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MYCOTIC DERMATITIS IN CAPTIVE CARPET SNAKES
(Morelia spilotes variegata)

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Abstract: Geotrichum candidum was isolated from necrotic skin lesions in one of three captive carpet snakes (*Morelia spilotes variegata*). Hyphae and arthrospores morphologically consistent with this organism were present in histological preparations of lesions from the three snakes.

INTRODUCTION

Mycoses of reptiles appear to be rare. Reichenbach-Klinke and Elkan[10] cite only four reported cases and describe three others. This apparent lack has led us to report an outbreak of mycotic dermatitis in captive carpet snakes.

HISTORY

Five locally-captured mature carpet snakes, *Morelia spilotes variegata,* were housed in a solid-sided wooden crate (90 x 75 x 45 cm) with a wire mesh lid, and fed live laboratory mice. Brown paper was provided on the floor of the crate to minimize trauma to the snakes' skin during pursuit of prey, and the crate was cleaned of excreta every 3 to 4 weeks. To assist ecdysis, a large rock and a large container of water were provided.

Over a period of four months, three snakes developed multiple skin lesions, refused food and eventually died.

GROSS PATHOLOGY

Skin lesions occurred along the entire body length, and affected numerous single or small groups of adjacent scales mainly over the ventral and lateral body surfaces. The margins of affected scales first developed a brown discoloration which spread to involve the whole scale. Finally the epidermis of affected scales sloughed exposing the dry necrotic dermis (Fig. 1).

The carcasses appeared dehydrated and the kidneys and ureters contained large amounts of urates. All snakes had large intra-abdominal fat deposits.

Eleven adult and 10 immature nematodes (*Polydelphis anoura*) were found in the intestine of one snake. No ectoparasites were present.



FIGURE 1. Vertical body surface of affected snake with dark necrotic scales.

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MICROBIOLOGY

At necropsy, swabs and specimens taken from skin lesions were cultured aerobically at 37 C on sheep blood agar and MacConkey agar for bacteria, and at 28 C on Sabouraud Dextrose agar for fungi. A mixed flora of bacteria was isolated including *Pseudomonas aeruginosa, Acinetobacter* sp., *Streptococcus* sp., *Proteus* sp., micrococci and unclassified coliforms. A similarly mixed fungal flora was isolated, namely, *Curvularia* sp. and *Fusarium semitectum*[3] from one snake, *Rhisopus nigricans* and *F. heterosporum*[3] from the second, and *Penicillium* sp., *Aspergillus niger, Mucor* sp., *Aureobasidium* sp. and *Geotrichum candidum* from the third.

HISTOPATHOLOGY

Specimens for histology were fixed in 10% buffered neutral formalin, processed by the paraffin block technique, sectioned at 6 μm, and stained with haematoxylin and eosin. Selected tissues were stained with Grocott's Methenamine Silver (GMS) and Brown and Brenn Gram stain.[8]

Early skin lesions occurred in all snakes at hinge regions between scales. These consisted of necrotic foci in the epidermis and upper dermis containing large numbers of degenerate granulocytes (probably eosinophils). In more advanced lesions the entire scale was necrotic (Fig. 2). The necrotic tissue contained numerous fungal hyphae. These penetrated beyond the necrotic zones in two directions: into the underlying dermis, and within the stratum corneum at



FIGURE 2. Cross section of skin lesions (diagrammatic). Shaded areas indicate extent of necrotic tissue. A. Early lesion; B. Advanced lesion.

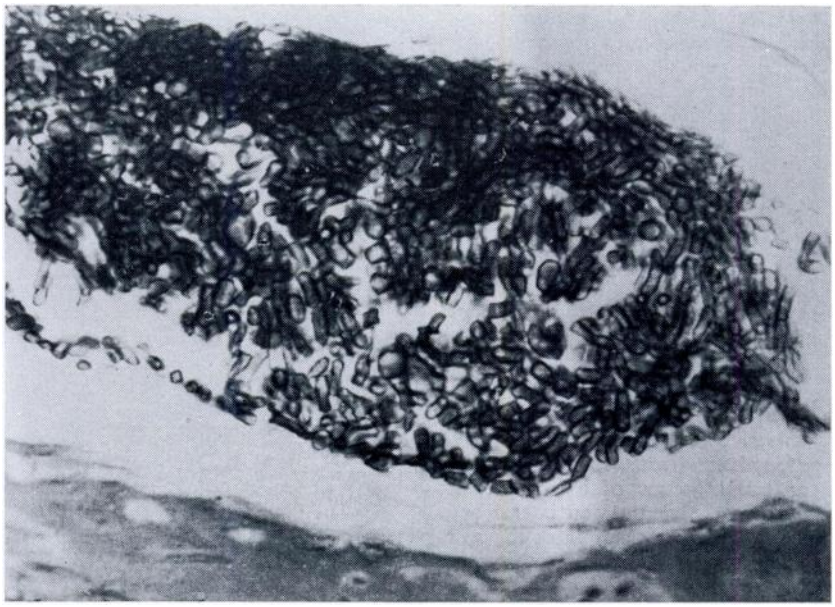


FIGURE 3. Mass of hyphal segments and arthrospores of **Geotrichum candidum** at the surface of a necrotic scale. GMS x 500

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the periphery of the lesions, causing vacuolation and necrosis of epidermal cells. Gram-positive cocci and gram-negative bacilli were present in the more superficial parts of the necrotic material, and accompanied the hyphae penetrating laterally in the stratum corneum.

The hyphae in the body of the lesion were regular, branching, septate, unpigmented and measured approximately 3 μm in diameter. At the lesion-air interface in the hinge regions between scales, hyphae were thickly matted and numerous segments extended into the air in pallisade arrays (Fig. 3). These aerial segments were fragmented into short cylindrical arthrospores approximately 5 to 7 μm in length.

DISCUSSION

A definitive diagnosis of the cause of death in these animals was not possible. Dehydration due to water loss from skin lesions, and possibly a toxaemia arising from them, were considered important contributing factors.

The morphology of the fungus in skin lesions was consistent with that of *G. candidum*.[5] This organism appeared to play the major part in the pathogenesis of these lesions. The development of lesions was thought to begin at the hinge region between scales. This area, being protected from direct contact with the exterior by the overlapping scales, and having a thinner stratum corneum,[2] may have been more susceptible to microbial attack than the exposed scale surface. Adverse environmental factors, such as high local humidity, also may have reduced the capacity of the skin in this area to resist infection. The lesions in our cases bear superficial resemblance to those of "blister disease" of reptiles due to excessive humidity,[12] but appear to be pathologically distinct from the vesicopustular lesions of that disease. However, Frye[6] illustrates gross lesions of dermatitis in a tegu lizard which closely resemble those in our snakes. He attributed these lesions to a contaminated environment. Necrotic dermatitis[4] also resembles the lesions in our cases.

G. candidum is recognised as a pathogen of the oral and respiratory mucosa of man and as a common saprophytic inhabitant of the intestine.[5] It has only very rarely been recognised as a pathogen of other animals.[1] Karstad[7] reported the isolation of *G. candidum* from caseous subcutaneous nodules in a captive banded watersnake (*Natrix sipedon*), and Page[9] cites Karstad as having isolated the same organism from pustules on the skin of a captive garter snake. These reports did not include descriptions of the pathology of lesions.

Generalised *G. candidum* infections have been experimentally produced in two species of turtles (*Chelydra serpentina serpentina* and *Pseudemys scripta elegans*) using isolates from plant and human sources and one isolate from a tortoise lung.[11] This study with turtles demonstrated the reciprocal pathogenicity of plant and animal isolates under experimental conditions. The present cases add to the evidence for the pathogenicity of this organism for reptiles.

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