

# Space-occupying Lesion Within the Calvarium of a Cat

**What Is Your  
Diagnosis?**

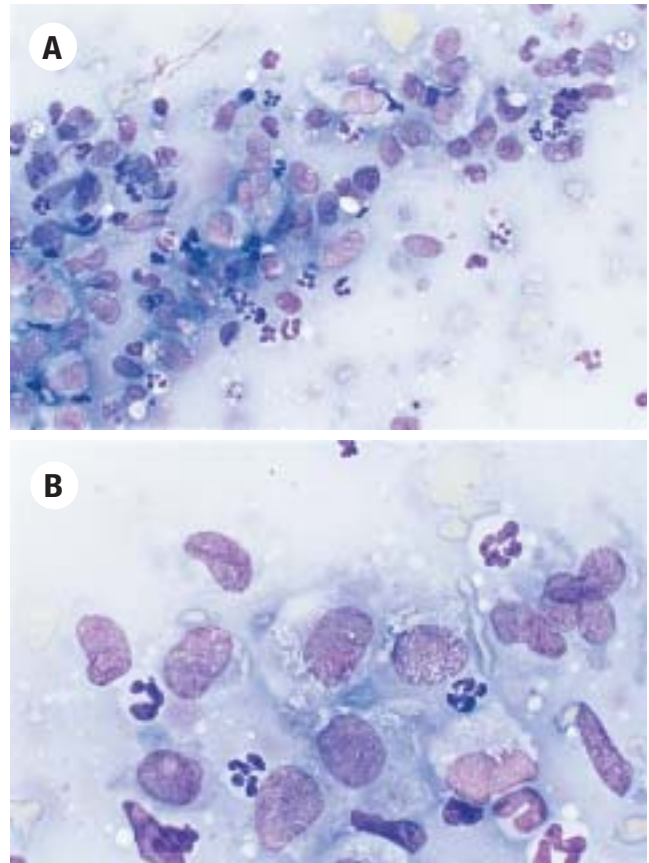
Melissa Blauvelt, Douglas Weiss, Alistair McVey, Jeff Bender, Elizabeth Aird

## Case Presentation

A 4-year-old, indoor, spayed female Domestic Shorthair cat was referred to the University of Minnesota Veterinary Teaching Hospital with a 4- to 6-week history of depression, decreased appetite, generalized muscle wasting, and a hypermetric gait. Diagnostic tests performed by the referring veterinarian were negative for feline leukemia virus and feline immunodeficiency virus infections, feline infectious peritonitis, and toxoplasmosis. No abnormalities were detected on whole-body radiographs. Results of a CBC revealed mild leukocytosis. The cat was treated with prednisone and initially improved; however, over several weeks, clinical signs worsened.

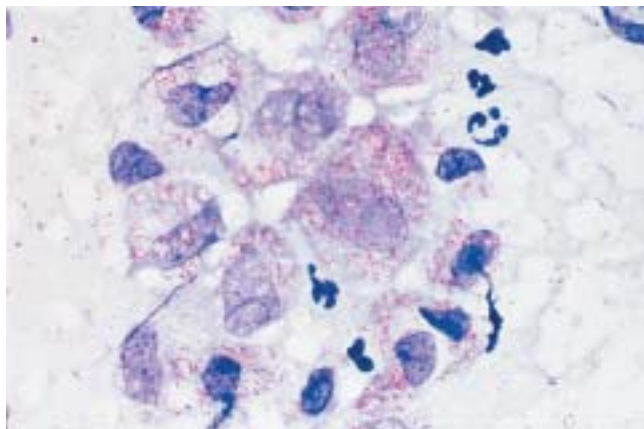
Upon presentation to the Veterinary Teaching Hospital, the cat was disoriented and agitated. Physical examination revealed blindness, tachycardia, tachypnea, and marked ataxia with left-sided neurological deficits. Results of a CBC included leukocytosis (WBC = 30,000/ $\mu$ L, reference interval, 4100-13,300/ $\mu$ L) with mature neutrophilia (22,880/ $\mu$ L, reference interval, 2100-11,200/ $\mu$ L). Results of a serum chemistry profile and urinalysis were within reference ranges. Evaluation of a computed tomography scan revealed a discrete right temporal lobe mass with compression of the right lateral ventricle. Because of the location and appearance of the mass, a meningioma was suspected. Exploratory craniotomy revealed a 1.5 $\times$ 1.0-cm mass in the right caudal aspect of the calvarium. The mass was surgically removed and impression smears were made for cytologic evaluation (Figure 1).

*(Continued on next page)*



**Figure 1.** Impression smears of a brain lesion from a cat. Wright's, (A)  $\times$ 100, (B)  $\times$ 500.

From the Department of Veterinary PathoBiology (Blauvelt, Weiss), Veterinary Small Animal Clinical Sciences (McVey), Clinical and Population Sciences (Bender), and Diagnostic Medicine (Aird), College of Veterinary Medicine, University of Minnesota, 1971 Commonwealth Ave, St Paul, MN 55108. Corresponding author: Dr. Melissa Blauvelt (blauv001@tc.umn.edu).



**Figure 2.** Impression smear of the brain lesion stained with Kinyoun-carbol-fuchsin. The macrophages contain long thin acid-fast bacilli consistent with *Mycobacterium* sp.  $\times 500$ .

### Cytologic Interpretation

Cytologic preparations were of low cellularity. The background was lightly eosinophilic, consistent with proteinaceous material. Cells consisted of clumped erythrocytes, small scattered clusters of macrophages, and non-degenerate neutrophils dispersed among the macrophages (Figure 1A). Cytologic findings were consistent with pyogranulomatous inflammation, characterized by approximately 60% nondegenerate neutrophils and 40% phagocytic macrophages. Occasionally, small lymphocytes and plasma cells were also noted. The phagocytic macrophages appeared foamy and were distended with significant numbers of negative-staining, long, thin, crescent-shaped bacilli (Figure 1B). The organisms were strongly acid-fast with Kinyoun-carbol-fuchsin stain, consistent with mycobacteria (Figure 2). *Nocardia* organisms are weakly acid-fast positive and frequently are noted extracellularly in large aggregates or "sulfur granules".<sup>1</sup> Therefore, the cytologic diagnosis was pyogranulomatous inflammation with mycobacterial infection.

### Additional Test Results

To identify a site of primary infection, whole-body radiographs were repeated; however, no lesions were seen in the thoracic or abdominal cavities. Blood, pleural fluid and urine were cultured under aerobic and anaerobic conditions, but no growth was detected. *Toxoplasma gondii* titers were done at the University of Minnesota and a positive IgM titer of 1:2048 was reported. The IgG titer was  $< 1:64$ .

Because the cat's condition worsened despite treatment with enrofloxacin (Baytril, Bayer, Shawnee Mission, Kans, USA), clindamycin (Antirobe, Pharmacia Upjohn, Kalamazoo, Mich, USA), and ethambutol HCl

(Myambutol, Lederle, Pearl River, NY, USA), the cat was euthanized 20 days postsurgery. Necropsy findings included cerebral- and leptomeningitis characterized histologically by granulomatous and pyogranulomatous inflammation and the presence of acid-fast organisms. No additional masses were evident in the brain. No other inflammatory lesions were found.

At the time of necropsy, bacterial culture of the mass and brain tissue was done using solid culture media and BACTEC 12B (Middlebrook 7H12) medium (Becton Dickinson Microbiology Systems, Sparks, Md, USA). The organism was identified as *Mycobacterium avium* complex using a chemiluminescent nucleic acid hybridization test (Accuprobe, Gen-Probe Inc, San Diego, Calif, USA).

### Discussion

Mycobacteriosis, in cats and other species, can manifest as 3 forms of disease depending on the causative organism.<sup>2,3</sup> These forms are categorized as classical tuberculosis, opportunistic or atypical infection, and leprosy. Classical tuberculosis results in nodular or granulomatous lesions. Organisms involved in feline infections include *M bovis*, *M tuberculosis*, and *M avium*.<sup>4,5</sup> Cats are most resistant to infection with *M avium* and least resistant to *M bovis*.<sup>2</sup> The location of lesions reflects the route of exposure to the organism, resulting in cutaneous, respiratory, or gastrointestinal tract disease, and frequently involving associated lymphoid tissue.<sup>6</sup> Disseminated disease is reported to occur in immunocompromised cats; however, none of the cats with disseminated disease were seropositive for feline leukemia virus or feline immunodeficiency virus.<sup>6</sup>

Cats most frequently acquire *M avium* infection from environmental sources such as soil or exposure to poultry.<sup>4,5</sup> *M bovis* infection may result from exposure to infected cattle or ingestion of infected rodents.<sup>5</sup> Feline infections with *M tuberculosis* most likely result from exposure to infected humans.<sup>4,5</sup>

Opportunistic or atypical mycobacterial infection usually results from organisms that are not generally considered pathogens and are ubiquitous in most environments.<sup>3</sup> These organisms include *M fortuitum*, *M chelonae*, *M marinum*, *M ulcerans*, and *M smegmatis*.<sup>2,5,6</sup> Lesions resulting from infections with these mycobacteria typically are cutaneous granulomas or draining tracts resulting from wound contamination.<sup>7</sup>

The third manifestation of mycobacteriosis is feline leprosy. *M lepraemium* results in cutaneous, sometimes ulcerative, nodular lesions, which rarely result in systemic disease.<sup>4</sup> Contact with infected rodents is typically the source of feline infections.<sup>2</sup>

Molecular techniques, which identify slow-growing

bacteria such as *Mycobacterium* via genotypic characteristics, have provided a significant diagnostic advancement over the past several years. These techniques are typically more rapid and sensitive than phenotypic characterization, which can take as long as 6 weeks.<sup>7</sup> The BACTEC system shortens the time typically required to culture mycobacterial organisms to 8-14 days.<sup>1</sup>

Identification of mycobacterial sepsis is of clinical significance in determining prognosis and treatment regimens as well for recognizing the zoonotic potential posed by an infected cat.<sup>8,9</sup> The zoonotic potential may be of greatest concern to immunocompromised humans. Over recent decades, the population of immunocompromised people in developed countries has increased significantly because of the growing prevalence of human immunodeficiency virus infection and aggressive chemotherapy regimens for patients with cancer, organ transplants, and autoimmune diseases. However, not all serologic and molecular epidemiologic studies support the presumed role of animals as a reservoir in human infections.<sup>2,10,11</sup>

The susceptibility of mice and humans to mycobacterial infections seems to be genetically determined. Certain alleles of the *Nrampl* gene are associated with susceptibility or resistance to a range of mycobacterial species.<sup>12</sup> Another gene, *IFNGR1*, when mutated, also is associated with susceptibility to mycobacterial infections.<sup>12</sup> Feline breed predilections to mycobacterial infection have been noted<sup>10</sup>; therefore, genetic predisposition may be a factor in the susceptibility of cats to mycobacterial infections.

To our knowledge, this case report is the first to doc-

ument a focal lesion within the calvarium of a cat, caused by *M avium*, in the absence of disseminated disease or an identifiable primary lesion elsewhere in the body. The location of the lesion described in this case is perplexing and is perhaps the result of hematogenous spread from a primary site of infection that subsequently resolved. Localized brain lesions in human patients are thought to be the result of hematogenous spread from a primary site elsewhere in the body.<sup>13</sup> Central nervous system infection in people develops in 2 stages. The first stage occurs during or shortly after a bacteremic phase, which may seed tuberculous lesions in the meninges, or the subependymal lining of the brain or spinal cord, and may remain dormant for years.<sup>13</sup> In the second stage, a stimulus can later result in growth of one or more of these lesions, resulting in CNS tuberculosis. Currently, it is presumed that host immunological mechanisms are the stimulus.<sup>13</sup> The 1:2048 IgM titer for *T gondii* in the cat in this case likely indicated concurrent *Toxoplasma* infection and thus possibly an immunocompromised state. ◇

#### Acknowledgments

We would like to acknowledge Dr Mary Walser (University of Minnesota Diagnostic Laboratory) for her gross pathology and histopathology contributions in this case.

**Key Words:** Cat, cytology, meningitis, *Mycobacterium avium*

**Citation:** Space-occupying lesion within the calvarium of a cat [mycobacteriosis]. *Vet Clin Pathol.* 2002;31:19-21.

© 2002 American Society for Veterinary Clinical Pathology

#### References

1. Spicer WJ. Specific pathogens. In: Horne T, ed. *Clinical Bacteriology, Mycology and Parasitology*, Edinburgh:Harcourt; 2000:55.
2. Taboada J, Merchant S. Protozoal and miscellaneous infections. In: Ettinger SJ, Feldman EC, eds. *Textbook of Veterinary Internal Medicine*. Vol 1. 5th ed. Philadelphia, Pa: WB Saunders; 2000:393-394.
3. Monroe WE, Chickering WR. Atypical mycobacterial infection in cats. *Feline Med.* 1988;10:1045-1048.
4. Gunn-Moore DA, Jenkins PA, Luck VM. Feline tuberculosis: a literature review and discussion of 19 cases caused by an unusual variant. *Vet Rec.* 1996;138:53-58.
5. Gunn-Moore DA, Shaw S. Mycobacterial disease in the cat. *In Pract.* 1997;19:493-500.
6. Paulsen DB, Kern MR, Weigand CM. Mycobacterial neuritis in a cat. *J Am Vet Med Assoc.* 2000;216:1589-1591.
7. Evans LM, Caylor KB. Mycobacterial lymphadenitis in a cat. *Feline Pract.* 1995;23:14-17.
8. Brahmer J, Small P. Tuberculosis and nontuberculous mycobacterial infection. In: Stein J, ed. *Internal Medicine*. Vol 1. St Louis, Mo: Mosby; 1998:1625-1639.
9. Grossman A. Mycobacterial hepatitis associated with long-term steroid therapy. *Feline Pract.* 1983;13:37-41.
10. Jordan HL, Cohn LA, Armstrong JP. Disseminated *Mycobacterium avium* complex in three Siamese cats. *J Am Vet Med Assoc.* 1994;204:90-93.
11. De Lisle GW. Mycobacterial infections in cats and dogs. *Surveillance.* 1993;20:24-26.
12. Newport M, Levin M. Genetic susceptibility to tuberculosis. *J Infect.* 1999;39:117-121.
13. Garg R. Tuberculosis of the central nervous system. *Postgrad Med J.* 1999;75:881:133-140.