Suppurative rhinitis associated with *Haemophilus* species infection in a cat

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ABSTRACT

A young cat with signs of chronic rhinitis was evaluated for underlying anatomical, inflammatory, or infectious disease. Initial diagnostics were significant for the isolation of an unusual pathogen, *Haemophilus* species. Isolation using a human RapID™ NH system erroneously identified the isolate as *H. segnis,* a human pathogen. No database of veterinary pathogens (*Haemophilus*) are included in the system and animal pathogens will either be erroneously identified or yield a unique biocode not listed. Because of the unique nature of the pathogen we explored the possibility of immunosuppression as a contributory factor to infection. A variety of laboratory tests were employed to evaluate immune function. The clinical indications and utility of immune function testing are discussed. No immune dysfunction was identified.

Key words: cats, flow cytometry, *Haemophilus segnis*, immune function, neutrophil respiratory burst, upper respiratory tract infection.

Milner R J, Horton J H, Crawford P C, O'Kelley J, Nguyen A **Suppurative rhinitis associated with *Haemophilus* species infection in a cat**. *Journal of the South African Veterinary Association* (2004) 75(2): 103–107 (En.). Department of Small Animal Clinical Sciences, College of Veterinary Medicine, University of Florida, PO Box 100126, Gainesville, FL 32610-0126, USA.

INTRODUCTION

Cats with persistent or recurrent nasal discharge and sneezing are familiar and frustrating visitors to the general practitioner and the specialist alike. When the history, physical examination findings, or diagnostic tests rule out other causes of nasopharyngeal disease, chronic rhinitis is usually attributed to some manifestation of viral upper respiratory tract disease. Previous infection with feline herpesvirus-1 may cause permanent damage to the nasal turbinates, or recrudescence of latent viral infection may lead to recurrence of signs associated with the original infection. In either event, secondary bacterial infection is expected. Chronic rhinitis infrequently resolves; repeated courses of antibiotics may be required to palliate clinical signs. Most often, therapy is empirical; bacterial culture and susceptibility tests are costly and, owing to the variety of normal flora present, may be difficult to interpret. The mechanism by which disease persists in these cats is incompletely understood. Albeit limited, the success of immunomodulating therapies lends support to the hypothesis that the chronic rhinitis syndrome may represent an ineffective immune response to persistent viral infection[26].

The case reported here is typical of those seen in general practice: a young adult cat with persistent nasal discharge and sneezing whose signs are only temporarily ameliorated by courses of broad-spectrum antibiotics. Unexpectedly, an unusual bacterium was isolated from the nasal passages. The case is significant for at least 2 reasons: *Haemophilus* is only rarely reported with upper respiratory tract disease in the cat and can easily be overlooked[14,19] and we believe this to be the 1st report of immune function testing (phagocytosis, oxidative burst, lymphocyte subsets) carried out by flow cytometry which was applied to a cat in the clinical setting[5,27].

CASE HISTORY

A 13-month-old, 5.3 kg Sphinx, castrated male cat was referred to the Veterinary Medical Teaching Hospital, University of Florida, for evaluation of chronic sneezing and bilateral purulent nasal discharge. The present owner acquired the cat from the breeder 6 weeks previously: signs of sneezing and nasal discharge were already evident at that time. The referring veterinarian had prescribed courses of amoxicillin (Amoxi-Drops, Pfizer, New York) (22 mg/kg per os (PO) q 12 h), enrofloxacin (Baytril, Bayer, Shawnee Mission, Kansas) (4 mg/kg PO q 12 h) and amoxicillin-clavulanic acid (Clavamox, Pfizer, New York) (12 mg/kg PO q 12 h). Clinical signs resolved for the duration of treatment, but returned as soon as treatment was discontinued. The cat had last received antibiotics 3 days prior to presentation. Immunisation history was not available, but the cat was believed to have been immunised against feline herpesvirus-1 (FHV1), feline calicivirus (FCV) and feline panleukopaenia at age 9 weeks and again at age 12 weeks. The cat had tested negative for feline leukaemia virus and feline immunodeficiency virus as a kitten, and again 2 weeks prior to referral. No other previous or concurrent health problems were reported and the cat had a normal appetite and activity level.

Abnormalities found on physical examination were limited to the eyes and upper respiratory tract. The left eye had hyperaemic conjunctivae and clear watery discharge. A yellowish, clear to mucopurulent discharge was evident from the right nares. Respiratory sounds were mildly stertorous. Body temperature was 37.6 °C. Blood glucose, packed-cell volume, and total solids were within normal reference ranges.

Bilateral turbinate damage was identified on radiographic examination of the skull. Following induction of anaesthesia for rhinoscopy, samples for fungal and aerobic bacterial culture were obtained by aspirating the right nares using a sterile 22G intravenous nylon catheter. Rhinoscopy revealed bilateral hyperplastic nasal tissue, a large amount of purulent nasal discharge within the nasal cavities, and a hyperaemic oropharynx. Biopsies of nasal tissue were collected for histological evaluation. While results were pending, the cat was discharged with a 14-day course of amoxicillin-clavulanic acid (Clavamox, Pfizer, New York) (12 mg/kg PO q 12 h).

Histology of the nasal mucosa was interpreted as chronic, active, and moderate to severe, suppurative rhinitis. Fungal culture was negative. Initial aerobic culture at 24 hours using regular media (Columbia Agar 5% Sheep's blood) yielded small pinpoint-sized colonies which were visible only by dissecting microscope. Gram's staining identified

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Received: July 2003. Accepted: March 2004.

them as Gram-negative cocci-bacilli, which increased our suspicion of *Haemophilus* species. The colonies where then transferred to chocolate agar. Culture on chocolate agar media was positive for pure culture of a *Haemophilus* species (see Table 1 for a description of the bacterial isolation). Further identification was deemed necessary and the isolate was submitted to a human laboratory for species identification. Using RapID™ NH System (Remel (Apogent), Lenexa, Kansas) the isolate was identified as *Haemophilus segnis,* a human pathogen[4,17] (see Table 1 for details).

Growth of a single organism was thought to represent significant colonisation, as opposed to the mixed flora typically isolated in cases of chronic rhinitis. The isolate was found to be susceptible to a variety of antimicrobials, including amoxicillin-clavulanic acid (Clavamox, Pfizer, New York) and enrofloxacin (Baytril, Bayer, Shawnee Mission, Kansas) which the cat had previously received.

Upon re-evaluation 2 weeks later, the owner reported the cat was still sneezing, but that the nasal discharge had decreased somewhat and was no longer purulent. Ocular discharge was still present from the left eye. No other clinical signs were reported, and the cat continued to have a normal appetite and activity level.

Physical examination was unremarkable except for stertorous breathing sounds attributed to obstructed nasal passages and watery ocular discharge from the left eye. Schirmer tear tests were within reference ranges for each eye. Fluorescein staining of each eye was negative for corneal ulcer or erosion, but the left nasolacrimal duct was not observed to drain properly.

Because of the unusual culture results obtained from the 1st visit, repeat cultures were attempted. A conjunctival swab grew very scant growth of *Corynebacterium* species. Culture from a retropharyngeal swab identified predominately *Mycoplasma* sp. and *Pasteurella* sp. No *Haemophilus* species were isolated. Virus isolation tests utilising kidney cell cultures were negative.

Because of the persistence of clinical signs in this patient, and the previous isolation of an unusual bacterium, tests were conducted to evaluate immune function. Tests were selected on the basis of availability and included a complete blood count and differential quantitation of serum immunoglobulins, immunophenotyping of lymphocyte populations by flow cytometry, and quantitation of neutrophil phagocytosis and induced oxidative burst. The white cell count and differential were within normal range. Serum concentrations of IgA, IgM, and IgG were normal and are reported in Table 2. Flow cytometry of lymphocyte subsets are reported in Table 3. The numbers of T and B lymphocytes, and the number of CD4+ and CD8+ T lymphocytes were normal; the expansion of B cells relative to T cells was interpreted as being consistent with antigenic stimulation.

Neutrophil phagocytosis and oxidative burst responses to the *Haemophilus segnis*-like isolate were compared to those for *Staphylococcus aureus* using a modified dual-colour flow cytometry assay[6,13] . The neutrophil responses to both bacteria were within the normal range reported for cats[6,13] (Table 4).

On reviewing the literature on *Haemophilus* species in cats[14,19], it became evident that the isolate was unlikely to be *H. segnis*

Table 1: **Bacteriological morphology and biochemical tests run from the clinical cat and a known *Haemophilus felis* isolate.**

Morphological description	Primary isolate	*H. felis*
Growth on chocolate agar	Yes	Yes
Colony colour	Grey	Yellow
Gram stain	G(–) cocci-bacillus	G(–) cocci-bacillus
Catalase	Positive	Positive
Attachment to media	Adherent	Adherent
Oxidase	Positive (weakly)	Negative
5 % Carbon dioxide	Yes	Yes
RapID™ NH System Reagents	Primary Isolate	*Hemophilus felis*
Proline p-nitroanilde	Negative	Negative
Gamma-glutamyl p-nitroanilide	Negative	Negative
o-Nitrophenyl, beta, D-galactoside	Negative	Negative
Glucose	Negative	Negative
Sucrose	Negative	Negative
Fatty Acid Ester	Negative	Negative
Resazurin	Negative	Negative
p-Nitrophenyl phosphate	Negative	**Positive**
Ornithine	Negative	Negative
Urea	Negative	Negative
Reduction of nitrate to nitrite	**Positive**	**Positive**
Tryptophane or indole production	Negative	Negative
Identified as	*Haemophilus segnis*	*Haemophilus ducreyi*

Table 2: **Quantification of serum immunoglobulins**[a] (units in mg/dℓ serum)**.**

Parameter	Patient	Reference range
IgA	200	25–200
IgM	260	75–260
IgG	1000	530–2000

[a]Cat IgG, IgA, IgM VET-RID kit, Catalog Number R40-103, Bethyl Laboratories, Montgomery, Texas. Tests performed by Clinical Pathology, College of Veterinary Medicine, Cornell University, Ithaca, New York.

Table 3: **Immunophenotyping of lymphocyte populations by flow cytometry**[a]**.**

Parameter	Units	Patient	Reference range
T lymphocytes	Cells/μℓ	1102	465–3555
Percentage		17	23–70
B lymphocytes	Cells/μℓ	3984	308–2856
Percentage		62	11–49
T: B ratio		0.28	0.7–4.6
CD4 T lymphs	Cells/μℓ	834	330–2448
Percentage		13	16-51
CD8 T lymphs	Cells/0μℓ	269	109–965
Percentage		4	5–18
CD4:CD8 ratio		3.1	1.5–5.6

[a]Comparative Clinical Immunology Laboratory, College of Veterinary Medicine, University of Florida, Gainesville, Forida. Antibodies used to detect lymphocyte subsets; mouse anti-feline CD4 (clone number vpg 34); mouse anti-feline CD8 alpha/beta (clone number vpg 9); mouse anti-feline CD5 (clone number FE1.1B11); mouse anti-canine B cells (clone number CA2.1D6). Antibodies were purchased from Serotec, Raleigh, North Carolina.

Table 4: **Flow cytometric quantitation of neutrophil phagocytosis and induced oxidative burst[a].**

Parameter	Patient	Reference range
% Neutrophils with PMA-induced oxidative burst[b]	78	59–79
% Neutrophils that phagocytosed *Staphylococcus aureus*	66	48–96
% Neutrophils with *Staphylococcus aureus*-induced oxidative burst	54	36–79
% Neutrophils that phagocytosed *Haemophilus segnis*	63	
% Neutrophils with *Haemophilus segnis*-induced oxidative burst	59	

[a]Comparative Clinical Immunology Laboratory, College of Veterinary Medicine, University of Florida, Gainesville, Forida.
[b]PMA = phorbol myristate acetate; PMA stimulates oxidative burst independent of phagocytosis: this is a measurement of the maximal oxidative burst capacity of the neutrophil.

but rather *H. felis* as *Haemophilus* species are known to be host-specific[15]. Unfortunately the isolate at this stage was lost and no further identification tests could be done. In an effort to compare results with a known isolate, a pure lyophilised isolate of *H. felis* was obtained (T J Inzana, Department of Pathobiology, Virginia-Maryland Regional College of Veterinary Medicine, Blacksburg, Virginia) and cultured. The pared isolates were then sent for identification using the same RapID™ NH System (Remel (Apogent)) as the original isolate. Results of the culture and identification by RapID™ NH System are reported in Table 1. Interestingly, the *H. felis* isolate was identified as *H. ducreyi* and not *H. segnis.*

DISCUSSION

Feline upper respiratory tract disease has been amply reviewed in the veterinary literature[1,7,10,11,18,25] . Chronic rhinitis has been defined as inflammation of the nasal passages that has been present for at least 4 weeks and typically presents clinically as sneezing and bilateral nasal discharge[1,3]. Differential diagnoses may include allergic, infectious and inflammatory causes, as well as nasal foreign bodies, neoplasms and advanced dental disease[1,3,25].

The most common infectious causes of rhinitis in the cat are FHV-1 or FCV[7,10,11,24,25]. Viral infections may produce both acute and chronic disease. Persistent or recurrent signs of rhinitis may result either from permanent viral-induced damage to the protective mucosal barriers that leads to impaired local immunity and repeated bacterial colonisation, or from reactivation of latent viral infection[18,24]. Diagnosis of viral infection is complicated by the ubiquity of these viruses: most cats are seropositive from prior infection or routine immunisation, and as many as 80–90% of these cats become carriers[10,18,25.] However, latently infected cats, even when exhibiting signs of upper respiratory tract disease, may shed few virus particles, and may only shed intermittently[24].

Primary bacterial infection is considered uncommon. Rhinitis may be seen, but infection with *Chlamydophila felis* should be considered unlikely in cats without conjunctivitis[20]. *Bordetella bronchiseptica* and *Mycoplasma* species can be isolated from healthy cats, but are occasionally implicated as primary agents in disease of the upper and lower airways[11,23]. Chronic rhinitis, regardless of underlying cause, is commonly complicated by secondary bacterial infection. *Pasteurella, Streptococcus,* coliforms, *Staphylococcus* and *Pseudomonas* species are considered normal flora of the feline nasopharynx and are typically isolated from cats with secondary bacterial infections[1]. Only 2 reports exist in the literature where *H. felis* was isolated from clinical cases. Inzana *et al.*[14] reported isolating *H. felis* from the nasopharynx in 6 of 28 normal laboratory cats and a clinical case with respiratory disease. Olsson and Falsen[19] reported isolating *H. felis* from 5 young cats with signs of conjunctivitis and upper respiratory tract infections. The general characteristics of the genus *Haemophilus* are small to medium-sized (0.5–1.0 μm), non-motile Gram-negative coccobacilli and following exposure to antibiotics threads or filaments may form[2,14]. Some species require carbon dioxide, especially during primary isolation and are capable of fermenting sugars. Importantly, owing to their fastidious nature, most species of *Haemophilus* will not grow on regular aerobic laboratory media (Columbia Agar 5% Sheep's blood, MacConkey Agar, and Columbia Agar CAN 5% Sheep's blood) used for most bacterial isolation but require X factor (hemin), or V factor (nicotinamide adenine dinucleotide), or both. The media of choice is chocolate agar. *Haemophilus* are confined to the mucosal surfaces of animals and humans and are host-specific. The host-specificity is due to the absolute requirement of iron only from iron-bound proteins of their natural host. Since *Haemophilus* species are species-specific, it seems unlikely that the specimen we isolated was the human pathogen *H. segnis.* Based on RapID™ NH system and morphology it was not possible to prove that the isolate was the same as *H. felis.* The colour of the isolate (grey versus yellow) and the oxidase-positive nature of the isolate in addition to the negative p-nitrophenyl phosphate results indicate that it may a different species. Precise identification would require sequencing of the bacteria's 16S rRNA gene which was not done.

In an informal (telephone) survey of diagnostic laboratories in southeastern USA, none reported using chocolate agar plates for routine aerobic culture of respiratory samples, and therefore it is likely that *Haemophilus felis* infections will be overlooked in cats. When not grown on chocolate agar, *Haemophilus* species can form satellite colonies close to *Staphylococcus* species because of growth factors supplied by the bacteria to the *Haemophilus* species 2. Since no veterinary pathogens are included in the RapID™ NH Systems or any other rapid culturing system database, animal pathogens will either be erroneously identified as was our case or yield a unique biocode not listed[21,22]. Many cats with chronic rhinitis, for which a specific underlying cause cannot be identified, or where chronic or recrudescent viral infection is suspected, require frequent, longterm courses of antibiotics to palliate clinical signs[9,25]. The strain isolated in our feline patient was susceptible to a variety of commonly used broad-spectrum antimicrobials but resistant to ampicillin and penicillin as has been reported for strains of *H. felis*[14,19].

Uncommon infections may be variously attributed to laboratory error, unusual exposure, or to compromised immune function. Growth of a solitary organism makes contamination of the culture improbable. In retrospect, obtaining samples from people or pets in the household might have contributed important information as to where the cat acquired the organism. Although neither FHV1 nor FCV were isolated, because the prevalence of virus is much lower in cats with chronic disease, a negative result did not rule out the possibility of latent infection. Thus, chronic or latent viral infection, especially with FHV1, remained likely. Given the history of chronic signs and the isolation of an unusual pathogen, immune dysfunction emerged as a possible

mitigating factor.

Immunosuppression in cats can be a consequence of debilitating and systemic diseases, and is associated with feline leukaemia virus and feline immunodeficiency virus infections[12]. Specific immune deficiency syndromes can be grouped in the following categories: congenital disorders, failure of passive transfer of maternal antibodies, acquired neutrophil dysfunction or neutropaenia, and disorders of lymphocytes. As in the present case, immunosuppression may be suspected when infections are recurrent or persistent, or when an unusual pathogen is isolated[12]. Immune function tests may include the following: evaluation of leukocyte number, morphology (complete blood count, differential and bone marrow cytology); evaluation of neutrophil function (chemotaxis, phagocytosis, oxidative burst and killing), evaluation of lymphocytes (quantitation of immunoglobulins, lymphocyte blast transformation, and flow cytometry of lymphocyte subsets) and evaluation for retroviral infection (feline leukaemia virus antigen test and feline immunodeficiency virus antibody test).

In the case reported here, normal total leukocyte count and normal distribution of neutrophils and lymphocytes ruled out a deficiency in the number of circulating immune cells, and examination of the cells excluded an inherited anomaly of leukocyte morphology. Quantitation of serum immunoglobulins established normal humoral immunity and circumstantial evidence for normal B lymphocyte numbers and function. B cells are most important for extracellular infections[12]. Flow cytometry was used to quantify lymphocyte subset populations: B cells, T cells, CD4 T cells (T-helper cells) and CD8 T cells (T-cytotoxic cells). Activated T-helper cells are key to generating both humoral and cell-mediated immune responses[8]. The major function of cytotoxic T cells is the killing of cells that express nonself antigens such as tumour cells and virus-infected cells[8]. Although not a comprehensive examination of the innate immune system, testing of phagocytosis and oxidative burst contributed to establishing normal function of this important arm of the immune response.

Results of retroviral and immune function testing were negative. With appropriate levels of serum immunoglobulins, B and T lymphocytes, and normal neutrophil function, the hypothesis of an immune abnormality appeared unlikely. It was not, however, able to measure secretory IgA to assess the strength or specificity of mucosal immunity. In addition, although neutrophilic phagocytic and oxidative burst functions were normal, actual killing of *Haemophilus* species was not quantified. It may be that disruption of the physical barriers of the nasal mucosa due to prior viral respiratory disease represented the most significant impairment of the immune response in this patient. Interestingly, the ability of some *Haemophilus* species to cause disease is related to its ability to cleave IgA1 (via IgA1-protease), rendering it non-functional[16]. While not reported for *Haemophilus* animal pathogens, it may be important in understanding the colonisation and pathogenesis of *Haemophilus* species on animal mucosal surfaces.

CONCLUSION

Cats with chronic or recurrent signs of rhinitis are often presumed to have bacterial infections secondary to previous infection with FHV-1 or FCV. Viral-induced cytopathic damage to the nasal mucosa, and host reaction to latent viral antigen, are thought to be factors involved in weakening host defenses against opportunistic pathogens. While it may be speculated that cats with latent viral infections suffer from some degree of immunosuppression, comprehensive immune profiling has not been performed.

The unusual bacterial isolate identified in this cat is important since culture technique employed in veterinary diagnostic laboratories for samples from companion animals generally do not include chocolate agar media. It is therefore likely that *Haemophilus* species are not going to be isolated. However, should a *Haemophilus* species be recovered, human rapid identification systems do not have animal pathogens listed on their database and give erroneous results.

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