Bartonellosis

Introduction

*Bartonella* spp. cause cat scratch disease (CSD) and other clinical syndromes in human beings, and are an important cause of endocarditis in dogs. On the other hand, there is scant documented scientific evidence that *Bartonella* infection causes overt clinical disease in naturally infected cats, in spite of a high prevalence of bacteremia and seropositivity in areas of the United States with warm temperatures and high humidity.

Microbiology

*Bartonella* spp. are facultatively intracellular gram-negative rods that are related closely to *Brucella* spp and the rickettsiae. Their intra-erythrocytic location precludes easy blood culture and a reliable response to antimicrobial therapy.

Four species of *Bartonella* have been shown to infect pet cats. *B. henselae* infection is most common, and is the most important cause of CSD. *B. clarridgeiae* may be responsible for a small number of cases of CSD. Rare infections of cats with *B. koehlerae* and *B. bovis* also have been reported. Two main genotypes of *Bartonella henselae* have been identified worldwide – Houston and Marseille. A third genotype, Berlin, has only been identified from one cat in Germany. Exotic cats have also been found to carry *Bartonella* sp. including: mountain lions, cheetahs, African lions, Florida panthers, pumas, and bobcats.

Transmission

*Bartonella* spp. are transmitted between cats by *Ctenocephalides felis* – the cat flea. Fleas ingest the organism during a blood meal from a bacteremic cat, and infect a naïve cat through regurgitation of infected saliva during a subsequent blood meal. Ticks may transmit the organism rarely between cats, and are the primary mode of transmission of *Bartonella* spp. between dogs. The organisms are not transmitted between cats by fighting, grooming, mating, or in-utero.

Human beings become infected with *Bartonella* spp. when flea feces from a bacteremic cat are inoculated into a cat scratch. Although not confirmed, rarely, infection may possibly be acquired directly through the bite of an infected flea.

Cats at Risk

Although *Bartonella* infections in cats have only been reported in the modern veterinary literature since 1992, the organism has apparently been infecting and adapting to cats for hundreds of years. A recent paper reports the isolation of *Bartonella* antigen from dental pulp by PCR assay in 800 year-old cat teeth from France. The prevalence of bacteremia and seropositivity in cats in the United States is highest in regions that favor the reproduction and persistence of fleas. Rates are highest in the southeastern United States (up to 40%), and lowest in the northern tier of states. The incidence in the EU, UK, and other countries also mirrors this pattern with higher seroprevalence rates in warm, moist locales, and much lower rates in colder climes.

Bacteremia is more likely to occur in cats with fleas, free-roaming cats, young cats, and those from multiple-cat populations.
Pathogenesis

After experimental inoculation into Bartonella-naïve cats, the organisms infect erythrocytes and the initial bacteremia lasts 2 to 32 weeks. After this, infected cats undergo bouts of cyclic bacteremia. Between bacteremic phases, when blood cultures (and blood PCR tests) are negative, the organisms may persist in endothelial cells, lymph nodes, or the central nervous system.

Following initial production of anti-Bartonella IgM antibody, IgG antibody is produced and remains detectable for months to years. There is no evidence that the height of the IgG antibody titer correlates with the presence of bacteremia – this observation is important when considering the diagnostic utility of antibody titers.

Clinical Signs – Experimental Infections

Part of the confusion about the clinical importance of Bartonella infections in cats arises from the observation that clinical signs are more likely to occur in cats after experimental inoculation that in naturally infected cats. Inappropriate extrapolation of data from experimental studies is one of many factors that have led to an overdiagnosis of clinical bartonellosis in the general cat population.

Following experimental inoculation, some infected cats have developed an inflammatory lesion at the injection site, mild generalized lymphadenopathy, splenomegaly, fever, lethargy, anorexia, myalgia, behavioral or neurologic changes, and reproductive abnormalities. In a number of other studies, no clinical signs were seen following infection. This probably relates to variable pathogenicity among the strains of B. henselae used in these studies.

Clinical Signs – Natural Infections

With rare exception, Bartonella spp. cause prolonged asymptomatic infections in naturally infected cats. Well-documented clinical signs arising directly from infection are very unusual and mostly anecdotal.

Bartonella spp. have been linked directly with endocarditis in one cat. Based on anecdotal reports, the organism may be a rare cause of lymphoplasmacytic gingivostomatitis (LPG) and uveitis. Clinicians should remember that feline calicivirus and plaque intolerance are very common causes of chronic LPG, and that many affected cats coincidentally will be Bartonella-seropositive given the high prevalence of infection with the organism.

A similar situation arises with uveitis, which usually is caused by viral or fungal infections. Because uveitis often is accompanied by intra-ocular bleeding, the isolation of Bartonella organisms or antibody from within the eye does not confirm infection. The diagnosis of Bartonella-induced uveitis is supported by the exclusion of all other more common causes, the demonstration of higher antibody levels in the aqueous humor than in serum, and a specific response to selective antimicrobial treatment.

Recent studies by Dr. Mike Lappin and the infectious disease group at Colorado State University have shown no statistical differences in Bartonella seropositivity between cats with and without uveitis, oral cavity disease, and central nervous system disease (ACVIM 2005).
**Bartonella Infections in Human Beings**

The CDC estimates that there are 24,000 cases of CSD/year in the U.S. This is an incidence of 9.3/10,000 ambulatory patients/year. The seropositivity rate for *Bartonella* in humans is between 3.6% to 15%; with the latter value occurring in a survey of veterinary professionals. Most persons inoculated accidentally with infected fleas feces through a cat scratch probably show no clinical signs or suffer from a vague, mild self-resolving, flu-like illness that does not prompt a visit to the physician. On the other hand, some immunocompetent people will develop typical CSD, with or without systemic complications. Persons with impaired immune systems are at risk for more severe complications of infection.

Typical signs of CSD include the development of a pustule (primary inoculation lesion) in the infected scratch within 7 to 10 days of the injury. Regional lymphadenitis, usually non-painful, occurs within 1 to 3 weeks of the injury. Lymph node enlargement may persist for weeks to months. Antimicrobial treatment does not shorten the duration of disease reliably.

Atypical signs of CSD include Parinaud’s oculoglandular syndrome (associated with a primary inoculation lesion on the conjunctiva and regional lymphadenopathy following infection of the conjunctiva with flea feces from a bacteremic cat), relapsing bacteremias and fevers, encephalitis, endocarditis, hepatitis, pneumonia, and osteomyelitis.

Angioproliferative lesions are more common in immunocompromised persons and include cutaneous lesions of bacillary angiomatosis, and cystic hepatic lesions of bacillary peliosis. Systemic complications of zoonotic *Bartonella* infections are more likely to be severe in immunocompromised human patients. Paradoxically, the response of these patients to antimicrobial treatment is better than that of immunocompetent patients with typical CSD.

**Diagnosis**

Laboratory tests to detect or exclude infection with *Bartonella* spp in cats include the detection of anti-*Bartonella* antibodies through immunofluorescent antibody (IFA) and Western Blot (WB) tests, blood cultures, and the amplification of *Bartonella* DNA by polymerase chain reaction (PCR) tests. These tests are used to place cats into one of the following categories:

1. Healthy cats that are not infected with *Bartonella* spp., and therefore are safe companion animals for immunocompromised persons.
2. Healthy cats that currently are infected with *Bartonella* spp., or that have been infected previously with *Bartonella* spp.
3. Sick cats (for example, cats with uveitis or stomatitis) with concurrent *Bartonella* infection that is not the cause of their clinical illness.
4. Cats with *Bartonella*-induced illness (an unusual occurrence in clinical practice).

Because of the high prevalence of seropositivity to *Bartonella* in the general cat population and the low incidence of *Bartonella*-induced disease, the detection of serum antibodies has a poor predictive value (42 to 46 per cent) for the confirmation of disease caused by the organism. Similarly, using the IFA test, there is a poor correlation between
the height of the antibody titer and the ability to detect bacteremia. Because titers in infected cats vary greatly over time, increases in titers associated with vague clinical signs should be interpreted with caution.

Conversely, a negative IFA titer has a high negative predictive value (>90 per cent), making it a useful screening test to exclude bacteremia in an asymptomatic or symptomatic cat. A small number of cats may be seronegative between cycles of bacteremia, and blood cultures and PCR tests may be needed to confirm the status of these cats, especially if they are being considered as companion animals for immunocompromised persons.

With the possible exception of endocarditis, the clinical diseases attributed anecdotally to infection with *Bartonella* spp. usually are caused by more common infections. Therefore, tests for these other diseases (for example, FeLV/FIV, toxoplasmosis, cryptococcosis, histoplasmosis, and the aforementioned causes of stomatitis) should be performed and interpreted before tests for *Bartonella* infection are ordered.

Based on our present scientific knowledge of the epidemiology of *Bartonella* infections in cats and human beings, there are no valid indications for the routine testing and subsequent treatment of healthy pet cats that live with healthy owners. This recommendation is also that of the Centers for Disease Control and this information is available on their website: www.cdc.org

**Treatment**

Treatment should be reserved for that small group of sick cats with apparent *Bartonella*-induced disease, based on careful interpretation of serological and culture/PCR results and an exhaustive exclusion of other more common diseases. At the present time, there is no evidence that antimicrobial therapy eradicates *Bartonella* organisms completely from infected cats. Although the level of bacteremia may be reduced temporarily, recurrence of bacteremia usually occurs due to the intra-erythrocytic location of the organism.

Enrofloxacin and doxycycline have been used to reduce the level of bacteremia. Unfortunately, the recommended dose of enrofloxacin has a high risk of inducing retinotoxicity, precluding its safe use in cats. Azithromycin (5-10 mg/kg PO q24h for 5d, then q48h for 40d) has been recommended as more effective. Unfortunately, because bacteremia is cyclic, and because organisms are rarely cleared with antibiotic therapy, there is no good endpoint on which to base apparent success of treatment. PCR testing may be negative at some point, and then return to positive weeks to months later. Antibodies will persist for years, often at high levels even after organisms are gone.

**Prevention**

Prevention of CSD in human beings sharing a house with cats depends primarily on scrupulous and effective flea control. Even if the cat is bacteremic, human infection from cat scratches, etc. will not occur unless the injuries are contaminated with flea feces. Children, who are at most risk for the development of CSD if infected, should be taught to play gently with their pet cats, especially new kittens, to avoid scratches.

Cats being considered as companion animals for immunocompromised persons should be selected from a flea-free background if possible. The cats should be screened initially with an IFA test. If the test is positive, it would be wise to consider the cat no further as a safe companion. If the IFA test is negative, blood cultures and PCR tests
should be performed. If these latter tests are negative, the cat can be considered safe, as long as it is kept indoors exclusively, not exposed to cats with fleas, and is treated diligently with a year-round flea-control program.

References


Virulent Systemic Calicivirus –

Signs of this strain of calicivirus may include:

High fever
Facial and limb edema
Ulceration, crusting and focal hair loss, especially on the face, muzzle and pinnae
Icterus
Dyspnea, DIC and death in severe cases

Death may occur in some cats with minimal preceding signs
Findings on blood chemistry panel may include hyperbilirubinemia, hyperglucosemia and increased CK.

Other signs seen with more typical feline URI may also occur, including nasal and ocular discharge, oral ulceration, anorexia and depression. Unless accompanied by the signs described above, cats showing these typical URI signs should not be considered suspect cases.

Course of disease:
The incubation period is between 1-5 days. Cats of all ages, including fully vaccinated cats, have been affected. No other species is known to be affected by this strain of calicivirus. There is no known risk to human health. Treatment, as for any virus, is supportive care. It is likely that a significant percentage of cats will continue to shed virus for some
time after recovery from clinical signs, as occurs with other strains of feline calici virus. Therefore cats may still be infectious to others following apparent recovery. Confirmed cases should have negative viral cultures before being reintroduced to other cats.

**Transmission:**
Virus is present systemically, and may be shed in feces and in nasal, ocular and oral secretions. The virus can be readily spread by fomites as well as direct transmission. It can be carried for at least several hours on contaminated hands, clothing, instruments, shoes, etc. Droplet transmission is possible over 1-2 meters. Although calici virus may be carried through ventilation systems on dust and hair, airborne transmission over distances greater than a few feet has not been documented in this outbreak.

**Prevention:**
Calicivirus is moderately hardy in the environment, but bleach (5% diluted at 1:32) is effective as a disinfectant. Suspect cases should be housed in strict isolation, with separate equipment, gowns, gloves, caps, and protective footwear used. Possibly contaminated surfaces should be thoroughly cleaned with bleach. Contaminated exam rooms should be cleaned with bleach, held empty for 24 hours, and cleaned again with bleach prior to reuse. If contamination of a home or clinic is suspected, all surfaces should be thoroughly cleaned and disinfected. If surfaces can’t be bleached, the facility or home should be quarantined for 1-2 weeks following cleaning, prior to allowing entry of naïve cats. Heavily contaminated objects such as bedding should be discarded or thoroughly washed. Veterinary staff and others who handle sick cats should change clothing prior to handling healthy cats and at the end of a shift. There has been no further spread of disease documented in clinics that have taken these precautions.

**The VS-FCV vaccine is NOT recommended.** VS-FCV is NOT a disease of household pets. The virus arises anew in each population in which it appears as a mutation from caliciviruses already carried in that group of cats. There is no single genetic mutation that accounts for virulence. No two strains are alike. The vaccines is adjuvanted and may lead to the development of injection-site sarcoma due to chronic site inflammation.

**VS FCV suspects in a Private Practice setting from UC Davis:**

Due to increased vigilance nationwide in regards to VS-FCV we are unable to handle to the overwhelming volume of inquiries regarding suspect cases. If you believe that you may have a suspect case please first review our VS-FCV information page http://www.sheltermedicine.com/portal/is_vsfcv.shtml

Key points to remember:

1. Neither VS-FCV nor field strain FCV can be diagnosed on clinical signs alone.

2. Diagnosis of calicivirus is further complicated by the fact that calicivirus can be isolated from the oral cavity of as many as 1 in 4 healthy cats, so simply detecting the virus in saliva does not provide a definitive diagnosis - its presence could be completely
coincidental. Finding calicivirus in other samples such as serum or tissue is more suggestive that an acute infection is present, but does not rule out the possibility that a co-pathogen such as Bordetella bronchiseptica or panleukopenia is responsible for severe manifestations of disease.

3. So far, no relationship has been discovered between the genetic sequence of a particular strain of calicivirus and the level of virulence. Virulent systemic strains are not particularly closely related to one another, although within each outbreak isolates from individual cats have been similar. Specialized laboratories can only distinguish between strains that are closely related to one another or to the vaccine. Therefore, within a given outbreak it is possible to identify which cats have been infected with that particular strain, but there is no way to say what the virulence may be of any particular strain from an individual cat based on genetic sequence.

4. If you have a single suspect case in your clinic the cat should be carefully isolated and all surfaces should be cleaned with soap and water, followed by application of freshly made bleach solution diluted at 1/2 cup per gallon or potassium peroxymonosulfate (Trifectant®). The area should be allowed to dry thoroughly and the process repeated before any other animals are placed on the surface or in the cage. All suspect cases should be treated symptomatically, the clinic should treat all suspects as being potentially contagious and staff should increase bio-security vigilance.

5. At this point we are unable to provide diagnostic support to facilities reporting a single suspect case. All professional staff who have been in contact with this suspect cat should be advised to protect the health of currently housed cats in the clinic and the health of their personnel pets by thoroughly washing their hand and by changing clothes before coming into contact with subsequent animals.

6. We are interested in receiving tissue samples from suspect cats examined by private practitioners that meet ALL of the following criteria.

a. The particular case meeting the criteria as outlined at http://www.sheltermedicine.com/portal/is_vsfcv.shtml AND...

b. The client reports that other cats in their home have similar symptoms beyond what is generally described as Feline Upper Respiratory Disease and the client has brought these cases into your clinic for examination OR...

c. A single case occurred in your clinic AND subsequent to this cats visit, other suspect cases occur in other animals currently housed in your clinic, or who were in your clinic during the same time as the initial suspect case, or in staff members personnel pets. Or, the individual cat has come from a shelter or rescue group with other suspect cases that have been examined by a veterinarian and have been determined to be suspect VS-FCV cases.

7. VS-FCV has been ruled out in overwhelming majority of VS-FCV cases submitted to
In cases VS-FCV has been confirmed, thorough disinfection and strict isolation of suspect cases has been sufficient to end the outbreak.

If you suspect a case:
Email Dr. Kate Hurley at kfhurley@ucdavis.edu  You may also call our program coordinator Mike Bannasch at: 530 754-7355.

**Methicillin-Resistant Staphylococcus Aureus**

**INTRODUCTION**

MRSA can be of concern to veterinarians, their staff and veterinary patients. Additionally, newly emerging antibiotic-resistant infections may stimulate questions from clients. Here’s what we know about MRSA.

### CLINICAL USE INFORMATION

What is MRSA and where does it come from?
Is MRSA a concern for my patients?
Do all animals with MRSA get sick?
My client claims that their MRSA infection was transmitted from their pets. What do I tell them?
When should I consider screening pets?
Are my staff at risk?
Should I treat an animal that is a carrier of MRSA?
Are there other methicillin-resistant staphylococci? Are they similar to MRSA?
What is the significance of coagulase negative methicillin-resistant staphylococcal species (MRCoNS)?

**What is MRSA and where does it come from?**

MRSA is a *Staphylococcus aureus* that has acquired the *meca* gene, which encodes a protein (PBP2A) that reduces the binding affinity of penicillins and cephalosporins *(Weese 2005, Duquette & Nutall 2004, Weese 2005Proc)*. Therefore, MRSA strains are resistant to all penicillins and cephalosporins, and frequently also possess genes conferring resistance to a variety of other antimicrobials *(Rich et al 2005)*. While MRSA was first noted in people over 40 years ago, it has become a significant pathogen over the last 10-15 years, and is now considered a potential serious infectious threat, because of the limited antibiotic sensitivity it exhibits.

In most cases MRSA is hospital-associated, that is, it occurs in patients that have been hospitalized or had invasive procedures performed on them. More recently, "community-associated MRSA", or CA-MRSA, has been identified. This form of MRSA occurs in people who have no history of risk factors for MRSA. The CA-MRSA isolates tend to differ genetically from hospital-associated MRSA and many express a toxin called
**Panton-Valentine Leukocidin** (van Duijkeren et al 2005). In humans, CA-MRSA typically causes skin and soft tissue infections, but life-threatening necrotizing fasciitis (the "flesh-eating bacteria"), necrotizing pneumonia and sepsis can also develop. Common venues for cross-infection with CA-MRSA include sporting facilities (changing-rooms) and correctional facilities. In October 2007, the death of student athletes in the US sparked interest in MRSA.

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**Is MRSA a concern for my patients?**


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**Do all animals with MRSA get sick?**


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**My client claims that their MRSA infection was transmitted from their pets. What do I tell them? What should I do?**

Interspecies transmission of MRSA occurs. Humans can infect dogs or cats, and pets can infect their owners (Boost et al 2007, Cefai et al 1994, Cuny et al 2006, Enoch et al 2005, Huijsdens et al 2006, Juhász-Kaszanyitzky et al 2007, Leonard et al 2006, Loefller et al 2005, Manian 2003, O'Mahoney et al 2005, Strommenger et al 2006, van Duijkeren et al 2005, Weese et al 2006a,c). Thus, it is often difficult to determine which way the infection has been transmitted. Concern has been raised about dogs involved in hospital visitation programs, both in terms of an increased risk of developing MRSA infection and a risk of transmitting MRSA to hospitalized patients (Enoch et al 2005). To date, recommendations have been to avoid contact between visiting animals and patients infected with MRSA, or with patients who are immunocompromised or otherwise
susceptible (Enoch et al 2005). However, new recommendations are being developed and will be available by early 2008. These will hopefully better define the issue of visitation by therapy pets.

**When should I consider screening pets?**

Routine screening in isolated CA-MRSA infections is not advised. The Canadian Council on Antibiotic Resistance has produced comprehensive guidelines for screening and decolonization of humans. These apply equally to pets.

If a patient suffers recurrent CA-MRSA, or multiple people develop MRSA infections, a pet fomite should be considered. MRSA infection is identified by routine culture and susceptibility testing. It is important that diagnostic labs identify coagulase positive staphylococci to the species level and test for methicillin resistance. Testing for MRSA colonization involves submitting swabs for culture. At this point, it appears that paired nasal and rectal swabs are optimal. However, it is important to realize that colonization may not be restricted to these sites - thus a negative finding may not exclude a pet as a fomite (May et al 2005). The sample submission must state that MRSA culture is required. The use of selective screening techniques, such as inoculation onto mannitol-salt agar containing oxacillin, is preferred. Real time PCR has been shown in one study to have poor agreement with culture results in horses (Anderson & Weese 2007). More information regarding testing is available from the CDC website on MRSA.

**Are my staff at risk?**

Several studies have examined the prevalence of MRSA in veterinary hospitals in the United Kingdom (Loeffler et al 2005, O’Mahoney et al 2005, Hanselman et al 2007). These studies show that veterinary staff and their pets have a higher prevalence of MRSA, although they are asymptomatic carriers. Thus, veterinary staff may be at increased risk of carrying MRSA. Good hygienic practice should be encouraged to minimize acquisition of MRSA by veterinary staff. Containment strategies, involving surveillance, barrier precautions and hand hygiene, were successfully adopted to curtail an outbreak of MRSA in animal patients in an ICU (Weese et al 2007).

**Should I treat an animal that is a carrier of MRSA?**

Routine decolonization therapy is not recommended in humans or animals that have mucosae colonized with MRSA. There is currently no evidence that it is effective, and most (if not all) pets will clear MRSA colonization spontaneously if re-infection is prevented. Nasal mupirocin is not practical because nasal passages cannot be adequately covered. Additionally, intranasal therapy does not address GI or cutaneous colonization, which appears to be relatively common. Finally, these strains of Staph. aureus have profound antimicrobial resistance so it is irrational to increase their resistance profile. Chlorhexidine baths can be used but they do not address the primary colonization sites.

The key to minimizing colonization is stringent household infection control practices, in particular avoiding high risk contact and frequent hand hygiene. If re-infection is
Prevented, culturing colonized animals every few weeks generally demonstrates that they have eradicated the organism within a few weeks. In rare situations where MRSA infections are rampant in people in the household and the entire household is undergoing eradication therapy, kenneling the pet for a couple weeks is a reasonable option, to allow it to clear the colonization and avoid re-infection.

Decolonization therapy has been used by some investigators in the UK with reports that it typically takes a few weeks, which is longer than the human therapy response (and also essentially the same as the observed response in non-treated pets with spontaneous eradication).

Are there other methicillin-resistant staphylococci? Are they similar to MRSA?
Methicillin-Resistant Staphylococcus intermedius (MRSI) is being recognized more and more frequently (Kania et al 2004, Morris et al 2006, Abraham et al 2007, El Zubeir et al 2007). However, it behaves differently from MRSA - it preferentially colonizes dogs, rather than humans, and is much less likely to infect humans than MRSA. However, minimizing or avoiding contact between the canine patient and immunocompromised, geriatric and pediatric humans is prudent. MRSI does not appear to be as pathogenic as MRSA.

Canine patients with MRSI should be treated as out-patients - your hospital patient population is much more at risk for colonization or infection than owners of MRSI-infected patients.

If MRSI infections are diagnosed, preventive measures to limit nosocomial infections should be implemented: rigorous hand-washing, wearing new gloves for each patient exam, treating MRSI-infected dogs last etc. Most common disinfectants kill MRSI and MRSA in the environment.

Two other methicillin-resistant Staphylococci have been identified - methicillin-resistant *S. schleiferi* and methicillin-resistant *S. pseudintermedius* (Frank et al 2003, Kania et al 2004, May et al 2005, Morris et al 2006b, Abraham et al 2007, Hanselman et al 2007, Sasaki et al 2007). However, little is known about these species. *Staphylococcus schleiferi* appears to be commonly associated with canine pyoderma, and many strains appear to be methicillin-resistant (Frank et al 2003, Kania et al 2004, May et al 2005).

What is the significance of coagulase negative methicillin-resistant staphylococcal species (MRCO NS)?
Staphylococci are divided into two main groups; coagulase positive and coagulase negative. The coagulase positive species (i.e. *S. aureus*, *S. pseudintermedius*, *S. schleiferi* coagulans) are the most pathogenic, although they are common commensals as well. Coagulase negative staphylococci are very commonly found in healthy animals as commensal of the skin, respiratory tract and intestinal tract. They are considered to be minimally pathogenic, in general, but they can cause opportunistic infections, usually in compromised hosts. Some CoNS have acquired methicillin-resistance, and MRCO NS are
commonly found in healthy animals. In human hospitals, CoNS (and MRCoNS) are considered a common pathogen in catheter-related infections (van Pelt C et al. 2003).

In dogs, S. epidermidis is an example of a CoNS - it is a normal skin inhabitant but can cause infections in some situations. S.schleiferi schleiferi is another CoNS, and one that might be more pathogenic as a cause of skin and ear infections.

Recent veterinary studies of MRCoNS in Slovenia found a prevalence of 40% in horses and 11% in dogs. (Vengust M et al 2006) However, little is known about these organisms in veterinary medicine. One problem is determination of the relevance of MRCoNS, especially when they are isolated from skin or ears, or sites associated with those (i.e. intravenous catheter sites) because it could be the cause of infection or a skin contaminant. Isolation of CoNS, including MRCoNS, should be interpreted with caution from superficial sites. They should not be dismissed as they could be the cause of infection but it is hard to definitively diagnose CoNS infection from such sites. If another pathogen is isolated, the CoNS is typically regarded as a contaminant.

MRCoNS do not appear to be of significant zoonotic concern or pathogenic concern in most animals. Eradication of CoNS is not generally advised and is likely impossible because of their role as part of the commensal microflora.

Link to excellent Dr. Scott Weese’s excellent website:

http://www.wormsandgermsblog.com/articles/diseases/test-subcategory/

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