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Case report

# Atypical lethal systemic granulomatous inflammation caused by *Pseudomonas aeruginosa* in a green iguana (*Iguana iguana*): A postmortem case report

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## Abstract

A 5-year-old male green iguana (*Iguana iguana*) from a privately maintained outdoor enclosure was submitted for necropsy following a brief period of lethargy and anorexia, which rapidly progressed to death. A gross examination revealed coelomic and pericardial effusions, pulmonary consolidation, multifocal nodular lesions in multiple organs, most prominent and numerous in the lungs, accompanied by verrucous endocarditis. The histopathology report indicated widespread heterophilic and histiocytic granulomas with central necrosis and basophilic bacterial remnants in the lungs, heart, liver, and spleen. Microbiological cultures yielded *Pseudomonas aeruginosa* from coelomic and pericardial effusions, while special stains (acid-fast, PAS) and PCR for Mycobacterium spp. were negative, thereby ruling out *mycobacterial* and fungal infections. This is the first report of systemic granulomatous inflammation caused by *Pseudomonas aeruginosa* in a green iguana, underscoring its pathogenic potential in reptiles housed under suboptimal husbandry conditions and highlighting the importance of comprehensive diagnostics in exotic species.

**Keywords**: Granulomatous inflammation, Green iguana, Necropsy, Pseudomonas aeruginosa.

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# INTRODUCTION

*Pseudomonas aeruginosa* is a Gram-negative, rod-shaped, aerobic bacterium belonging to the class Gamma-proteobacteria, commonly inhabiting humid environments such as soil, aquatic habitats, and artificial surfaces. This opportunistic pathogen impacts hosts kept under poor husbandry conditions or with compromised immune systems (Wood et al., 2023). It can survive in various environmental conditions and is naturally resistant to many antimicrobial agents, making it a significant clinical and pathological concern in veterinary medicine (Morales-Espinosa et al., 2024; Nadăş et al., 2025).

*P. aeruginosa* is commonly isolated from reptiles kept in stressful, suboptimal, or immunocompromised captive conditions, where it acts as an asymptomatic colonizer or causes severe infections (Foti et al., 2013). In a retrospective study of exotic pets, *Pseudomonas* spp. accounted for 23% of all bacterial isolates from reptiles, making it the most common genus identified, with the highest prevalence of antimicrobial resistance among reptile isolates (Muñoz-Ibarra et al., 2022). Similarly, the most frequently isolated Gram-negative bacterium in a study among 129 isolates from 96 reptiles was *Pseudomonas* spp. (Tang et al., 2020), which commonly form part of the reptilian microbiota. They have been reported to cause localized lesions such as pneumonia, dermatitis, and stomatitis as well as systemic infections, but fatalities caused by this bacterium are relatively rare (Divers and Comolli, 2025). One such case described a fatal disseminated infection in a captive green iguana (*Iguana iguana*), highlighting the potential of this pathogen to cause severe disease in this species (Šupić et al., 2021).

Granuloma formation is a hallmark of inflammatory responses in reptiles, typically triggered by bacterial, fungal, or parasitic infections. Two principal types—heterophilic and histiocytic granulomas—exhibit distinct pathogeneses (Montali, 1988). Granulomas typically involve persistent pathogens like mycobacteria or fungi (Jacobson and Garner, 2021), but *P. aeruginosa* infections in reptiles have previously been associated with necrotizing lesions (Šupić et al., 2021). Granulomatous inflammation caused by *P. aeruginosa* is extremely uncommon in reptiles, although it has been documented sporadically in other species such as ostriches and cattle (Momotani et al., 1995; de Lima et al., 2020). This report describes the first documented case of disseminated granulomatous inflammation caused by *P. aeruginosa* in a green iguana, characterized by widespread heterophilic and histiocytic granulomas involving multiple organs, and occurring without identifiable immunosuppression or concurrent fungal/mycobacterial infections.

# CASE HISTORY

A 5-year-old intact male green iguana, weighing 1.9 kg, with a body condition score of 3/5 and total body length of 129 cm, was submitted for necropsy at the Center for Veterinary Diagnosis, Faculty of Veterinary Medicine, Mahidol University. The animal was housed alone in a 2 square meter outdoor enclosure with natural sunlight, cement flooring, wire mesh walls, and a vegetable-based diet provided in food and water containers. The enclosure had no artificial lighting or environmental enrichment such as climbing branches.

The owner stated that this iguana was one of ten kept under similar conditions. Over the previous three months, several iguanas in the group had developed oral lesions, labored breathing, and lethargy, with one to two animals dying each month. Some affected iguanas were taken to a veterinary hospital, where they were diagnosed with bacterial respiratory infections and treated with oral antibiotics but failed to improve and subsequently died. The submitted iguana exhibited decreased activity, anorexia, and abnormal resting posture for two days before death, and no veterinary intervention was sought.

## GROSS PATHOLOGY

No ectoparasites or skin lesions were observed during the necropsy. The oral cavity, eyes, and nostrils showed no gross lesions, but presented about 40 mL of slightly turbid yellowish serous coelomic effusion. Mild adhesions were observed between the thoracic wall, right lung lobe and pericardium. The lungs were consolidated and firm, exhibited a dark red discoloration, and contained multiple white to yellow nodules varying in diameter from 0.2 to 0.7 cm (Figure 1A). In cross-section, the white to brown nodules appeared firm and encapsulated. The pericardium was markedly thickened and contained yellowish, jelly-like material with 15 mL of serofibrinous effusion (Figure 1B).

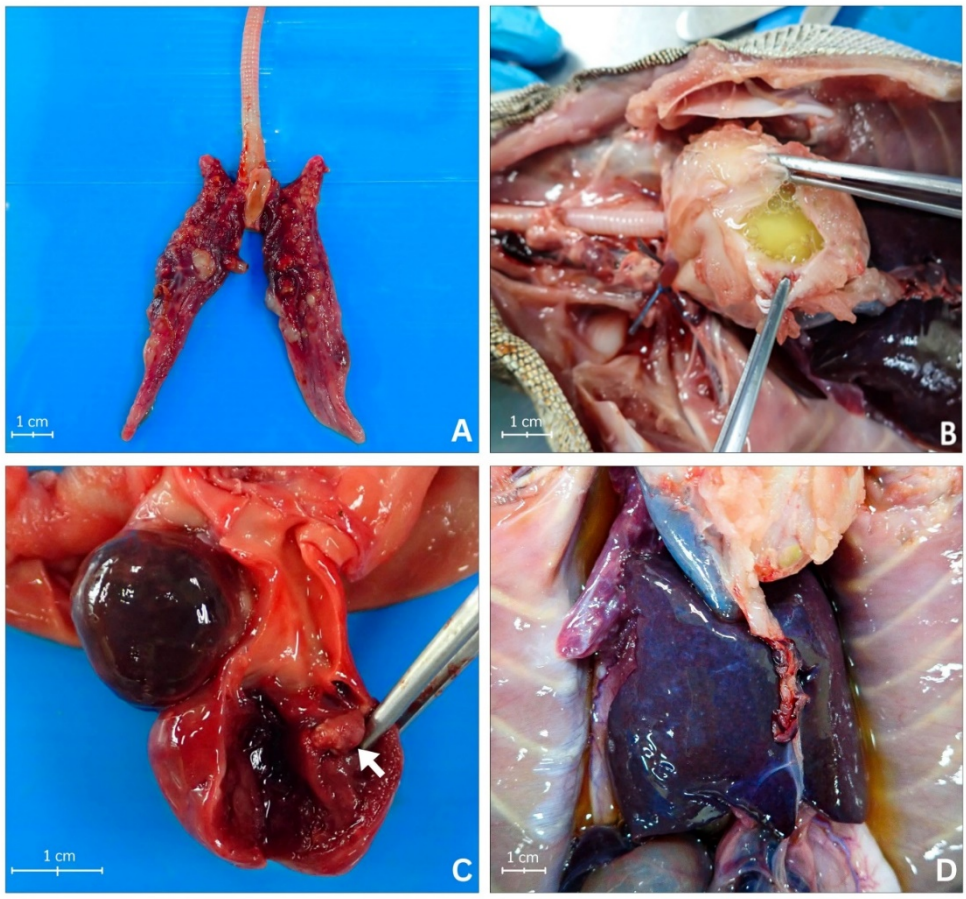


**Figure 1** Gross appearance. (A), The lungs show consolidation. Multiple nodules (0.2-0.7 cm in diameter), varying from white to yellow, are scattered throughout the parenchyma. The nodules are firm, encapsulated, and white to brown in color. (B), Thickened pericardium with accumulation of approximately 15 mL of serofibrinous effusion within the pericardial cavity. (C), The right atrioventricular valve shows marked distortion with proliferative, vegetation-like lesions (arrow), consistent with verrucous endocarditis. (D), The liver shows multifocal pale discoloration and numerous small white foci within the parenchyma.

A solitary 3 mm diameter dark red nodule was found at the left apex of the heart, and multiple small white foci were observed on the epicardium. The right atrioventricular valve demonstrated significant anatomical changes consistent with verrucous endocarditis, characterized by proliferative, vegetation-like lesions on the valve (Figure 1C). The liver exhibited pale discoloration and multiple small white foci on the surface (Figure 1D). The spleen was enlarged and swollen, with multiple small white nodules observed on the cut surface. No remarkable lesions were

observed in the gallbladder, pancreas, kidneys, or urinary bladder. In the gastrointestinal tract, the stomach contained mucus and large amounts of digesta; mild mucosal hyperemia was present throughout, with no other notable lesions.

## HISTOPATHOLOGY

Tissues from the lung, liver, spleen, and heart were collected for histopathological examination. The samples were processed following standard laboratory protocols, and fixed in 10% neutral of and stained with hematoxylin and eosin (H&E) before examination under a light microscope. All the sections were stained using acid-fast and periodic acid–Schiff (PAS) techniques to determine the presence of pathogens.

Histopathological examination of the lung, heart, liver, and spleen revealed multifocal granulomatous lesions affecting multiple organ systems, indicative of a systemic granulomatous inflammatory response. These granulomas showed two predominant types of granulomatous inflammation: heterophilic and histiocytic granulomas (Figure 2). Heterophilic granulomas (Figure 3A) featured central aggregates of necrotic tissue surrounded by intact heterophils. In contrast, histiocytic granulomas (Figure 3B) consisted primarily of densely packed macrophages (histiocytes) with a necrotic core, surrounded by palisading macrophages and occasional multinucleated giant cells. These lesions also contained radiating eosinophilic amorphous material consistent with the Splendore–Hoeppli phenomenon (SHP) and indistinct basophilic bacterial colonies (bacterial debris). Heterophils were occasionally present in the latter but in much lower numbers.

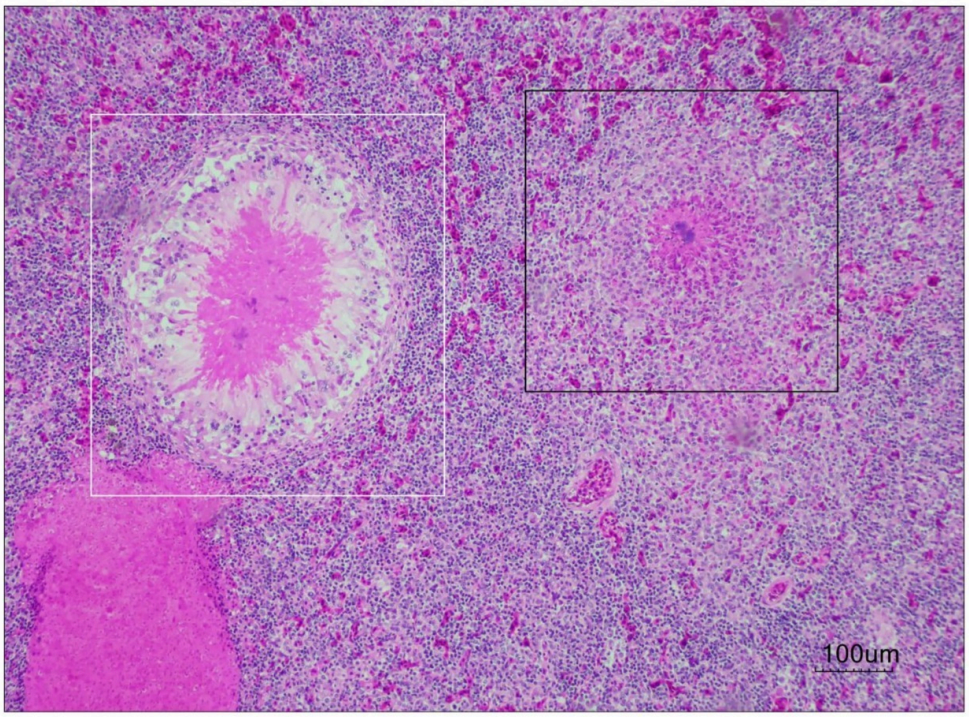


**Figure 2** Histology of Heterophilic and Histiocytic Granulomas. Hematoxylin and eosin (H&E) staining at 10× magnification reveals a heterophilic granuloma (black rectangle) and a histiocytic granuloma (white rectangle) in the liver of an iguana.

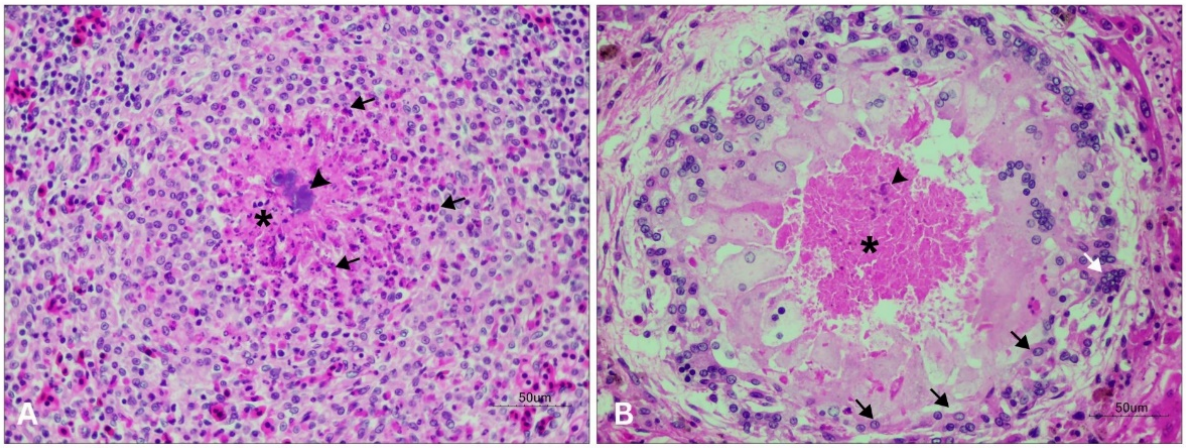


**Figure 3** Microscopic Characteristics of Granulomas. Hematoxylin and eosin (H&E) staining at 40× magnification. (A); Heterophilic granuloma shows a central area of necrosis (star) containing indistinct basophilic bacterial debris (arrowhead), surrounded by intact heterophils (black arrows). (B); Histiocytic granuloma consists primarily of densely packed macrophages (histiocytes) with a necrotic core (star), surrounded by palisading macrophages (black arrows) and occasional multinucleated giant cells (white arrow). These lesions are consistent with the Splendore–Hoeppli phenomenon (SHP) and contain indistinct basophilic bacterial debris (arrowhead).

In the lung (Figure 4A), the interstitial tissue showed moderate to severe expansion with marked hyperemia, edema, and substantial infiltration of heterophils and lymphocytes. Multiple granulomas were scattered throughout the lung parenchyma. The alveolar spaces contained polymerized fibrin, heterophils, macrophages, and cellular debris, while clusters of alveolar pneumocytes exhibited hyperplasia and dysplasia. In the heart (Figure 4B), multiple granulomas were distributed throughout the myocardium. Hepatic sections (Figure 4C) showed mild hepatocellular swelling and vacuolation in scattered regions, accompanied by diffuse aggregates of hemosiderin-laden macrophages within the parenchyma. Granulomas were also observed in multiple areas of the liver. The spleen (Figure 4D) exhibited enlarged lymphoid follicles of variable size and shape, with some coalescing into irregular structures, and granulomas were scattered throughout the splenic parenchyma.

H&E staining of all the examined organs revealed a systemic granulomatous inflammatory response, with some granulomas showing the SHP, a hallmark of chronic granulomatous inflammation. Scattered basophilic foci, resembling fragmented nuclear debris or patchy bacterial colonies, were occasionally present within the necrotic cores (Figure 4A-D and 5A). Histochemical tissue staining with periodic acid–Schiff (PAS) and Ziehl–Neelsen methods did not demonstrate fungal elements or acid-fast bacilli (Figure 5B, 5C). By contrast, Gram staining revealed scattered bacterial colonies with indistinct morphology, suggestive of Gram-negative bacilli and potentially consistent with *P. aeruginosa* (Figure 5D). Collectively, these findings were consistent with a systemic granulomatous inflammatory process involving multiple organs, with both heterophilic and histiocytic components, accompanied by tissue necrosis, cellular infiltration, and organ-specific pathological alterations.

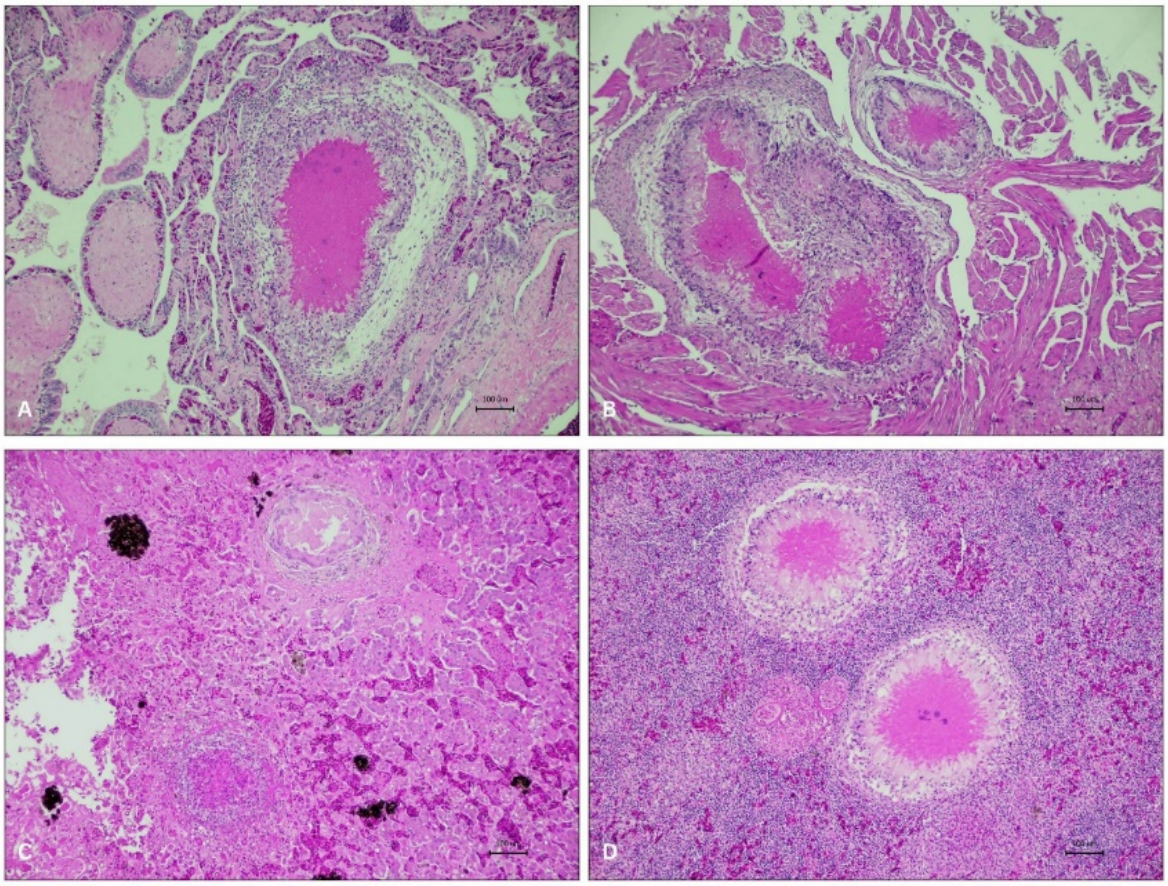


**Figure 4** Multiorgan Granulomatous Inflammation, Microscopic Appearance. Hematoxylin and eosin (H&E) staining at 10x magnification reveals widespread multifocal granulomas across multiple organs. (A); Lung tissue shows multiple granulomas, accompanied by alveolar fibrin, and reactive pneumocyte hyperplasia. (B); Multiple granulomas are identified throughout the myocardium. (C); Diffuse aggregates of hemosiderin-laden macrophages and multiple granulomas are observed within the hepatic parenchyma. (D); Multiple granulomas are diffusely scattered throughout the splenic parenchyma.

## MICROBIOLOGICAL AND MOLECULAR ANALYSES

Samples from nodular lesions of lung tissue, coelomic effusion, and pericardial effusion were submitted for bacterial culture to determine the causative agents associated with the systemic lesions. The coelomic and pericardial effusion cultures yielded *P. aeruginosa*, characterized by medium-sized, β-hemolytic colonies with a metallic sheen on blood agar, and pale, non-lactose-fermenting colonies on MacConkey agar. The isolate was a Gram-negative rod, oxidase- and catalase-positive, grew at 42°C, and showed typical biochemical traits including pyocyanin production, nitrate reduction, and citrate utilization. Lung tissue culture yielded *Enterococcus* spp., with small, grayish, non- to weakly α-hemolytic colonies on blood agar. The organism was Gram-positive cocci in pairs or short chains, catalase-negative, able to grow in 6.5% NaCl and at 10°C and 45°C, and hydrolyzed esculin in bile, consistent with *Enterococcus* spp. A multiplex PCR assay for *Mycobacterium* spp. was performed to investigate potential mycobacterial infection. DNA was extracted and amplified using primers targeting conserved genomic regions of the genus and specific pathogenic complexes. Gel

electrophoresis revealed no detectable amplicons, indicating a negative result for *Mycobacterium* spp. in the examined tissue.

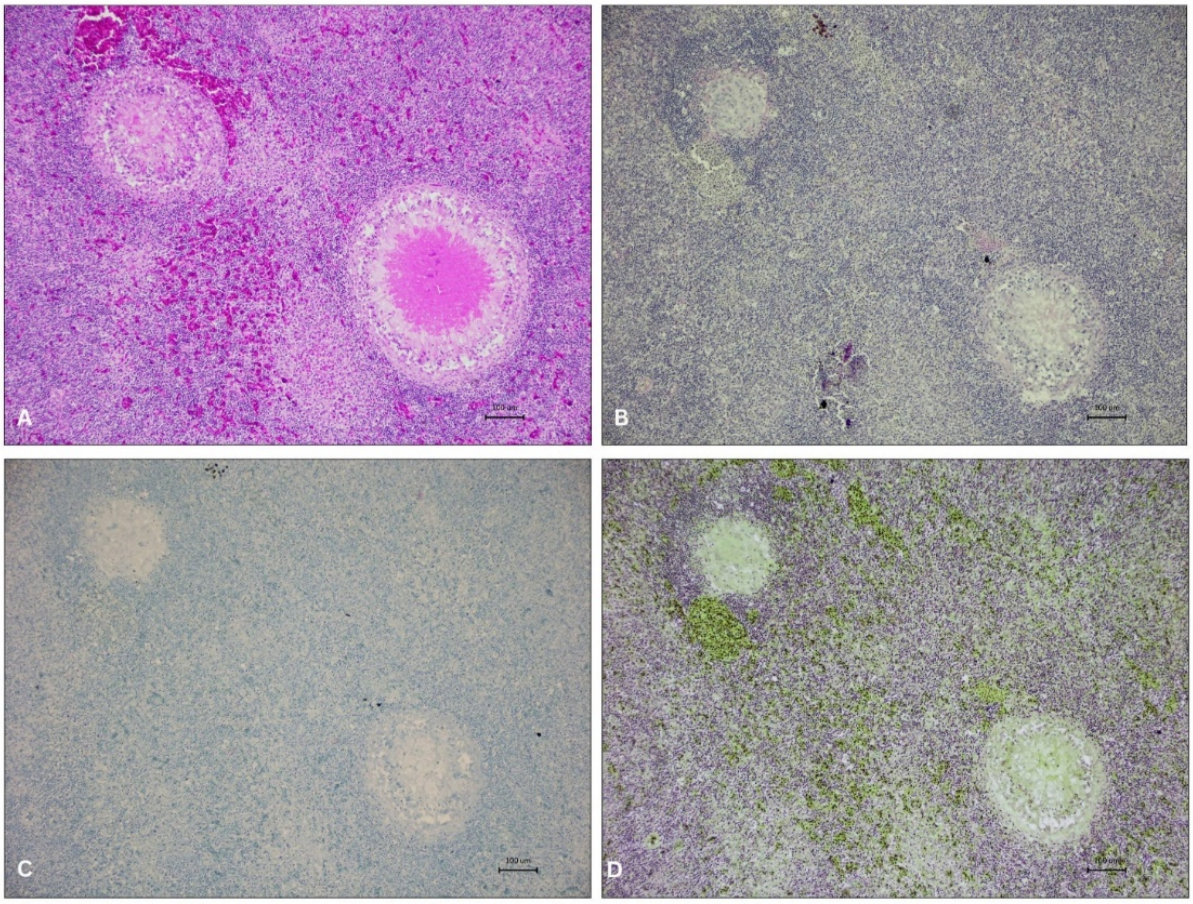


**Figure 5** Histochemical staining of granulomatous inflammation in the spleen. At 10× magnification, histochemical staining demonstrates the following findings: (A); Hematoxylin and eosin (H&E) stain shows granulomas associated with Splendore-Hoeppli phenomenon. (B); Periodic acid–Schiff (PAS) stain is negative for fungal elements. (C); Ziehl–Neelsen stain shows no acid-fast bacilli. (D); Gram stain shows scattered, poorly defined bacterial colonies, suggestive of Gram-negative bacilli and potentially consistent with *Pseudomonas spp*.

## INTERPRETATIVE SUMMARY

The microbiological and molecular findings, in conjunction with the necropsy, histochemical, and histopathological evidence of granulomatous inflammation and systemic lesions, supported *P. aeruginosa* as the primary etiological agent. *Enterococcus spp*. was likely a secondary or opportunistic pathogen, emphasizing the virulent nature of *P. aeruginosa* in causing rapidly progressive and fatal disseminated disease in this reptile.

# DISCUSSION

This report documents the first case of multisystemic granulomatous inflammation caused by *P. aeruginosa* infection in a green iguana. In this case, suboptimal husbandry practices, including insufficient lighting, lack of environmental enrichment, and poor ventilation may have compromised the animal's immune system, increasing its susceptibility to opportunistic infection. Reptiles often exhibit nonspecific or subtle clinical signs when developing respiratory diseases, allowing infections to progress without obvious symptoms (Schumacher, 2003). Consequently, diagnosis and intervention are often delayed. In this case, clinical signs such as lethargy and anorexia appeared only shortly before death, by which time the disease had already reached a terminal stage. These findings emphasize the challenges of early clinical detection and highlight the importance of proper environmental management in preventing opportunistic infections in captive reptiles.

Several previous reports also demonstrated the pathogenic potential of *P. aeruginosa* in other reptilian species, frequently resulting in severe inflammation, tissue necrosis, and mortality. For instance, cutaneous infections caused by *P. aeruginosa* were reported in Chinese crocodile lizards (*Shinisaurus crocodilurus*) at a breeding facility in China (Xiong et al., 2022), where the outbreak resulted in high morbidity and mortality. Similarly, the bacterium was identified as the causative agent of fatal necrotizing enteritis in a captive Nile monitor (*Varanus niloticus*) (Seixas et al., 2014). *P. aeruginosa* has also been associated with systemic granulomatous inflammation in non-reptilian species, such as a case in an ostrich (*Struthio camelus*) that developed multiple organ granulomas leading to death (Momotani et al., 1995). Granulomatous inflammation, characterized by the aggregation of macrophages and heterophils, typically arises in response to chronic infections. While such lesions have been documented in avian species, similar findings in reptiles have not been previously documented. Unlike the previously reported *P. aeruginosa* infection in a green iguana (Šupić et al., 2021), which exhibited multifocal necrotizing lesions dominated by heterophilic exudation, this case revealed well-developed heterophilic and histiocytic granulomas with central necrosis and peripheral palisading macrophages. The Splendore–Hoeppli phenomenon, characterized by intensely eosinophilic radiating material around bacterial remnants, was evident within multiple granulomas, reflecting a chronic antigen–antibody response and prolonged disease course. To the best of our knowledge, this is the first reported case of multisystemic granulomatous inflammation caused by *P. aeruginosa* in a reptile, highlighting a potentially underrecognized manifestation and the need for further pathological investigation in similar presentations.

The pathogenesis of *P. aeruginosa* infection is multifactorial, involving bacterial virulence factors and host immune responses. *P. aeruginosa* produces multiple extracellular enzymes and toxins, including elastases, proteases, hemolysins, and exotoxin A that facilitate tissue invasion, impair phagocytic function, and promote necrosis (Qin et al., 2022; Kessler, 2024). In reptiles, the host immune function is strongly influenced by environmental conditions (temperature, humidity, photoperiod), given their ectothermic physiology. Suboptimal environmental conditions impair innate and adaptive immunity, reducing the host's ability to contain bacterial proliferation (Field et al., 2022). As mentioned above, in most reptiles, *P. aeruginosa* infections are associated with acute necrotizing inflammation; however, the chronic granulomatous response observed in this case suggested prolonged antigenic stimulation and a shift toward a cell-mediated immune reaction dominated by macrophage and heterophil recruitment (Drake et al., 2019). This sustained immune activation led to the development of the SHP characterized by intensely eosinophilic material around microbial antigens within the granulomas, reflecting intense antigen–antibody interactions and immune complex deposition (Gopinath, 2018). These symptoms have been described in

bacterial infections including *Pseudomonas* spp., where chronic antigen presence induced immune-mediated encapsulation (Williams and Dellinger, 2016; Ecimovic et al., 2024). This immune response aims to localize the infection, but it may be insufficient to prevent systemic dissemination, ultimately resulting in multisystemic organ failure and death, especially in immunocompromised states (Bilevicius et al., 2001; Kumar et al., 2023).

In this case, both heterophilic and histiocytic granulomas were identified in multiple tissues, suggesting a prolonged inflammatory process involving acute and chronic immune responses. Central necrosis containing basophilic, poorly defined colonies—presumptively identified as bacterial fragments—supported a bacterial etiology, consistent with previous reports in reptiles and birds (Momotani et al., 1995; Seixas et al., 2014). Distinct Gram-negative bacilli were not clearly observed, but these colonies appeared as amorphous basophilic debris within the necrotic areas, and special stains failed to demonstrate definitive organisms. These foci may represent remnants of non-viable bacterial fragments, organisms embedded in biofilm, or degenerated nuclear material. The extensive central necrosis observed in the granulomas likely resulted from impaired clearance of apoptotic, infected macrophages by uninfected phagocytes—a process known as efferocytosis. When efferocytosis is insufficient, apoptotic macrophages undergo secondary necrosis, releasing viable bacteria and exacerbating tissue damage (Lyu et al., 2024). This mechanism provided a plausible explanation for the accumulation of necrotic debris and bacterial remnants in this iguana, but could not be confirmed in this case because the available histological findings did not permit direct assessment of the cellular clearance mechanisms. In the iguana granuloma, similar patchy eosinophilic material surrounding bacteria-like structures indicated that the SHP was due to Pseudomonas-associated immune response. However, the SHP is not pathognomonic to any single pathogen and has been previously reported in botryomycosis caused by non-filamentous bacteria, including *P. aeruginosa* in bovine visceral cases (de Lima et al., 2020).

A differential diagnosis was performed in this case to exclude other common infectious causes of granulomatous inflammation in reptiles. Fungi and parasites were excluded based on H&E and PAS staining, while mycobacterial infection was ruled out using acid-fast staining and PCR. Microbiological cultures of pericardial and coelomic effusions yielded *P. aeruginosa*.

In reptiles, the coelomic cavity encompasses the thoracic and abdominal organs, and any effusion present within this space bathes multiple visceral compartments. Therefore, the coelomic fluid serves as a representative sample of the entire intracoelomic environment for bacterial culture. In this case, *P. aeruginosa* was isolated from coelomic and pericardial effusions, both normally sterile compartments, indicating systemic dissemination. Pericardial effusion, in particular, strongly supports hematogenous spread of infection to the pericardium (Pankuweit et al., 2005). The concurrent histologic detection of the SHP in multiple organs further supported the occurrence of disseminated infection. The presence of verrucous vegetations on the cardiac valves in this case was consistent with bacterial endocarditis secondary to septicemia caused by *P. aeruginosa*, as reported in previous studies, although such demonstrations have been rarely documented in reptiles (Morpeth et al., 2007; Baddour et al., 2015). *P. aeruginosa* is a recognized cause of systemic infections in reptiles, especially under poor environmental conditions, and these findings strongly indicated septicemia (Šupić et al., 2021). Bacterial culture of lung tissue yielded *Enterococcus* spp. but was negative for *P. aeruginosa.* This discrepancy suggested a mixed infection, where *P. aeruginosa* acted as the primary systemic pathogen, while Enterococcus represented a secondary opportunistic invader. In reptiles, pulmonary tissue damaged by a primary infection often provides a niche for secondary colonizers such as Enterococcus, which are known commensals of the gastrointestinal tract and environment (Dubin and Pamer, 2017; Torres et al., 2018). One possible explanation for the absence of *P. aeruginosa* growth in lung culture was local

bacterial clearance by the host's immune response (McCaffrey et al., 2022), leaving only non-viable remnants at the time of sampling. *P. aeruginosa* may have persisted in the coelomic cavity and pericardium, where conditions favored its survival. These findings suggested that *P. aeruginosa* was the primary pathogen causing the systemic disease, whereas Enterococcus represented a secondary opportunistic invader of the already-damaged lungs. By contrast, chronic inflammation, immune cell infiltration, and fibrosis in the lung created a less hospitable environment, leading to bacterial clearance and leaving only debris detectable on histopathology. Mixed infections involving *P. aeruginosa* and other opportunistic bacteria have been previously documented in reptiles, particularly under suboptimal environmental conditions (Lazarkevich et al., 2024; Divers and Comolli, 2025).

Based on the pathological findings of widespread granulomatous inflammation, control and treatment of *P. aeruginosa* in reptiles should focus on environmental management and targeted therapy. Proper husbandry, including cleaning, ventilation, nutrition, and temperature control reduces multidrug resistance and prevents immune suppression (Sala et al., 2019). Early diagnosis through culture and antimicrobial susceptibility testing is crucial, as *P. aeruginosa* often exhibits multidrug resistance. Treatment should follow sensitivity results, typically using fluoroquinolones, aminoglycosides, or ceftazidime (Cristina et al., 2022), with supportive care to enhance immune response. Once systemic granulomatous lesions develop, therapeutic success is limited, highlighting the importance of preventive husbandry and early detection in captive reptiles.

This case highlighted the importance of a comprehensive diagnostic approach for systemic granulomatous disease in reptiles. Necropsy and histopathology revealed widespread granulomas, and bacterial culture from coelomic and pericardial effusions confirmed *P. aeruginosa* as the primary pathogen, with additional special stains and PCR ruling out mycobacterial and fungal infections. Integrating pathology, microbiology, and molecular techniques enabled a precise diagnosis, emphasizing that *P. aeruginosa* can mimic more familiar infections yet progress rapidly to fatal systemic disease in iguanas. Further studies on the occurrence, antimicrobial susceptibility, and pathogenic mechanisms of *P. aeruginosa* in reptiles are warranted to provide valuable guidance for veterinarians, breeders, and owners in implementing effective preventive measures, biosecurity practices, and evidence-based treatment strategies for iguanas and other reptile species.

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## CONFLICT OF INTEREST

The authors declare no conflict of interest.

## AUTHOR CONTRIBUTIONS

**Patanakorn Petaipanakij:** Writing – Original Draft Preparation(lead); Visualization(lead); Investigation(equal); Writing – Review & Editing(supporting) Conceptualization (supporting).
**Suriyo Klinsrithong:** Resources (equal).

**Sirima Chatrungruengkul:** Resources (equal).
**Sarita Paenkoed:** Resources (equal).
**Parin Suwannaprapha:** Supervision(lead); Writing – Review & Editing(lead); Conceptualization(lead); Project Administration(lead); Investigation(equal).

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