and subsequent precipitation of monosodium urate crystals within tissues. Depending on the location of crystal deposition, gout is classified as either articular or visceral. Visceral gout is characterized by multifocal to diffuse urate deposits affecting serosal surfaces and visceral organs, particularly the kidneys, heart, liver, lungs, spleen, and reproductive organs, where progressive crystal accumulation induces chronic inflammation and organ dysfunction (Divers and Stahl, 2019; Johnson and Watson, 2020).

The development of visceral gout in reptiles is complex and multifactorial. Chronic dehydration, prolonged anorexia, excessive dietary protein intake, nephrotoxic agents, infectious diseases, environmental stress, and inadequate husbandry have all been recognized as important predisposing factors for renal injury. Progressive nephron loss decreases glomerular filtration and tubular secretion of uric acid, leading to sustained hyperuricemia. Once the solubility threshold of urate is exceeded, monosodium urate crystals precipitate within renal tubules and on visceral serosal surfaces. These crystals act as potent pro-inflammatory stimuli, activating macrophages and multinucleated foreign-body giant cells, ultimately producing chronic granulomatous inflammation, fibrosis, and irreversible tissue damage. As renal function deteriorates, impaired urate clearance perpetuates further crystal deposition, creating a self-amplifying cycle that culminates in multisystemic visceral gout and uremic syndrome (Johnson and Watson, 2020; Halán *et al.*, 2022; Soare *et al.*, 2022; Divers, 2025).

Although visceral gout has been described in a variety of reptilian species, including chelonians, snakes, and other varanid lizards, comprehensive pathological descriptions in the Komodo dragon remain exceptionally limited. Given the conservation importance of this species and the scarcity of detailed clinicopathological reports, documentation of naturally occurring disease is essential for improving diagnostic approaches, clinical management, and preventive husbandry in captive populations.

Here, we describe the gross and histopathological features of severe multisystemic visceral gout in a captive female Komodo dragon (*Varanus komodoensis*). Particular emphasis is placed on the relationship between chronic renal disease, systemic urate deposition, and the development of multisystemic lesions, thereby providing additional pathological information for this endangered species.

CASE HISTORY

A mature female Komodo dragon (*Varanus komodoensis*) maintained at ex-situ conservation, Indonesia, died after an undetermined period of illness. A complete necropsy was performed at the Division of Pathology, School of Veterinary Medicine and Biomedical Sciences, IPB University, Bogor, Indonesia. Representative samples of the heart, lungs, liver, stomach, intestine, pancreas, kidneys, spleen, and ovaries were collected for histopathological examination.

Tissues were fixed in 10% neutral buffered formalin, routinely processed, embedded in paraffin wax, sectioned at approximately 4-5 μm, and stained with hematoxylin and eosin (HE). Methenamine silver staining was performed to confirm urate crystal deposition, whereas Periodic acid-Schiff (PAS) staining was used to identify fungal organisms within affected tissues. Histopathological evaluation was performed using light microscopy.

GROSS FINDINGS

A complete necropsy was performed on an adult female Komodo dragon (*Varanus komodoensis*) from ex-situ conservation. According to the clinical history, the animal exhibited progressive weakness, anorexia, and subsequently died. Gross examination revealed severe multisystemic lesions affecting the cardiovascular, respiratory, digestive, urinary, lymphoid, and reproductive systems (Table 1). The most remarkable finding was widespread deposition of white, chalky material on multiple visceral organs, consistent with visceral urate tophi.

Table 1 Summary of Gross Pathological Findings

Organ System	Organ	Gross findings
Cardiovascular	Heart	Diffuse urate tophi covering epicardium, thickened pericardium, serous atrophy of epicardial fat, pale myocardium, chicken fat clots and blood clots within both ventricles, and multifocal mineralization of aorta
Respiratory	Trachea	Intraluminal hemorrhage
	Lungs	Diffuse congestion, multifocal pleural urate deposits, multifocal mineralization, emphysema
Lymphoid	Spleen	Moderate splenomegaly
Digestive	Stomach	Severe ulcerative hemorrhagic gastritis
	Intestine	Serosal urate deposition with catarrhal enteritis
	Liver	Hepatomegaly, diffuse pallor, multifocal petechial and ecchymotic hemorrhages
Urinary	Kidneys	Severe diffuse visceral gout replacing cortex and medulla
Reproductive	Ovaries	Multifocal urate deposition with hemorrhagic oophoritis

The pericardial sac was markedly thickened and contained diffusely distributed white, chalky deposits covering nearly the entire epicardial surface (Figure 1A, 1B). Epicardial fat was translucent, gelatinous, and markedly reduced, consistent with serous atrophy of fat. The epicardial surface was roughened and irregular due to extensive urate deposition. The myocardium and endocardium were diffusely pale. Large yellow-white postmortem fibrin-rich clots (chicken fat clots) together with dark red blood clots were present within both ventricles (Figure 2A), and marked thickening of the aortic wall with multifocal atherosclerotic plaques (Figure 2B).

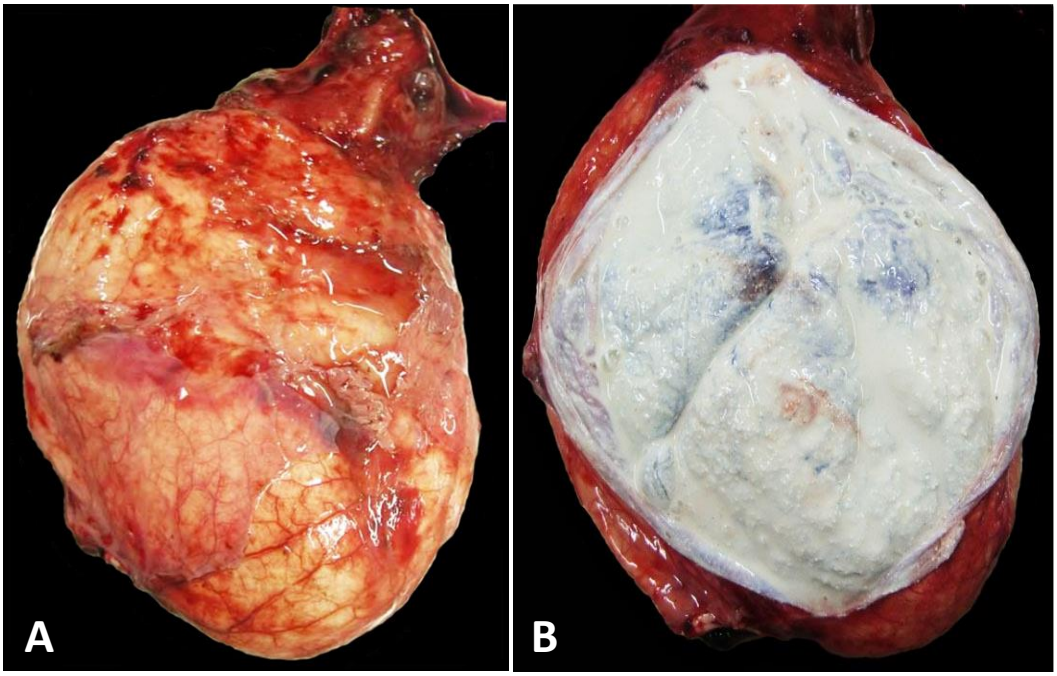

Figure 1 Gross cardiac lesions in a female Komodo dragon **(***Varanus komodoensis***)** with visceral gout. (A) Thickened pericardium with serous atrophy of the epicardial fat. (B) Diffuse chalky white urate deposits on the epicardial surface consistent with visceral gout

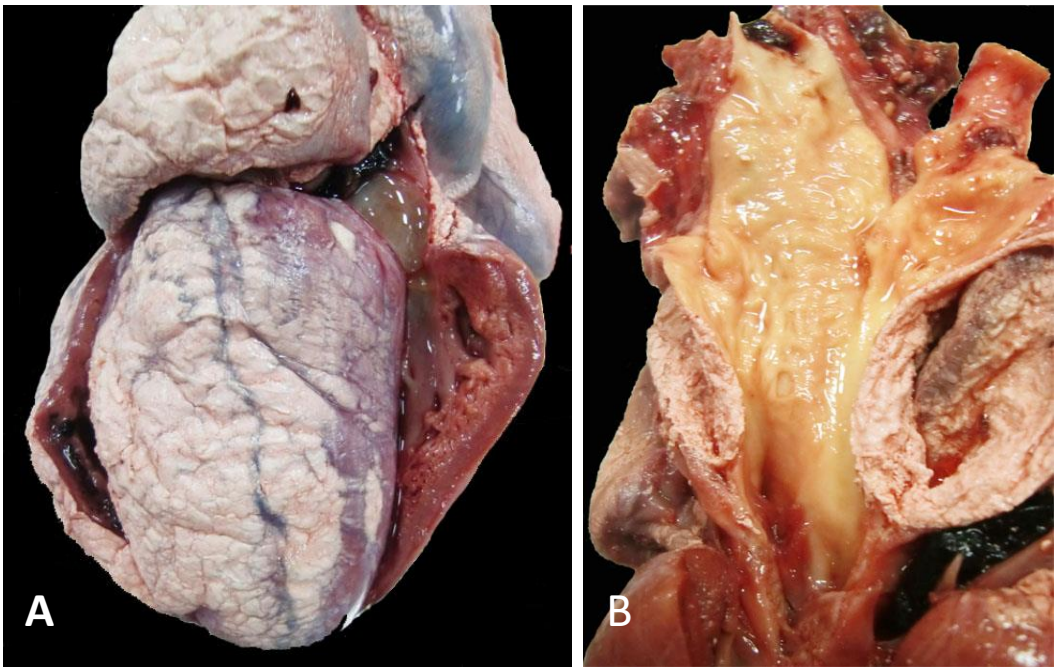

Figure 2 (A). Visceral gout with myocardial pallor consistent with cardiomyopathy, accompanied by chicken fat clot and blood clot in both ventricles. (B) Marked thickening of the aortic wall with multifocal vascular mineralization plaques

Both kidneys were extensively enlarged and diffusely covered by thick white chalky material (Figure 3). On cut surface, abundant white deposits extended throughout both cortex and medulla, replacing a substantial proportion of the renal parenchyma.

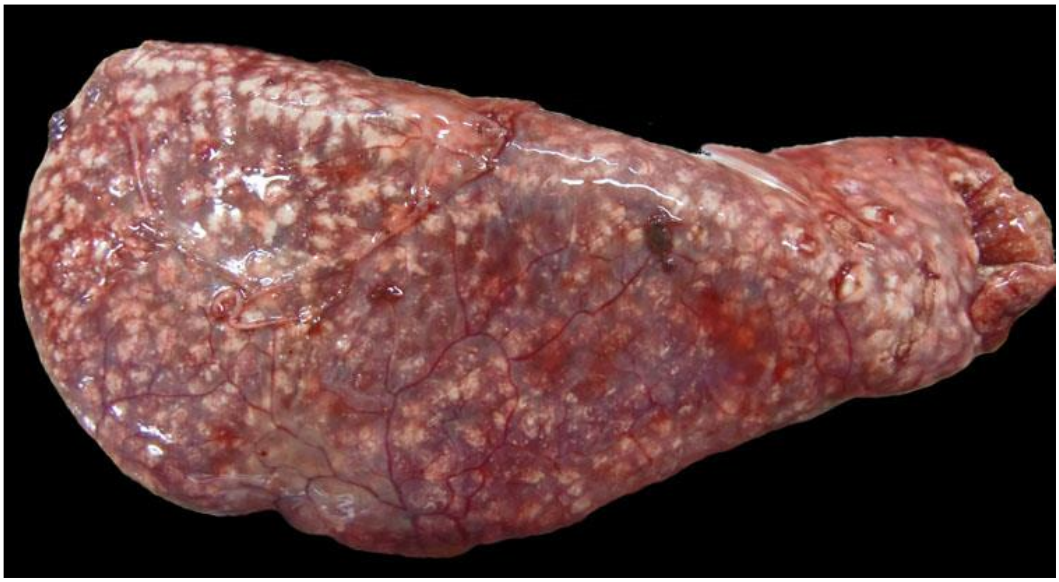

Figure 3 Gross renal lesions consistent with visceral gout in a female Komodo dragon **(***Varanus komodoensis***).** Extensive chalky white urate deposits on the renal surface throughout the renal parenchyma, extending from the cortex to the medulla.

Both lungs were diffusely mottled dark red and contained multifocal white chalky deposits distributed over the pleural surface. Numerous pinpoint, firm, white nodules consistent with multifocal mineralization were scattered throughout the pleura. Pulmonary parenchyma was congested and edematous. Blood was present within the tracheal lumen. Lung flotation testing demonstrated that all pulmonary lobes floated, indicating retained aeration.

The liver was diffusely enlarged with rounded margins. Hepatic parenchyma appeared pale, friable, and contained multifocal petechial to ecchymotic hemorrhages distributed throughout the capsular surface.

Multiple ovarian follicles contained extensive intrafollicular hemorrhage. Multifocal white chalky deposits were observed on several ovarian follicles, consistent with visceral urate deposition.

The stomach was devoid of ingesta and contained abundant serohemorrhagic exudate (Figure 4 above). Multifocal to coalescing ulcers measuring approximately 0.5-1.0 cm in diameter were distributed throughout the gastric mucosa. Ulcer bases were variably dark red or pale and exhibited irregular margins, consistent with severe ulcerative hemorrhagic gastritis. The intestinal serosa exhibited multifocal white urate deposits (Figure 4 below). Intestinal mucosa was covered by abundant catarrhal exudate.

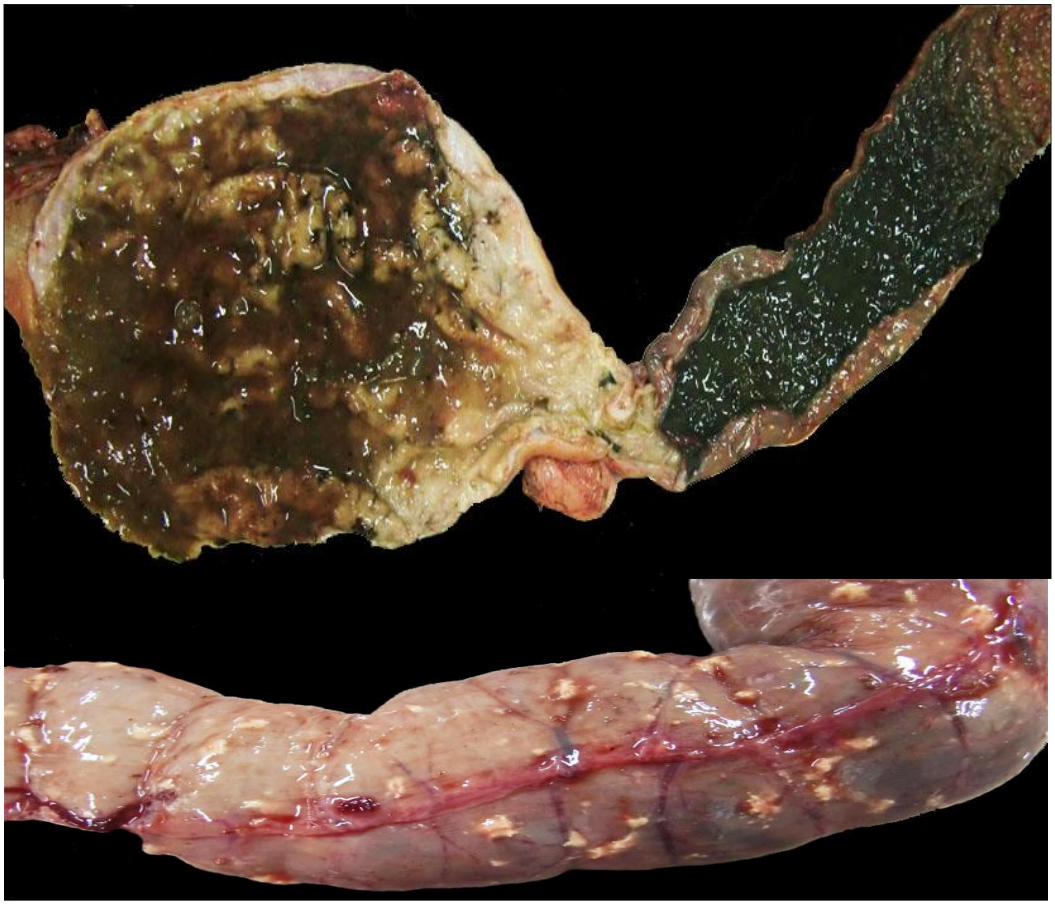

Figure 4 Gastrointestinal tract. Upper panel**:** Severe ulcerative hemorrhagic gastritis with abundant serohemorrhagic exudate. Lower panel**:** Multifocal chalky white urate tophi on the intestinal serosa (visceral gout).

The gross lesions were dominated by extensive multisystemic urate deposition involving the kidneys, heart, lungs, gastrointestinal tract, and ovaries. The distribution of lesions was consistent with severe chronic visceral gout accompanied by advanced renal disease. Widespread serosal urate tophi, pulmonary mineralization, ulcerative gastritis, and marked renal involvement suggested long-standing hyperuricemia with multisystemic organ dysfunction. Similar gross pathological findings have been reported in reptiles with chronic renal insufficiency and advanced visceral gout, particularly in captive lizards and chelonians (Divers & Stahl, 2019; Johnson & Watson, 2020; Halán et al., 2022).

HISTOPATHOLOGICAL FINDINGS

Representative histopathological findings are summarized in Table 2. The predominant microscopic lesions involved the cardiovascular, urinary, respiratory, gastrointestinal, lymphoid, and reproductive systems, with the kidneys exhibiting the most severe pathological alterations.

Table 2 Summary of Histopathological Findings

Organ	Histopathological findings
Heart	Severe granulomatous urate pericarditis (visceral gout), myocardial hyaline degeneration and necrosis
Kidney	Chronic granulomatous nephritis, diffuse interstitial fibrosis, glomerular atrophy, tubular degeneration and necrosis, intratubular urate deposition, metastatic mineralization
Lung	Severe congestion, hemorrhage, emphysema, metastatic mineralization
Stomach	Ulcerative hemorrhagic gastritis with intralesional fungal organisms consistent with candidiasis
Liver	Diffuse hepatic congestion
Spleen	Granulomatous splenitis
Ovary	Hemorrhagic oophoritis with metastatic mineralization

Microscopically, the epicardium was markedly expanded by extensive deposits of amorphous eosinophilic material corresponding to dissolved urate crystals, accompanied by severe granulomatous inflammation (Figure 4). The inflammatory infiltrate consisted predominantly of macrophages, lymphocytes, multinucleated foreign-body giant cells, and scattered heterophils surrounding the urate tophi. The underlying subepicardial connective tissue was edematous. The myocardium exhibited diffuse hyaline degeneration characterized by homogeneous hypereosinophilic sarcoplasm with loss of normal cross striations. Multifocal myocardial necrosis was characterized by pyknotic, karyorrhectic, and karyolytic nuclei with fragmentation of cardiac myofibers (Figure 5). Mild vascular congestion was also present.

These lesions are consistent with chronic granulomatous urate pericarditis secondary to visceral gout. In reptiles, persistent hyperuricemia results in precipitation of monosodium urate crystals on serosal surfaces. The crystals activate macrophages through inflammasome-mediated inflammatory pathways, inducing chronic granulomatous inflammation with multinucleated giant cell formation and progressive fibrosis. Chronic pericardial inflammation may compromise myocardial perfusion, contributing to ischemic myocardial degeneration and necrosis (Divers & Stahl, 2019; Johnson & Watson, 2020).

The kidneys displayed severe chronic diffuse nephropathy with extensive destruction of normal renal architecture. Cortical glomeruli were markedly reduced in number and frequently replaced by dense interstitial fibrosis (Figure 6,7, 8). Remaining glomeruli were shrunken and collapsed, with marked widening of Bowman's space. Multifocal glomerular mineralization was observed.

The renal interstitium was markedly expanded by mature fibrous connective tissue containing abundant macrophages, lymphocytes, heterophils, eosinophils, and numerous multinucleated foreign-body giant cells, consistent with chronic granulomatous nephritis. Renal tubules exhibited diffuse hydropic degeneration, epithelial attenuation, tubular dilation, coagulative necrosis, and multifocal epithelial detachment from the basement membrane. Tubular lumina contained

abundant eosinophilic proteinaceous casts, hemorrhage, cellular debris, dissolved urate crystal clefts, and multifocal mineralization.

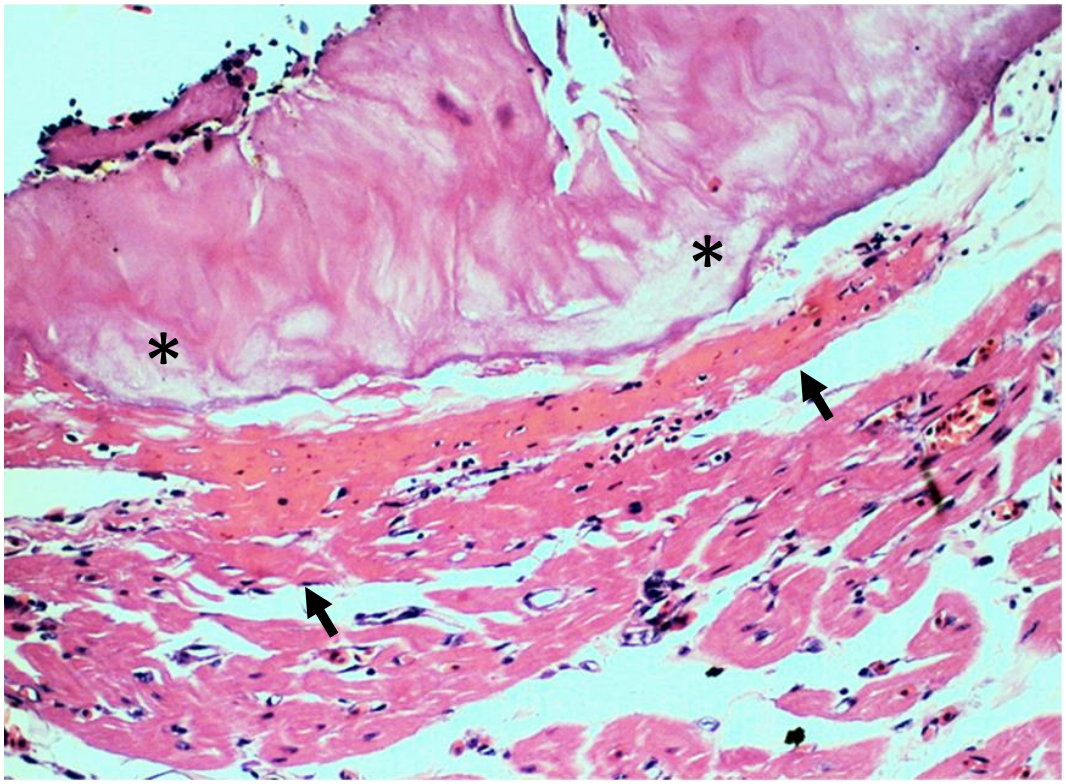

Figure 5 Heart. Severe granulomatous epicarditis associated with visceral gout in a female Komodo dragon **(***Varanus komodoensis***).** The epicardium is markedly expanded by urate tophi (asterisk), with subjacent myocardial hyaline degeneration (arrows). H&E, 10×

The renal lesions strongly support chronic renal failure as the primary pathological process. In reptiles, prolonged renal dysfunction markedly reduces uric acid excretion, producing persistent hyperuricemia. Excess circulating urate precipitates within renal tubules, causing mechanical obstruction, tubular epithelial injury, activation of macrophages, and chronic granulomatous inflammation. Progressive nephron loss further decreases glomerular filtration, establishing a vicious cycle of worsening hyperuricemia and continued urate deposition. The extensive interstitial fibrosis observed in this case indicates long-standing irreversible renal injury preceding the development of generalized visceral gout (Johnson & Watson, 2020; Halán et al., 2022; Divers, 2025).

Methenamine silver staining demonstrated numerous negative silhouettes of urate crystals within tubular lumina, while tubular basement membranes stained intensely black, serving as an internal positive control (Figure 8).

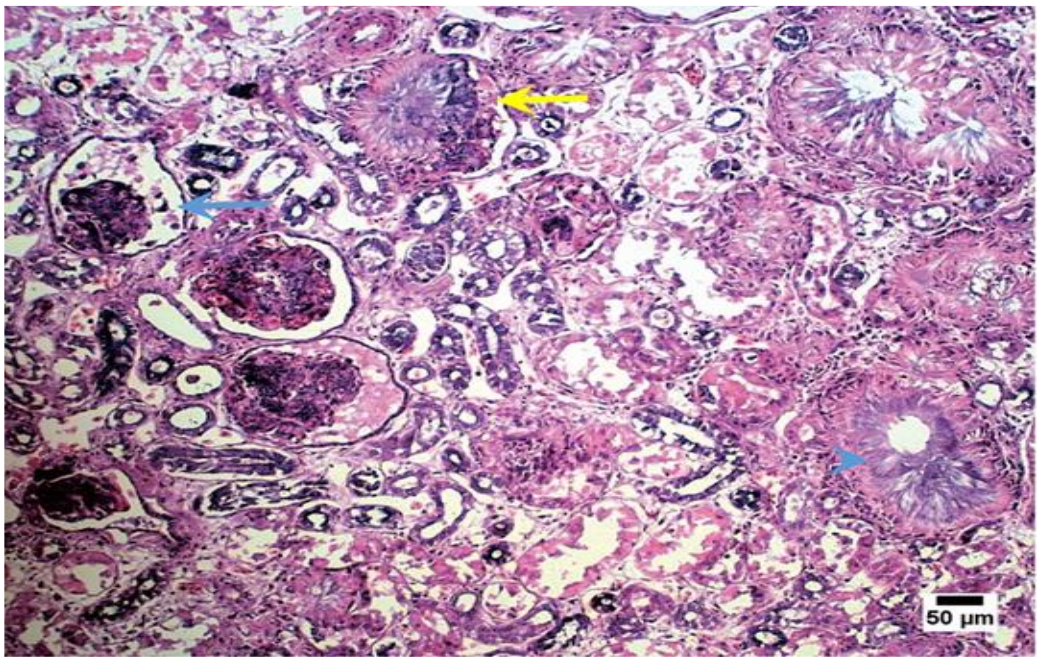

Figure 6 Kidney. Histopathologic lesions consistent with visceral gout in a female Komodo dragon (*Varanus komodoensis*)**.** Urate tophi occupy the tubular lumina (arrowheads), accompanied by glomerular atrophy (blue arrows) and intratubular urate deposits associated with metastatic mineralization (yellow arrows). H&E. Bar = 100 µm.

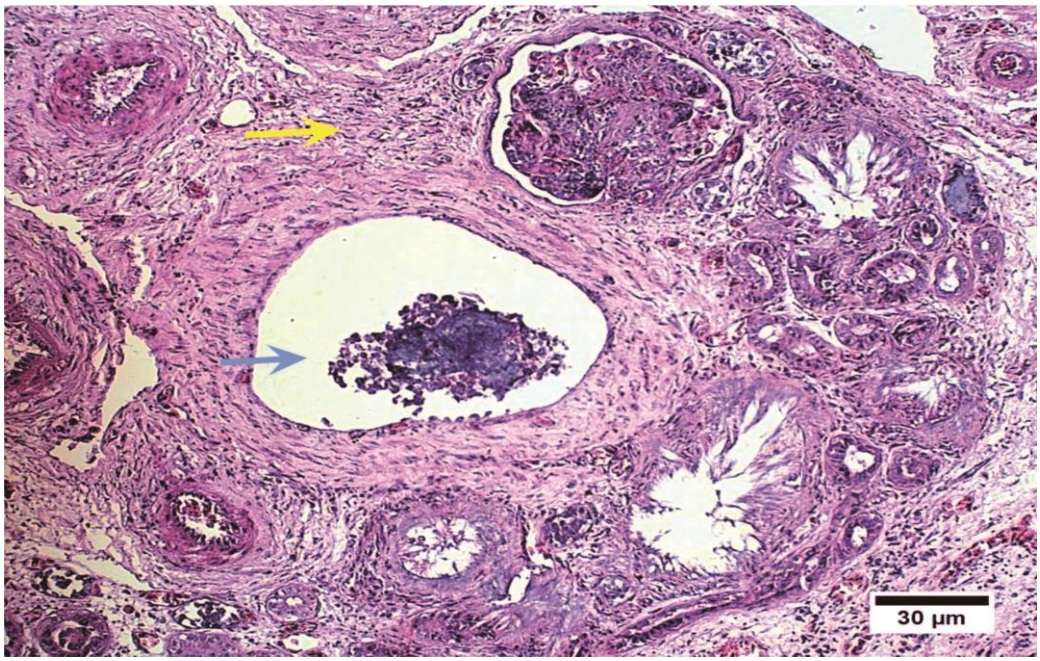

Figure 7 Kidney. Chronic renal lesions associated with visceral gout in a female Komodo dragon **(***Varanus komodoensis***).** Arterial and glomerular metastatic mineralization (blue arrows), severe interstitial fibrosis (yellow arrows), and abundant intratubular urate tophi (asterisk), consistent with advanced chronic kidney disease. H&E, 40× objective.

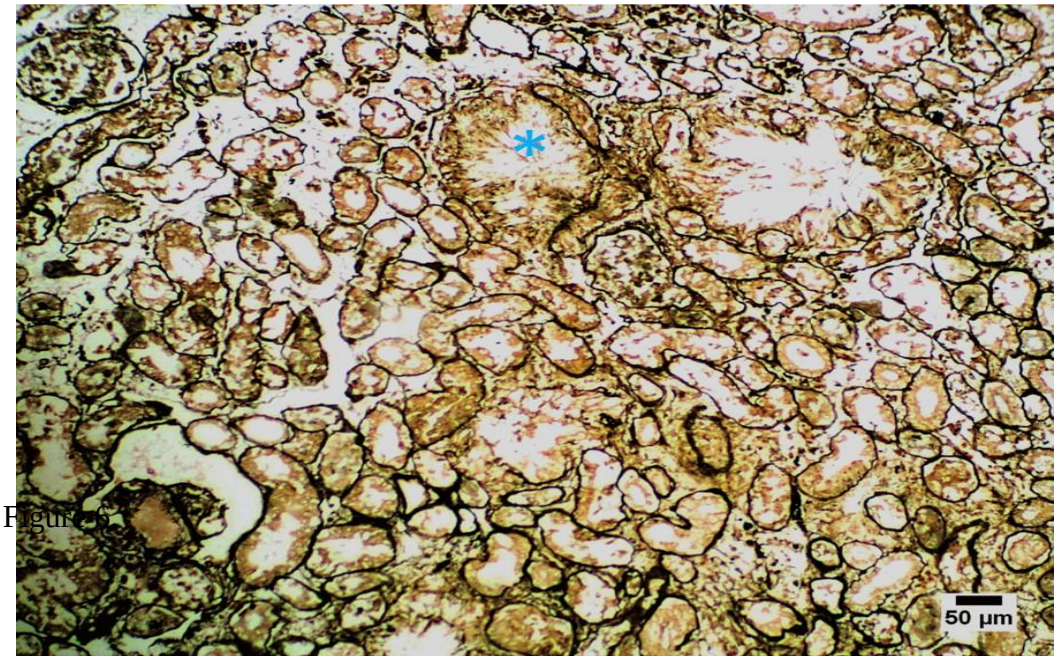

Figure 8 Kidney. Histochemical demonstration of visceral gout. Dissolved urate tophi are represented by unstained intratubular spaces (asterisk), while the tubular basement membranes stain dark brown to black with methenamine silver. Methenamine silver, 10× objective

Pulmonary lesions consisted of severe vascular congestion, multifocal hemorrhage, emphysema, and multifocal metastatic mineralization (Figure 8). Hemorrhage extended into bronchiolar lumina, corresponding to the grossly observed blood within the trachea. Multifocal basophilic mineral deposits expanded alveolar septa and occasionally occupied bronchiolar lumina.

No urate tophi were identified within pulmonary parenchyma, suggesting that crystal deposition was confined primarily to the pleural surface observed grossly. Pulmonary mineralization is compatible with metastatic calcification associated with chronic uremic syndrome. Renal failure disrupts calcium-phosphorus homeostasis, promoting calcium salt deposition within highly vascular tissues such as the lungs, kidneys, and blood vessels. Similar pulmonary lesions have been described in reptiles with advanced chronic kidney disease and visceral gout (Johnson & Watson, 2020; Divers & Stahl, 2019).

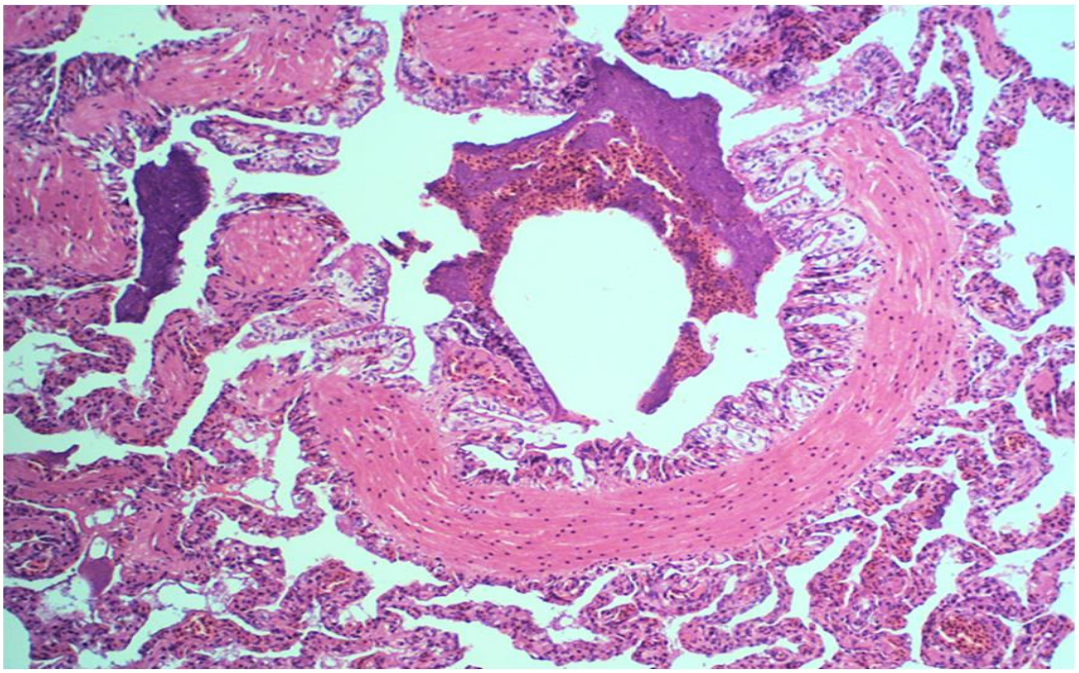

Figure 8 Lung. Pulmonary emphysema (asterisk), multifocal metastatic mineralization (yellow arrows), and bronchiolar congestion and hemorrhage (blue arrows) in a female Komodo dragon (*Varanus komodoensis*) with visceral gout. H&E, 10× objective.

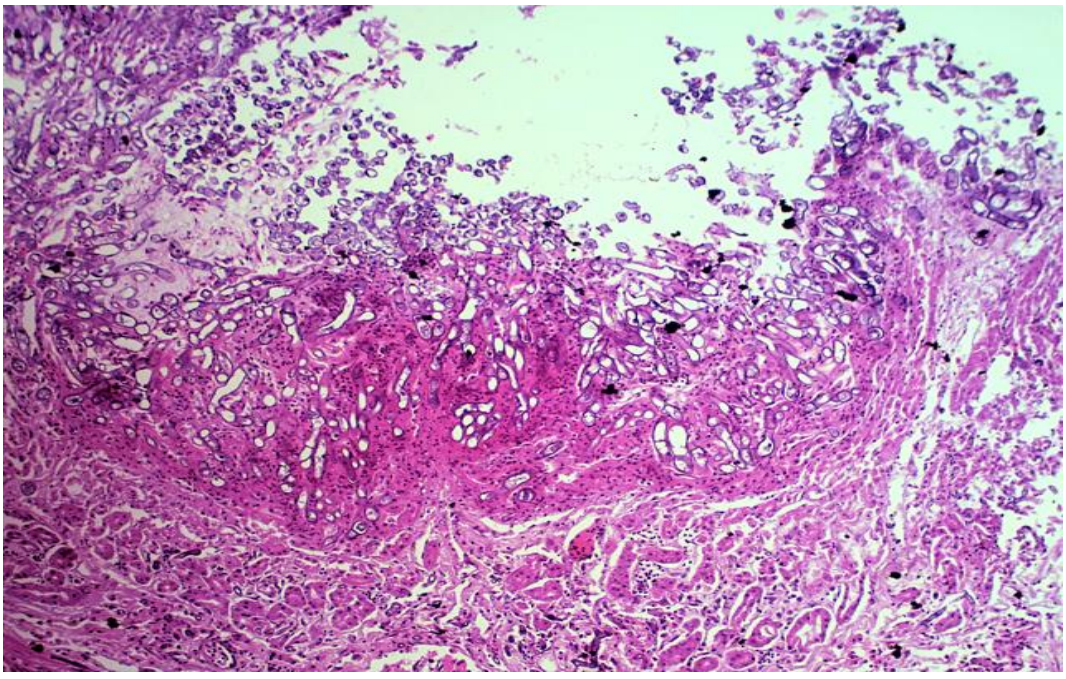

Figure 9 Stomach. Ulcerative gastritis with abundant fungal hyphae infiltrating the gastric mucosa, consistent with *Candida* spp. infection. PAS, 20× objective.

PATHOGENESIS OF VISCERAL GOUT IN REPTILES

Visceral gout represents the end stage of prolonged hyperuricemia and is one of the most important pathological manifestations of chronic kidney disease in reptiles. Unlike mammals, reptiles are uricotelic and excrete nitrogenous waste primarily as uric acid rather than urea. This physiological adaptation is highly efficient for water conservation but also renders reptiles particularly susceptible to urate accumulation whenever renal excretion is compromised. In captive reptiles, chronic dehydration, prolonged anorexia, excessive dietary protein, nephrotoxic agents, infectious diseases, inadequate environmental conditions, and chronic renal ischemia are recognized as the principal factors contributing to renal dysfunction and subsequent visceral gout (Divers and Stahl, 2019; Johnson and Watson, 2020; Divers, 2025). These risk factors are especially relevant in zoological collections, where husbandry, nutrition, hydration, and environmental management substantially influence long-term renal health. Because the Komodo dragon (*Varanus komodoensis*) is an endangered species maintained in ex situ conservation programs, early recognition of renal disease is particularly important for individual welfare and population management (Koch *et al.*, 2013; Jessop *et al.*, 2018; IUCN, 2021).

The renal lesions observed in the present case strongly support chronic kidney disease as the initiating event in the development of generalized visceral gout. Histologically, the kidneys exhibited diffuse interstitial fibrosis, severe glomerular atrophy, tubular degeneration and necrosis, granulomatous inflammation, and abundant intratubular urate tophi. Such chronic structural damage would markedly reduce both glomerular filtration and tubular secretion of uric acid, resulting in persistent hyperuricemia. Once plasma urate concentrations exceed their solubility threshold, monosodium urate crystals precipitate initially within renal tubules and subsequently accumulate on serosal surfaces and within highly vascularized organs, including the pericardium, lungs, gastrointestinal tract, spleen, and ovaries. The multisystemic distribution of urate deposits observed in this Komodo dragon closely mirrors the pathological pattern described in advanced visceral gout in other captive reptiles (Johnson and Watson, 2020; Halán *et al.*, 2022; Divers, 2025).

The inflammatory response induced by urate crystals has become increasingly well characterized over the past two decades. Although chemically inert, monosodium urate crystals are recognized by the innate immune system as damage-associated molecular patterns (DAMPs). After phagocytosis by resident macrophages, the crystals activate the NLRP3 inflammasome, triggering caspase-1 activation and subsequent maturation of interleukin-1β and interleukin-18. These cytokines amplify local inflammation by recruiting additional macrophages, heterophils, lymphocytes, and fibroblasts while promoting production of other inflammatory mediators, including tumor necrosis factor-α (Martinon *et al.*, 2006; Joosten *et al.*, 2020). Persistent crystal deposition therefore evolves into chronic foreign-body granulomatous inflammation characterized by multinucleated giant cells, macrophage-rich granulomas, and progressive fibrosis surrounding urate tophi. The numerous multinucleated giant cells and granulomatous nephritis identified in this case are entirely consistent with this well-recognized crystal-induced inflammatory mechanism.

As renal fibrosis progressed, nephron loss would have further impaired urate excretion, establishing a self-perpetuating cycle in which worsening renal dysfunction promoted additional hyperuricemia and continuous crystal deposition throughout the body. This positive feedback mechanism explains the generalized distribution of urate tophi observed both grossly and microscopically and is considered a hallmark of end-stage visceral gout in reptiles (Divers and Stahl, 2019; Johnson and Watson, 2020). Similar progression has been emphasized in contemporary reptile nephrology, where irreversible nephron loss is regarded as the principal determinant of systemic urate accumulation and poor clinical outcome (Divers, 2025).

The consequences of chronic renal failure extended well beyond urate deposition. Progressive renal insufficiency produces uremic syndrome, a complex metabolic disorder characterized by disturbances in calcium-phosphorus metabolism, electrolyte balance, acid-base homeostasis, and accumulation of uremic toxins. These alterations predispose reptiles to metastatic mineralization, vascular injury, myocardial degeneration, gastrointestinal ulceration, pulmonary hemorrhage, and impaired immune function (Divers and Stahl, 2019; Johnson and Watson, 2020). Accordingly, the myocardial hyaline degeneration, pulmonary mineralization, hemorrhagic gastritis, vascular mineralization, and multifocal hemorrhages documented in this Komodo dragon are most consistent with secondary consequences of chronic uremia rather than direct effects of urate crystal deposition alone.

An additional finding was multifocal gastric candidiasis associated with ulcerative gastritis. Rather than representing the primary disease process, this lesion was considered opportunistic and likely reflected prolonged systemic illness. Chronic renal disease is frequently accompanied by anorexia, malnutrition, mucosal injury, chronic physiological stress, and impaired immune competence, creating favorable conditions for opportunistic fungal colonization of the gastrointestinal tract. Thus, the gastric candidiasis observed in this Komodo dragon was interpreted as a secondary lesion resulting from chronic debilitating disease and uremia.

Reports describing naturally occurring visceral gout in *Varanus komodoensis* remain exceptionally limited despite the increasing importance of this species in zoological medicine and conservation programs. Given the endangered status of the Komodo dragon and its restricted natural distribution, comprehensive pathological characterization of spontaneous diseases provides valuable baseline information for clinicians, pathologists, and conservation biologists involved in the management of captive populations (Koch *et al.,* 2013; Jessop *et al.,* 2018; IUCN, 2021). Detailed documentation of naturally occurring renal disease may also facilitate earlier clinical recognition and improve preventive health strategies for large varanid lizards maintained under human care.

The pathological findings support a sequential pathogenic process in which chronic kidney disease initiated persistent hyperuricemia, followed by generalized monosodium urate crystal deposition, inflammasome-mediated granulomatous inflammation, progressive renal fibrosis, uremic syndrome, and ultimately multisystem organ failure. This case emphasizes that visceral gout should be regarded not merely as a consequence of hyperuricemia but as the culmination of chronic renal disease. Early detection of renal dysfunction, together with appropriate hydration, nutritional management, and routine monitoring of renal

health, is therefore essential to reduce morbidity and mortality in captive Komodo dragons and other large varanid species.

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