Recovery of Two Mycoplasma Species from Abscesses in a Cat following Bite Wounds from a Dog

Robert D. Walker, Richard Walshaw, Craig M. Riggs and Theresa Mosser

J VET Diagn Invest 1995 7: 154
DOI: 10.1177/104063879500700126

testine of a pscittacine bird affected by inclusion body hepatitis. J Wildl Dis 28:319-322.
5. McCracken RM, Adair BM: 1993, Avian adenoviruses. *In:* Virus infections of birds, ed. McFerran JB, McNulty MS, pp. 123-144. Elsevier Science, New York, NY.
6. McFerran JB: 1991, Adenovirus (group I) infections of chickens. *In:* Diseases of poultry, ed. Calnek BW, Barnes HJ, Beard CW, et al., 9th ed., pp. 553-563. Iowa State University Press, Ames, IA.
7. Mori F, Touchi A, Suwa T, et al.: 1989, Inclusion bodies containing adenovirus-like particles in the kidneys of psittacine birds. Avian Pathol 18:197-202.
8. Nazerian K, Fadly AM: 1987, Further studies on in vitro and in vivo assays of hemorrhagic enteritis virus (HEV). Avian Dis 31:234-240.
9. Pass DA: 1987, Inclusion bodies and hepatopathies in psittatines. Avian Pathol 16:581-597.
10. Ramis A, Marlasca MJ, Majo N, Ferrer L: 1992, Inclusion body hepatitis (IBH) in a group of eclectus parrots *(Eclectus roratus).* Avian Pathol 21:165-169.
11. Scott PC, Condron RJ, Reece RL: 1986, Inclusion body hepatitis associated with adenovirus-like particles in a cockatiel (Psittaciformes; *Nymphicus hollandicus).* Aust Vet J 63:337-338.
12. Sileo L, Carlson HC, Crumley SC: 1975, Inclusion body disease in a great homed owl. J Wildl Dis 11:92-96.
13. Sileo L, Franson JC, Graham DL, et al.: 1983, Hemorrhagic enteritis in captive American kestrels *(Falco sparverius).* J Wildl Dis 19:244-247.

J Vet Diagn Invest 7:154-156 (1995)

Recovery of two mycoplasma species from abscesses in a cat following bite wounds from a dog

Robert D. Walker, Richard Walshaw, Craig M. Riggs, Theresa Mosser

Mycoplasmas, the smallest free-living microorganisms, may be found as normal flora on the mucous membranes of numerous animal species.[8,15] These organisms, having a limited genome for metabolic activity, require a close association with their host. They usually exhibit a high degree of host specificity, although there are instances where *Mycoplasma* species considered specific for 1 host have been isolated from other animal hosts. For example, *Mycoplasma felis,* which has been associated with conjunctivitis in cats, has also been isolated from clinically normal horses and horses with respiratory disease.[17] *Mycoplasma feliminutum* and *M. gateae* are both considered to be normal flora of cats but may also be part of the canine normal flora and have been isolated from pneumonic lesions in dogs.[14,15] *Mycoplasma arginini* has been isolated from dogs but may be considered as part of the normal flora in cats.[14,17] Other *Mycoplasma* species generally associated with mucous membranes and/or infectious disease processes in dogs include *M. canis, M. spumans, M. maculosum, M. edwardii, M. molare, M. cynos, M. opalescens,* and *M. bovigenitalium.*[14,17]

In the cat, mycoplasmas have been associated with infectious disease processes involving the conjunctiva, upper respiratory tract, and urogenital tract.[14,15] There are also 2 reports of mycoplasma-like organisms being associated with abscesses in cats.[2,7] One of these reports involved cervical and pulmonary abscesses, which developed following a cat fight. The second report described chronic abscesses in 3 different cats but did not indicate the initial cause. In each case, the infections persisted despite treatment with several antimicrobial agents. This report concerns a cat with multiple bite wounds inflicted by a dog. The wounds failed to resolve following surgical debridement and antimicrobial chemotherapy with both first- and second-generation cephalosporins. Two species of *Mycoplasma,* previously unreported in the cat, were isolated from the necrotic tissue associated with the wound.

A 25year-old intact male mixed-breed cat was examined at the Veterinary Teaching Hospital, Michigan State University, after being attacked by a pit bull terrier. Physical examination revealed multiple wounds over the right axillary and brachial regions. A large, open wound was located on the ventral midline over the thorax, which had extensive separation of the skin from the underlying subcutaneous tissues. Two puncture wounds were found near the ventral midline of the caudal edge of the wound. The most caudal wound was considered to have penetrated the thoracic cavity. An additional wound was located dorsal to the anus at the base of the tail. The cat was stabilized, and the wounds were appropriately cleaned and covered with bandages.

The following day the cat was taken to surgery for thorough surgical wound debridement and lavage. Following routine preparation, the large wound on the ventral thorax was carefully explored. All necrotic and devitalized tissue was removed, and the deeper muscle layers were apposed with sutures. Further wound closure was not attempted because of the size of the wound and the degree of tissue trauma. The

From the Departments of Microbiology (Walker) and Small Animal Clinical Sciences (Walshaw, Riggs) and the Animal Health Diagnostic Laboratory (Walker, Mosser), College of Veterinary Medicine, Michigan State University, East Lansing, MI 48824,

Received for publication March 3, 1994.

wound was covered with triple antibiotic ointment[a] and bandaged with a padded, nonadherent dressing. The smaller bite wounds were carefully debrided and closed because the degree of tissue damage was minimal. Daily bandage changes were performed for 4 subsequent days prior to discharge.

At the time of the initial examination, cephalothin[b] was administered intravenously (IV) at 24 mg/kg. This therapy was changed to cefoxitin[c] administered IV at 22 mg/kg every 8 hours preoperatively. After 24 hours, the therapy was changed to oral cephadroxyl[d] administered at 31 mg/kg every 12 hours.

Eight days after the initial surgical debridement, the cat was taken back to surgery for secondary closure of the open wound on the ventral thorax. Although wound healing was progressing in the majority of the wound, at the caudal extent several pockets of purulent material and necrotic fat were found under the skin. A sterile swab was used to collect samples for bacterial examination. The tissues were thoroughly debrided, penrose drains were placed, and the wound was closed. At the time of surgery, cephalothin (24 mg/kg) was administered IV, followed by cephadroxyl administered orally at 31 mg/kg every 12 hours. Partial dehiscence of the closure was noted by the third postoperative day. The penrose drains were removed on the fourth postoperative day. The wound was protected with a bandage until healing was complete.

The sample of exudate taken from the surgical site at the time of second debridement was immediately transported to the microbiological laboratory, where the swab was used to inoculate a trypticase soy agar[e] supplemented with 5% defibrinated sheep blood,[f] 1% horse serum and 1% yeast extract (EBA),[g] phenolethyl agar,[g] a MacConkey agar (MAC),[g] and a thioglycolate broth[g] supplemented with 1% hemin and 1% vitamin K[h] in an atmosphere of 5% CO_2. The MAC plate and the thioglycolate were incubated at 35 C aerobically.

After 24 hours of incubation, there was no distinct growth on any of the plates or in the broth. There was, however, a green haze observed in the area of inoculation on the EBA plate. After 48 hours of incubation, there were > 1,000 colony-forming units of a-hemolytic colonies on the EBA plates only. Because the organisms forming these colonies appeared as faint gram-negative pleomorphic bodies using the Gram stain procedure, the colonies were subcultured to a modified Hayflick's medium.[6] This medium consisted of pleuropneumonia-like organism agar[i] supplemented with 20% pig serum and 2.5% yeast extract) After 48 hours of incubation in a 10% CO_2 environment, there was growth of the typical "fried egg" colonies of mycoplasmas. Identification of the mycoplasmas, by indirect fluorescent antibody testing using antibodies produced in rabbits against American Type Culture Collection specific strains, revealed 2 different colony types, *M. canis* and *M. spumans.*

Following the identification of mycoplasma as the etiologic agent associated with the tissue necrosis and purulent exudate, the antimicrobial chemotherapy was changed to ciprofloxacin[k] administered orally at 20 mg/kg every 12 hours for 20 days. Ciprofloxacin was chosen because of its pharmacologic properties as well as its antimycoplasma activity. Also, although we do not routinely do susceptibility testing of mycoplasmas, we have tested some isolates that are resistant to tetracycline. The wounds from which the mycoplasmas were isolated had completely closed and were considered healed by the 15th day after the initiation of ciprofloxacin therapy. There have been no further complications.

Cats with fight wounds may be frequently encountered in small animal veterinary practices. The bacteria most commonly associated from infected fight wounds include *Pasteurella multocida,* [11,16] *Peptostreptococcus anaerobius,*[9] *Actinomyces* spp.,[12] *Bacteroides* spp.,[10] and *Fusobacterium* spp.[12] Following surgical debridement and/or antimicrobial chemotherapy using either penicillins or cephalosporins, these wounds generally resolve without any complications. However, there are some instances in which such wound infections persist or wounds fail to resolve despite what appears to be adequate, or even aggressive, surgical and antimicrobial therapy. When treating such wounds, the small animal practitioner needs to consider the possibility that an infectious agent such as mycoplasma may be present and should alter the course of therapy appropriately. Ideally, culture and susceptibility results from the infected tissue will provide the clinician with the information needed to make a correct therapeutic adjustment. However, not all laboratories are capable of isolating mycoplasma, and even fewer laboratories can determine their antimicrobial susceptibility.

Because mycoplasmas lack a rigid cell wall, they are resistant to cell wall active antimicrobial agents such as penicillins, cephalosporins, bacitracin, and vancomycin. If the wound has been contaminated with mycoplasma, chronic infections may develop when such antimicrobial agents are used. In this case and others, chronic infections have developed in the presence of ß-lactam antimicrobial therapy.[2,7]

In the present case, the use of the primary isolation medium, EBA, contributed to the isolation and identification of the mycoplasma associated with the cat's abscesses. Mycoplasmas have a limited genome and as such are highly fastidious and require an enrichment medium for in vitro cultivation.[8] The EBA contains both serum and yeast extract, which provides cholesterol and nucleic acid precursors that are required by mycoplasma for growth.[8] This medium has been used to recover mycoplasma from numerous types of infectious processes, the most frequent being urinary tract infections in dogs. However, when a mycoplasma-like organism was recovered from a pulmonary abscess in a cat using blood agar plates,[2] incubation for 4 days was required before pinpoint, a-hemolytic colonies were observed on the medium. In a second report, the authors indicated samples from the cats were used to inoculate blood agar and a modified Hayflick's medium.[7] They did not indicate which medium successfully isolated the mycoplasma-like organisms, but subculture growth was insufficient for further tests. In neither report were the isolated organisms specifically identified as mycoplasma using biochemical and serologic tests. Instead, the organisms were classified as mycoplasma-like because they produced "fried egg"-type colonies and the infections from which the organisms were isolated responded to treatment with tylosin (pulmonary abscess) or tetracycline.

In the report of the suspected mycoplasma infections associated with the pulmonary abscess, the source of organisms was thought to be a cat bite.[2] In the second report, the authors

speculated that the mycoplasma-like organisms were spread from cat to cat during treatment by the referring veterinarian.

In the patient of the present report, mycoplasmas were isolated from a soft-tissue infection following surgical debridement and aggressive antimicrobial chemotherapy of wounds inflicted by a dog bite. The 2 species of mycoplasma isolated have both been associated with the dog but have not previously been described in the cat, which suggests that the source of these mycoplasmas was the dog.

Once the etiologic agents associated with these abscesses were identified as mycoplasmas, antimicrobial therapy was changed from cephalosporin to ciprofloxacin. This drug has been shown to be effective against mycoplasma[13] and has an excellent volume of distribution with good penetration into both tissues and cells.[1,3-5,18] In the dog, ciprofloxacin has a long serum half-life, making it suitable for twice-a-day dosing.[18] These characteristics permit ciprofloxacin to achieve therapeutic concentrations at the site of infection. Other antimicrobial agents, such as erythromycin, tetracyclines, chloramphenicol, lincomycin, clindamycin, and the aminoglycosides, are also active against mycoplasma.[15] However, because of previous experience with the fluoroquinolones and their reported activity against mycoplasma, we selected ciprofloxacin. This cat's abscesses resolved with ciprofloxacin therapy, and there were no further complications. The successful resolution of this case with ciprofloxacin and appropriate surgical management suggests that this drug, and possibly the other fluoroquinolones, are effective in treating infections caused by mycoplasma species. However, additional cases should be evaluated before the therapeutic efficacy of the fluoroquinolones against *Mycoplasma* spp. can be confirmed.

Sources and manufacturers

a. NMC Labs, Glendale, NY.
b. Eli Lilly & Co., Indianapolis, IN.
c. Merck Sharpe & Dohme, West Point, PA.
d. Fort Dodge Labs, Fort Dodge, IA.
e. BBL-Becton Dickinson, Cockeysville, MD.
f. Cleveland Scientific, Bath, OH.
g. GIBCO Laboratories, Grand Island, NY.
h. Sigma Chemical Co., St. Louis, MO.
i. Difco, Detroit, MI.
j. Red Star Active Dry Yeast, Universal Food Corp., Milwaukee, WI.
k. Miles, West Haven, CT.

References

1. Bergan T, Dalhoff A, Rohwedder R: 1988, Pharmacokinetics of ciprofloxacin. Infection 16(suppl. 1):S3–S13.
2. Crisp MS, Birchard SJ, Lawrence AE, Fingeroth J: 1987, Pulmonary abscess caused by a *Mycoplasma* sp. in a cat. J Am Vet Med Assoc 191:340-342.
3. Crump B, Wise R, Dent J: 1983, Pharmacokinetics and tissue penetration of ciprofloxacin. Antimicrob Agents Chemother 24: 784-786.
4. Dalhoff A, Eickenberg HU: 1985, Tissue distribution of ciprofloxacin following oral and intravenous administration. Infection 13:78-81.
5. Drusano GL, Standiforod HC, Plaisance IS, et al.: 1986, Absolute oral bioavailability of ciprofloxacin. Antimicrob Agents Chemother 30:444-446.
6. Hayflick L: 1965, Tissue cultures and mycoplasmas. Tex Rep Biol Med 23(suppl. 1):285-303.
7. Keane DP: 1983, Chronic abscesses in cats associated with an organism resembling mycoplasma. Can Vet J 24:289-291.
8. Kenny GE: 1991, Mycoplasmas. *In:* Manual of clinical microbiology, ed. Hausler WJ Jr, Herrmann HL, Isenberg HD, Shadomy HJ, 5th ed., pp. 478-482. American Society for Microbiology, Washington, D.C.
9. Love DN, Jones RF, Bailey M: 1979, Description of strains of *Peptostreptococcus anaerobius* isolated from subcutaneous abscesses in cats. J Gen Microbiol 112:401-403.
10. Love DN, Jones RF, Bailey M: 1981, Characterization of *Bacteroides* species isolated from soft tissue infections in cats. J Appl Bacteriol 50:567-575.
11. Love DN, Jones RF, Bailey M, Johnson RS: 1979, Isolation and characterization of bacteria from abscesses in the subcutis of cats. J Med Microbiol 12:207-212.
12. Love DN, Jones RF, Bailey M, et al.: 1978, Bacteria isolated from subcutaneous abscesses in cats. Aust Vet Pract 8:87-90.
13. Ridgway GL, Mumtaz G, Gabrial FG, et al.: 1984, The activity of ciprofloxacin and other 4-quinolones against *Chlamydia trachomatis* and mycoplasmas in vitro. Eur J Clin Microbiol 3:344-346.
14. Rosendal S: 1979, Canine and feline mycoplasmas. *In:* The mycoplasmas, vol. II, pp. 217-234. Academic Press, New York, NY.
15. Rosendal S: 1990, Mycoplasmal infections. *In:* Infectious diseases of the dog and cat, ed. Green CE, pp. 446-449. WB Saunders Co., Philadelphia, PA.
16. Smith JE: 1964, Symposium on diseases of cats. III. Some pathogenic bacteria of cats with special reference to their public health significance. J Small Anim Pract 5:517-524.
17. Stalheim OHV: 1990, Mycoplasmas of animals. *In:* Diagnostic procedures in veterinary bacteriology and mycology, ed. Carter GR, Cole JR Jr, 5th ed., pp. 343-370. Academic Press, New York, NY.
18. Walker RD, Stein GE, Hauptman JG, et al.: 1990, Serum and tissue cage fluid concentrations of ciprofloxacin after oral administration of the drug to healthy dogs. Am J Vet Res 51:896-900.