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CASE REPORT

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Diagnosis, treatment, and outcome in a dog with systemic *Mycoleptodiscus indicus* infection

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Abstract

Objective: Describe the diagnosis, clinical course, and management of a dog with systemic *Mycoleptodiscus indicus* infection.

Case Summary: A 5-year-old male neutered Giant Schnauzer presented with left eye anterior uveitis, peripheral lymphadenopathy, hyperglobulinemia, anemia, and thrombocytopenia. A diagnosis of *M. indicus* infection was made based on histopathology and PCR. Treatment with itraconazole and terbinafine resulted in resolution of the hyperglobulinemia, anemia, thrombocytopenia, and peripheral lymphadenopathy. No evidence of fungal organisms was identified on lymph node, liver, or ocular histopathology after 7 months of treatment.

New or Unique Information Provided: This case is the first report of a systemic *M. indicus* infection in an apparently immunocompetent dog. Clinical resolution was achieved with systemic itraconazole and terbinafine.

KEYWORDS
fungal, hyperglobulinemia, opportunistic, phaeohyphomycosis, uveitis

1 | INTRODUCTION

Mycoleptodiscus indicus is a dematiaceous hyphomycete fungus that is a common plant pathogen in tropical and subtropical zones throughout the world.[1-3] In the last 2 decades, there have been increasing reports of *M. indicus* causing opportunistic phaeohyphomycosis in human and veterinary medicine.[4-8] Typically, it causes local cutaneous or subcutaneous infections after traumatic tissue inoculation. Initially reported in immunocompromised patients, this opportunistic fungus also may affect immunocompetent patients.[7,8]

2 | CASE SUMMARY

A 5-year-old male neutered Giant Schnauzer presented for left eye (OS) blepharospasm and mucoid discharge in December 2018. The dog was trained to find human remains in various environments throughout the United States, but predominantly resided in Florida. Pertinent past medical history included left pelvic limb 5th digit amputation because of chronic inflammation (April 2018).

Physical examination identified a hypermature cataract, anterior uveitis, and glaucoma OS and generalized peripheral lymphadenopathy. Laboratory findings included moderate hyperglobulinemia (6.7 g/dL; reference range [RR], 2.4-4.0 g/dL), mild normocytic normochromic nonregenerative anemia (Hct, 37.7%; RR, 38.3-56.5%; MCV, 71 fL; RR, 59-76 fL; MCHC, 34.5 g/dL; RR, 32.6-39.2 g/dL; reticulocyte count, $16 \times 10^3/\mu L$, RR, 10-110 $\times 10^3/\mu L$), and mild thrombocytopenia ($134 \times 10^3/\mu L$; RR, 143-448 $\times 10^3/\mu L$). Urinalysis showed an inactive sediment with no proteinuria and no bacterial growth was obtained on urine culture. Serum protein electrophoresis identified a spike in the beta fraction (5.33 g/dL; RR, 0.80-1.50 g/dL).

Abbreviations: FNA, fine needle aspirate; LN, lymph node; MIC, minimum inhibitory concentration; OD, right eye; OS, left eye; RR, reference range.

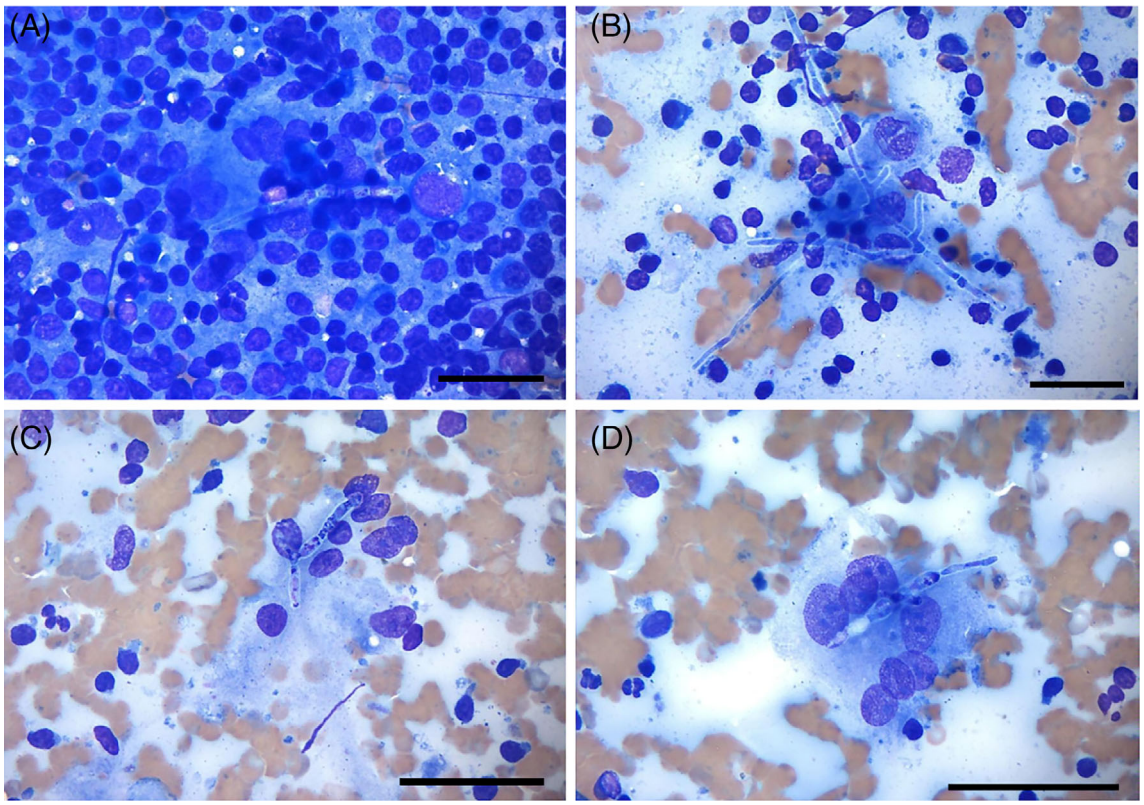

FIGURE 1 Cytology of peripheral lymph node (A, B) and spleen (C, D). A,B, Lymph node cytology contains mostly small mature lymphocytes with a moderate increase in the number of well-differentiated plasma cells. Occasional eosinophils, mast cells and macrophages are noted. Very rare fungal hyphae appear to branch at 45° angles, are septate, and contain an internal basophilic staining structure. Hyphae are approximately 1 to 2 μm in diameter. Low numbers of multinucleated macrophages are noted. C,D, Spleen cytology contains numerous large aggregates of splenic stroma with rare intralesional fungal hyphae occasionally engulfed within multinucleated macrophages. Wright-Giemsa stain. Scale bar = 25 μm

Thoracic radiographs disclosed sternal lymphadenopathy and a diffuse bronchial pattern. Abdominal ultrasound examination identified mesenteric lymphadenopathy and splenomegaly. Cytology of peripheral lymph node (LN), mesenteric LN, and spleen showed reactive lymphoid hyperplasia, moderate plasmacytosis, and rare intralesional fungal hyphae (Figure 1).

Peripheral LN aspirates were submitted for fungal culture and PCR. Aqueous humor cytology showed prior and ongoing hemorrhage but no fungal hyphae. Enzyme immunoassays (MiraVista Diagnostics, Indianapolis, Indiana) were negative for *Blastomyces*, *Aspergillus*, and *Coccidioides* antigens. A comprehensive infectious disease panel (North Carolina State University Vector Borne Disease Diagnostic Laboratory) was negative on indirect immunofluorescent assay for *Babesia* spp., *Ehrlichia* spp., *Rickettsia* spp., and *Bartonella* spp.; negative on PCR for *Babesia* spp., *Bartonella* spp., *Anaplasma* spp., *Ehrlichia* spp., *Rickettsia* spp., hemotropic *Mycoplasma* spp., and *Piroplasma* spp.; and negative on SNAP4Dx PLUS for *Borrelia burgdorferi*, *Ehrlichia* spp., *Anaplasma* spp., and *Dirofilaria immitis*. The dog was treated with itraconazole (8 mg/kg/day PO), tramadol (2.7-4.0 mg/kg to PO q8-12h), carprofen (2 mg/kg PO q12h), and ophthalmic preparations of 2% dorzolamide, 0.3% ofloxacin, 1% prednisolone acetate, and 1% atropine (OS q8h).

Laboratory testing a week later showed progressive hyperglobulinemia (7.2 g/dL; RR, 2.4-4.0 g/dL), but resolved anemia (Hct 44.4%; RR, 38.3-56.5%) and thrombocytopenia ($177 \times 10^3/\mu L$; RR, 143-448 $\times$ $10^3/\mu L$). Left popliteal LN histopathology showed lymphoid hyperplasia with no infectious organisms on routine staining. Silver stain showed occasional septate structures consistent with degenerate fungal hyphae (Figure 2). Fungal culture combined with phenotypic characterization and PCR sequencing identified *M. indicus* in the LN biopsy and aspirate specimens with >99% sequence homology (University of Texas Health in San Antonio). A segment of popliteal LN was submitted to a second laboratory for fungal identification. Results indicated 2 fungal species. The first species showed 93% sequence homology with *Rhizophydium aestuarii* (Washington Animal Disease Diagnostic Laboratory). The second species could not be identified because of low tracing amplitude.

Because of seroma formation after LN extirpation, cephalexin (25 mg/kg PO q12h) was started. Terbinafine (25 mg/kg PO q12h) was added to broaden the antifungal spectrum. One week later, peripheral lymphadenopathy and hyperglobulinemia (5.4 g/dL) were improved. Soon after, OS hyphema, left thoracic limb lameness, and pain on palpation of the left axilla developed. A CBC, left forelimb and cervical radiographs, magnetic resonance imaging, and skeletal

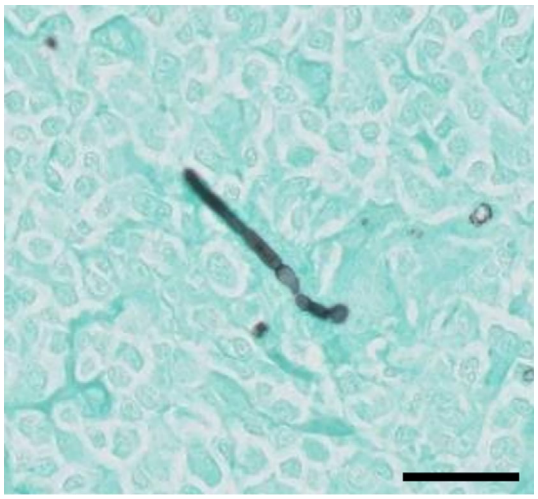

FIGURE 2 Left popliteal lymph node histopathology reveals occasional septate structures consistent with degenerate fungal hyphae scattered randomly throughout the cortex amid inflammatory cells and debris. These measure typically 3-3.5 μm in diameter and up to 40 μm long. Silver stain. Scale bar = 25 μm

scintigraphy disclosed no abnormalities. Because of progressive glaucoma and vision loss OS, enucleation was performed. Financial constraints precluded submission of OS for histopathology.

Serum itraconazole concentration was subtherapeutic (2.8 μg/mL; ≥3.0 μg/mL suggested for systemic infections) prompting a dose increase of itraconazole to 10 mg/kg/day PO. One month later, serum itraconazole concentration was within the suggested range (4.3 μg/mL). Serial serum biochemistry showed progressive improvement in hyperglobulinemia, with normalization 2 months after starting antifungal treatment. Globulins, Hct, platelet count, and peripheral LN size remained within normal limits throughout the 7 months of treatment. Antifungal medication was discontinued because of financial constraints.

Despite continued health and normal laboratory test results, the dog experienced progressive glaucoma and vision loss in the right eye (OD) and was euthanized 4 months after discontinuation of antifungals because of inability to care for a blind dog. Necropsy histopathology showed a normal spleen, minimal to mild plasmacytic periportal hepatitis with mild generalized biliary hyperplasia and mild to moderate cholestasis in the liver, reactive changes with mild edema in the LN, and mild melanocyte proliferation and mild loss of ganglion cells OD. Silver stains of all tissues failed to identify any fungal agents.

3 | DISCUSSION

Our report describes the clinical signs, diagnosis, management, and outcome of a systemic *M. indicus* infection in a dog with no known immune deficiency. Focal *M. indicus* infections have been described in 2 immunocompromised and 1 immunocompetent human patients.[4,5,7] The first report of *M. indicus* infection was in an immunosuppressed 72-year-old man from South Carolina with Wegener's granulomatosis being treated with methotrexate and prednisolone.[4] He suffered a local infection in his right knee after trauma while gardening. He was successfully treated with surgical debridement, irrigation, and amphotericin B. The second report of *M. indicus* infection was in an immunosuppressed 51-year-old Florida man with acquired immunodeficiency syndrome, *Pneumocystis jiroveci* (carinii) pneumonia, hepatitis C, and prior liver transplant being treated with antiretroviral drugs, methylprednisolone, and tacrolimus.[5] He developed cutaneous nodules on his right hand and forearm that were successfully treated with amphotericin B lipid complex, voriconazole, granulocyte-macrophage colony-stimulating factor, decrease of methylprednisolone, and discontinuation of tacrolimus. Cutaneous lesions resolved without recurrence 3 months after discontinuing antifungal drugs. The first patient presumably was injured during gardening. Lastly, another case report described *M. indicus* in an immunocompetent 54-year-old Canadian man who developed fungal arthritis of the knee after vacationing in Costa Rica where he was scratched by sharp leaves and a feral cat.[7] Antifungal sensitivity showed susceptibility of *M. indicus* to itraconazole (minimum inhibitory concentration [MIC] 0.5 mg/L) and fluconazole (MIC >64 mg/L). Susceptibility to terbinafine was not included. Clinical resolution was achieved with surgical debridement, methotrexate, and naproxen. No evidence of recurrence was identified during 36 months of monitoring.

In veterinary medicine, focal *M. indicus* infections have been reported in an immunocompromised dog and an immunocompetent cat.[6,8] An 8-year-old Pointer in Illinois with immune-mediated hemolytic anemia developed left hindlimb cellulitis with regional LN involvement. The dog had been receiving prednisone, cyclosporine, aspirin, famotidine, and doxycycline. The route of infection was unknown. Antifungal susceptibility testing showed sensitivity to itraconazole (0.25 μg/mL) and terbinafine (0.015 μg/mL). Susceptibility to fluconazole was not included. Despite treatment with itraconazole and terbinafine, the dog suffered progressive disease and died 2 months later from aspiration pneumonia. Previous reports of opportunistic systemic mycoses in immunosuppressed dogs showed improved response with antifungals and concurrent weaning of immunosuppressive drugs.[9] Unfortunately, this patient could not be tapered off immunosuppressive drugs without recurrent anemia.

A recent report of *M. indicus* in an immunocompetent 8-year-old cat in Georgia described another localized infection causing distal left forelimb cellulitis.[8] There was no known history of prior trauma, but it was suspected based on the patient's exposure to the outdoors. Full resolution was achieved after a 60-day course of fluconazole. No recurrence of clinical signs was reported in 10 months of follow-up.

All prior reports of infection with *M. indicus* have described local cutaneous and SC infections. Our case report confirms the potential for *M. indicus* to cause systemic infection. Furthermore, it indicates that systemic *M. indicus* in apparently immunocompetent patients may be treated with commonly utilized systemic antifungal drugs. Prior reports in people have described local infections treated with surgical debridement, systemic antifungal drugs or both, with no reports of recurrence after treatment. The SC infection in an immunosuppressed dog did not subside because the dog could not be tapered off immunosuppressive drugs. The SC infection in an immunocompetent cat showed clinical

resolution on fluconazole with no evidence of recrudescence 10 months after cessation of treatment. The authors concluded that the patient was successfully treated. However, it is uncertain if recrudescence would have occurred because the patient was euthanized 4 months after discontinuation of antifungal drugs.

With more frequent travel and expanding tropical and subtropical zones, atypical fungal infections, such as those caused by *M. indicus*, likely will become increasingly important differential diagnoses in both immunocompromised and immunocompetent patients. As such, it will be increasingly important to identify these organisms promptly and correctly. Unfortunately, in our case, it took 6 weeks after LN cytology and 2 weeks after LN biopsy to identify the causative agent. One of the 2 laboratories identified *M. indicus* on 2 separate submissions (LN aspirate and biopsy specimens) after forwarding the samples to a secondary facility. A portion of the extirpated LN was concurrently submitted to a second laboratory, which was unable to identify a causative agent based on its available gene bank. Two fungal species were sequenced, including 1 with too low an amplitude tracing to be identified and the other with 93% sequence homology with *R. aestuarii*. A 93% sequence homology is considered a low degree of sequence identity and only indicates relatedness. The second fungal species was characterized by a low tracing amplitude, perhaps reflecting either lower quantity of this second species or lower degree of affinity for the universal primers. The inability of a laboratory to identify the organism highlights the need to include *M. indicus* in more gene banks and include specific primers that might enhance the likelihood of identifying this organism. *Mycoleptodiscus indicus* has been reclassified, based on molecular phylogeny, as *Muyocopron laterale*.[10]

One limitation of our study was the lack of antifungal susceptibility. Future reports of opportunistic infections with *M. indicus* should include antifungal susceptibilities to help establish sensitivity profiles. Sensitivity profiles and efficacy studies for more common fungal infections (eg, blastomycosis, coccidioidomycosis) have been used to establish standard treatment protocols. Given the paucity of information regarding treatment of *M. indicus* infections in dogs, cats, and people, and the potential for systemic infection, performing antifungal susceptibilities on documented infectious strains will be helpful in directing treatment in future cases. Two of the 5 case reports reported antifungal susceptibility.[6,7] Although the infection in a cat responded to fluconazole, its MIC was higher compared to the other antifungal drugs tested.[7] Although *M. indicus* appeared susceptible to all antifungal drugs tested in previous reports, no defined breakpoints have been determined. This highlights the need for both antifungal susceptibility profiles and breakpoints. In our case report, the patient did not show improvement within 9 days of starting itraconazole, which was later found to be at subtherapeutic serum concentration. Terbinafine was added because of its reported synergy with itraconazole in treatment of a broad range of mycoses.[11-17] This resulted in apparent clinical resolution. Furthermore, no fungal organisms were identified on histopathology at the time of euthanasia 4 months after discontinuing antifungal drugs. It is possible that sole treatment with itraconazole, given more time and within the recommended dosage range, may have abolished systemic *M. indicus* infection. However, until additional research is done and susceptibility panels with breakpoints are defined, we recommend combined systemic treatment using itraconazole and terbinafine.

The route of infection in our case was not identified. It is suspected from other reports that infection was by direct inoculation of a wound after exposure to plants that serve as normal hosts. The patient was a cadaver-finding dog that traveled extensively throughout subtropical areas of the United States, including Texas, Tennessee, Florida, and other South Atlantic states, as well as other areas of the country, including the Pacific Northwest, Wyoming, the Midwest, and the East Coast as far north as New Hampshire. The dog had a chronically inflamed digit amputated 1 year before presentation at our hospital. It is unknown where or when the dog initially injured the digit. Histopathology with additional silver staining of the digit showed no fungal organisms. Still, the digit could have been the entry point for infection.

Response to treatment was judged in part by resolution of hyperglobulinemia, thrombocytopenia, and anemia. Protein electrophoresis identified a spike in the beta fraction. Proteins known to migrate to the beta fraction include transferrin, β-2 lipoprotein, immunoglobulin M, immunoglobulin A, and the positive acute phase proteins C-reactive protein, fibrinogen, complement factor 3a, and hemopexin.[18] The specific protein or proteins contributing to this spike were not determined. The β-globulins may be increased in acute or chronic inflammation.[19,20] Mild hyperglobulinemia (4.0 g/dL; RR, 1.6-3.6) was observed on the dog's laboratory testing 18 months before presentation at our hospital, indicating that infection may have been present chronically and the dog may have remained subclinical until development of progressive uveitis and glaucoma. No underlying B lymphocyte or plasma cell neoplasm was identified, and the hyperglobulinemia resolved with antifungal treatment. Therefore, a more appropriate term for this patient's protein abnormality may have been "restricted oligoclonal gammopathy," which results when infectious or immune-mediated diseases stimulate production of antibodies that are alike in charge and weight, resulting in similar migration during electrophoresis.[20] The nonregenerative anemia was presumed to be secondary to chronic inflammation. The thrombocytopenia was presumed to be secondary to decreased platelet production or life span, sequestration, or increased consumption as can be seen with other infections.[21,22] Both the anemia and thrombocytopenia resolved rapidly with antifungal treatment.

Another uncertainty was the link between the patient's systemic *M. indicus* infection and uveitis. Uveitis in dogs has been associated with various infectious agents, including mycotic (*Blastomyces dermatitidis*, *Histoplasma capsulatum*, *Coccidioides immitis*, *Cryptococcus neoformans*, *Aspergillus* spp.), rickettsial (*Ehrlichia canis*, *Rickettsia rickettsii*), algal (*Prototheca* spp.), bacterial (*Leptospira* spp., *B. burgdorferi*, *Brucella canis*, *Bartonella vinsonii* subsp. *berkhoffi*), parasitic (*D. immitis*, *Leishmania* spp., *Toxocara* spp.), and viral (infectious canine hepatitis) pathogens.[23] Extensive infectious disease testing excluded other possible infectious causes of anterior uveitis. Although other manifestations of the fungal infection (eg, lymphadenopathy, hyperglobulinemia, anemia, thrombocytopenia) resolved with antifungal treatment, the ocular manifestations progressed. Antemortem OS aqueous humor cytology and postmortem OD histopathology showed no infectious organisms. It is plausible that the

patient developed immune-mediated uveitis secondary to the systemic mycoses through molecular mimicry, epitope spreading, or persistent inflammation.[23] Severe cases of anterior uveitis may require systemic anti-inflammatory or immunosuppressive doses of corticosteroids if resolution does not occur with treatment of the underlying cause and also ophthalmic steroids. Therefore, the dog's anterior uveitis may have been related to the systemic *M. indicus* infection, despite its failure to resolve with systemic antifungal treatment.

To the authors' knowledge, this dog represents the first reported case involving systemic infection in an apparently immunocompetent dog with resolution of clinical signs after antifungal treatment. Clinicians should be aware of the potential for unusual fungal infections and the need for collaboration among clinicians, mycologists, and laboratories with capabilities for fungal testing. Future studies should include antifungal sensitivity and define tissue breakpoints for antifungal drugs. If concurrent anterior uveitis is seen, systemic corticosteroids may be warranted if the uveitis progresses despite successful treatment of the underlying mycosis.

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CONFLICT OF INTEREST DECLARATION

Authors declare no conflict of interest.

OFF-LABEL ANTIMICROBIAL DECLARATION

Itraconazole, terbinafine, doxycycline, and ofloxacin 0.3% ophthalmic solution used off-label in dogs.

INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

Authors declare no IACUC or other approval was needed.

HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

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