

## Morganelliasis Pneumonia in a Captive Jaguar

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**ABSTRACT:** Suppurative bronchopneumonia was discovered in a 6-yr-old male jaguar (*Panthera onca onca*) that died after a 1 wk history of anorexia, depression, and respiratory difficulty. *Morganella morganii* was isolated as a pure culture from the lung, spleen, and heart blood. This is the first record of *M. morganii* induced pneumonia in a jaguar.

**Key words:** Amazon jaguar, case history, *Panthera onca onca*, *Morganella morganii*, pneumonia.

*Morganella morganii*, previously known as *Proteus morganii*, belongs to the family Enterobacteriaceae (Quinn et al., 1994; Koneman et al., 1997). It is a Gram-negative, urease-positive bacillus that deaminates phenylalanine, grows on a potassium cyanide medium, ferments xylose, and is motile (Koneman et al., 1997). There are two subspecies: *M. morganii morganii* and *M. morganii sibonii*, depending on the ability to utilize trehalose (Jensen et al., 1992). Furthermore, *M. morganii* is part of the normal fecal flora and is classified as an opportunistic pathogen known to cause infection in humans and animals (Quinn et al., 1994).

Information on bacterial pneumonia in the wild Felidae is scarce, and most available data are based on comparative findings of domestic cats and dogs. Pneumonia associated with *M. morganii* has not been reported previously in these animals. In the present report, we describe a case of *M. morganii*-associated pneumonia in a jaguar (*Panthera onca onca*).

A 6-year-old male jaguar had been injected with ampicillin (5 mg/kg/day; Bayer Inc., Seoul, Korea) and cephazolin sodium (10 mg/kg/day; Yoohan Inc., Seoul, Korea) for 1 wk due to traumatic skin injury on the right chest area. Although the injury was fully healed, the jaguar then presented with clinical signs of anorexia, weight loss,

depression, and respiratory difficulty 6 wk after the termination of treatment. The jaguar was unresponsive to ampicillin (10 mg/kg/day) and was found dead 3 days later. Postmortem examination was performed at College of Veterinary Medicine (Seoul National University, Suwon, Republic of Korea).

On necropsy, the trachea and bronchi were found to contain moderate amounts of blood-tinged frothy fluid and purulent exudate. Cranioventral areas of both cranial and cardiac lung lobes were consolidated, and a small amount of yellowish-white purulent exudate oozed from the cut surface. The distal one sixth of the esophagus was filled with regurgitated ingesta.

Lung, liver, spleen, heart, kidney, stomach, small intestines, and large intestines were collected, fixed in 10% neutral buffered formalin, and processed through routine methods for light microscopic examination. For bacteriology, portions of the lung, liver, spleen, and heart blood were taken aseptically, inoculated onto a blood agar plate containing 5% defibrinated sheep blood, and incubated aerobically and anaerobically at 37 C overnight. After incubation, suspect colonies were collected and identified through the Vitek system (Biomérieux Vitek, Hazelwood, Missouri, USA). Antimicrobial susceptibility test of the isolate was performed using 15 antimicrobial drugs through the disk diffusion method (Becton Dickinson Microbiological Systems, Cockeysville, Maryland, USA).

Microscopically, bronchioles and the associated alveolar spaces were filled with desquamated or necrotic epithelial cells, neutrophils, a few mononuclear cells, serofibrinous fluid, and red blood cells. The alveolar septa were thickened due to con-

gestion and mild infiltration of mixed inflammatory cells. No significant microscopic changes were noted in any other parenchymal organs examined.

Grayish colonies with hemolysis on the blood agar were isolated as a pure culture from the lung, spleen, and heart blood and identified as *M. morganii* through the Vittek system, indicating that *M. morganii* was the etiological agent of the suppurative pneumonia in this case. The bacteria were resistant to tylosin, bacitracin, tetracycline, streptomycin, and norfloxacin, while susceptible to nalidixic acid, chloramphenicol, carbenicillin, ampicillin, gentamicin, neomycin, sulfamethoxazole/trimetoprim, enrofloxacin, amikacin, and cefotofur. Since the necropsy was performed shortly after death (maximum 3 hr), the organism isolated was regarded as the primary pathogen rather than a simple post-mortem overgrowth. Diagnosis of *M. morganii*-induced pneumonia was made based on histopathology and bacteriology.

*Morganella morganii* is a relatively common cause of urinary tract infections in man and animals (Koneman et al., 1997). It also has been determined to cause diarrhea, wound infection, and septicemia. Furthermore, meningitis caused by *M. morganii* was recently reported in a patient with AIDS (Mastroianni et al., 1994). Severe suppurative polyarthritis, secondary to the bacteremia caused by *M. morganii*, was discovered in a West African dwarf crocodile (Heard et al., 1988). Along with five other species in family Enterobacteriaceae that are not commonly associated with diseases, *M. morganii* was documented in Gram-negative septicemia in American alligators (*Alligator mississippiensis*) (Novak and Seigel, 1986). Moreover, *M. morganii* also is considered as a possible cause of swollen head syndrome of broiler chickens in Japan (Tanaka et al., 1995).

Generally, *M. morganii* causes disease in sites previously infected by other organisms and may cause pyogenic infection when accidentally introduced into the

body. However, the exact source and mode of infection of our case study could not be determined. No evidence of a urinary tract infection was noted in this jaguar during necropsy. Since the animal seemed to have been immunologically compromised following the skin injury, it is possible that *M. morganii* as part of the normal fecal flora may have entered into the body through the aerosol of the contaminated fecal material, thereby causing pneumonia and secondary bacteremia. Immunocompromise following feline leukemia virus or feline immunodeficiency virus might serve as a predisposing factor for *M. morganii* infection, but the presence of such viral infection was not examined. It is also possible that the development of pneumonia was secondary to septicemia, which originated at a site other than the lung.

Although *M. morganii* as pneumonic pathogen in animals needs to be further elucidated, this case documents the pathogenic potential of this organism. Suggestion is thus made that this organism should always be considered in the differential diagnosis of bacterial pneumonia in domestic and wild animals.

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