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Canine superficial bacterial folliculitis: Current understanding of its etiology, diagnosis and treatment

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**Abstract**

Superficial bacterial folliculitis (SBF) is more common in the dog than other mammalian species. Until recently, a successful outcome in cases of canine SBF was possible by administering a potentiated amoxicillin, a first generation cephalosporin or a potentiated sulfonamide. Unfortunately, this predictable susceptibility has changed, because methicillin resistant *Staphylococcus pseudintermedius* (MRSP) and *Staphylococcus aureus* (MRSA) are becoming more prevalent in canine SBF cases. The increasing frequency of multidrug resistance complicates the selection of antimicrobial therapy. Antimicrobial agents that were once rarely used in cases of canine SBF, such as amikacin, rifampicin and chloramphenicol, are becoming the drugs of choice, based on bacterial culture and susceptibility testing. Furthermore, changes in antimicrobial susceptibility have helped to re-emphasize the importance of a multimodal approach to treatment of the disease, including topical therapy. Due to the increasing frequency of identification of highly resistant *Staphylococcus* spp., topical antimicrobial therapy, including the use of diluted sodium hypochlorite (bleach), is becoming necessary to successfully treat some cases of canine SBF. Other important antiseptics that can be used include chlorhexidine, benzoyl peroxide, ethyl lactate, triclosan and boric acid/acetic acid. This review discusses the diagnostic and therapeutic management
of canine SBF, with a special emphasis on treating methicillin resistant staphylococcal infections.

*Keywords*: *Staphylococcus*; Pyoderma; Canine; Methicillin; Antimicrobial resistance

**Introduction**

Bacterial pyoderma is more common in the dog than other mammalian species. In contrast to *Staphylococcus aureus* infections in human beings, virulence factors, such as protein A, leukocidin, hemolysins and epidermolytic toxin, have not been shown to play a role in the pathogenesis of canine pyoderma. Numerous studies have failed to identify any differences in toxin profiles between *Staphylococcus* spp. from normal dogs and those from dogs with pyoderma (Allaker et al., 1991). Since *Staphylococcus pseudintermedius*, the most common organism that causes canine pyoderma, is a normal commensal of the dog, it appears that abnormal ‘host factors’ are the primary cause of these infections. The most common primary causes include hypersensitivities, ectoparasites, endocrinopathies and cornification abnormalities. Long term success in treating canine bacterial pyoderma requires identifying and treating the primary cause.

Bacterial pyoderma can be classified on the basis of the depth of the lesion(s). The different classifications are (1) surface pyoderma (pyotraumatic dermatitis, mucocutaneous pyoderma and skin fold dermatitis); (2) superficial bacterial folliculitis (SBF); and (3) deep pyoderma, namely, deep folliculitis and furunculosis, and cellulitis (subcutaneous involvement). This review article is focused on superficial bacterial infection of the hair.
follicle (folliculitis). It is beyond the scope of this article to completely review canine SBF and the objective is to provide an update on the disease in dogs, especially in regards to methicillin resistance.

Etiology

Historically, *Staphylococcus intermedius* had been the most commonly isolated pathogen in dogs with SBF (Cox et al., 1984; Medleau et al., 1986). More recently, microbiologists have shown that all the organisms identified in the past as *S. intermedius* were really *S. pseudintermedius* (Devriese et al., 2005). This has been modified further, such that there is now a *Staphylococcus intermedius* group (SIG) with members including *S. intermedius*, *S. pseudintermedius* and *S. delphini*.

*S. pseudintermedius* remains the organism most commonly causing SBF in dogs (Sasaki T et al., 2007). However, for clinicians, these name changes have no bearing on the medical management of the cases. What is important is to differentiate SIG from the bacterium that causes human infections, i.e. *S. aureus*. SIG can be differentiated from *S. aureus* based on a variety of different techniques, including phenotypic testing or molecular identification using whole-cell fingerprinting by matrix-assisted laser desorption-ionization (MALDI)-time-of-flight mass spectrometry (MS), biochemical features, DNA-DNA hybridization and PCR (Dubois et al., 2010; Sasaki et al., 2010).

*Staphylococcus schleiferi* has also been associated with bacterial pyoderma. *S. schleiferi* may be either coagulase positive (*S. schleiferi coagulans*) or coagulase negative (*S. schleiferi schleiferi*) (Frank et al., 2003). In the past, coagulase negative staphylococci were considered to be contaminants when found on a culture from a superficial lesion in a dog.
However, coagulase negative *S. schleiferi schleiferi* is a pathogen that is potentially zoonotic. For this reason, it is important that laboratories identify coagulase negative staphylococci down to the species level (for example, to differentiate non-pathogenic *S. epidermidis* from pathogenic *S. schleiferi schleiferi*).

**Clinical signs**

Pruritus in dogs with SBF ranges from non-existent to intense. Clinically, SBF presents differently in different breeds of dogs. Most dogs have multifocal areas of alopecia, follicular papules or pustules, epidermal collarettes and serous crusts involving the trunk, abdomen and axillary areas. Short-coated breeds often present with a ‘moth-eaten’ appearance to the hair coat due to alopecic lesions associated with the folliculitis. Cocker spaniels have their own special presentation, i.e. vegetative plaques, which are frequently mistaken for seborrheic plaques associated with primary seborrhea of Cocker spaniels. Clinically and histopathologically, they can be quite similar; therefore, if plaques are found on a Cocker spaniel, the dog should be treated for a bacterial pyoderma before assuming that the dog has ‘idiopathic seborrhea’. The diagnosis of SBF is usually based on physical signs (i.e. multifocal areas of alopecia, papules, pustules and epidermal collarettes), supported by cytology and/or bacterial culture.

**Diagnosis**

Investigation of the underlying cause of the disease should be performed because primary canine bacterial pyoderma does not occur. When a dog is presented for the first time with SBF, only a limited number of diagnostic tests need to be undertaken. However, with recurrent or chronic cases of SBF, or with any dog with a deep bacterial pyoderma, there is a need for the underlying cause to be pursued aggressively.
The predisposing causes of SBF include: (1) hypersensitivities (atopy, cutaneous adverse food reactions, flea allergy dermatitis); (2) ectoparasites (e.g. Sarcoptes spp.); (3) endogenous (hyperadrenocorticism) or exogenous exposure to corticosteroids; (4) demodicosis (Demodex spp.); (5) hypothyroidism; (6) follicular dysplasias (e.g. color dilution alopecia); (7) ectodermal dysplasia (e.g. Chinese crested dogs); and (8) cornification abnormalities (sebaceous adenitis, ichthyosis) (Mason et al., 1989, Chesney, 2002).

**Cutaneous cytological examination**

Cutaneous cytology is an easy, inexpensive and rapid diagnostic test that should be performed on any dog that is presented with skin lesions. There are a variety of methods to collect cytology specimens (Mueller, 2000), with each method having advantages and disadvantages. Cytology is used to identify the presence (and/or type) of: (1) bacterial or fungal organisms (e.g. Malassezia); (2) neoplastic cells; (3) inflammatory cells; and (4) abnormal cells (e.g. acantholytic keratinocytes associated with pemphigus foliaceus). When evaluating cutaneous cytology specimens for infection, a semiquantitative scale ranging from 0 to 4+ should be used (Budach et al., 2012).

**Bacterial culture**

Bacterial culture may be necessary when managing a case of SBF. Before culturing a lesion, cutaneous cytology should be performed on a representative lesion to confirm the presence of bacterial infection (neutrophils with intracellular bacteria). Bacterial culture and susceptibility (c/s) testing should always be performed in poorly responsive SBF cases, but is not necessary in antimicrobial responsive but recurrent cases, since these cases will mostly benefit from identifying and treating the primary cause only. Since Gram negative, rod shaped
bacteria isolated from the skin are frequently resistant to many antibacterial agents (Petersen et al., 2002), a bacterial culture should be submitted if neutrophils and rod-shaped bacteria are identified on cutaneous cytology.

If a culture and susceptibility test is submitted, the minimum inhibitory concentration (MIC) broth microdilution technique rather than the disc diffusion method should be used to determine susceptibility. The disk-diffusion susceptibility test (DDST) is semiquantitative in that the drug concentration achieved in the agar surrounding the disc can be roughly correlated with the concentration achieved in the dog’s serum. It will only report the organism’s susceptibility (susceptible, intermediate or resistant, SIR) based on an approximation of the effect of an antimicrobial agent on bacterial growth on a solid medium. Tube dilution (MIC) is quantitative, not only reporting SIR, but also the amount of drug necessary to inhibit microbial growth. The MIC is reported as the lowest concentration of an antimicrobial agent (in µg/mL) necessary to inhibit visible growth of the tested bacteria. This allows a clinician to decide, not only susceptibility or resistance, but also the proper dosage and frequency of administration of the antimicrobial agent. The MIC method can imply the relative risk of emerging resistance and thus the need for a high treatment dose.

Samples from a pustule or intact nodule should be used for culturing, but if an intact pustule or papule is not available, culturing an epidermal collarette has also been shown to be reliable for sampling for SBF (White et al., 2005). Submitting a crust is also useful for a dog with SBF if any of the classical lesions are not present; culture results from the crust are the same as those from a macerated tissue punch biopsy sample (Vaughan and Lemarie, 2008).
**Systemic treatment**

Recently, successful treatment of SBF could be accomplished predictably with a β-lactam antibiotic (a first generation cephalosporin, such as cephalaxin, or a potentiated amoxicillin). However, increasingly methicillin resistant *Staphylococcus* spp. (MRS) are being identified as causes of skin infections in dogs. MRS may be *S. aureus* (MRSA), *S. pseudintermedius* (MRSP), *S. intermedius* (MRSI) or *S. schleiferi* (MRSS). No member of the β-lactam family of antibiotics will be effective when MRS is identified.

The incidence of MRSP has been increasing over the last decade, rendering many commonly used antibacterial agents ineffective (Jones et al., 2007). An additional complication is that these bacteria are frequently multi-drug resistant (MDR). In a recent study, >90% of MRSP were MDR, defined as being resistant to ≥4 antimicrobial drug classes (Bemis et al., 2009).

The cause of the increased frequency of MRSP has not been clearly established, but one of the many risk factors for MRSA and MDR *Staphylococcus* spp. is the administration of fluoroquinolones. Reducing the use of antimicrobial agents and, particularly, fluoroquinolones and third generation cephalosporins, may help to prevent persistent carriage of MRSA in human beings (Monnet et al., 1998; Muto et al., 2003). Fukatsu et al. (1997) reported MRSA outbreaks in human beings in a hospital that were correlated with the overuse of third generation cephalosporins for prolonged periods; the incidence of these outbreaks decreased by restricting the use of these antibiotics.
In the past, oxacillin was used in antimicrobial sensitivity testing panels to identify MRS. If the *Staphylococcus* sp. was an MRS, then it would be resistant to all of the β-lactams (Weese et al., 2010). Currently, the protocol for identifying MRSA in vitro is to use cefoxitin as the surrogate, since certain strains of MRSP may be falsely identified as methicillin susceptible if the laboratory uses cefoxitin susceptibility as the indicator (it is unknown if this applies to other members of the SIG) (Schissler et al., 2009; Weese et al., 2009). In order to avoid mislabeling MRSP as susceptible, a new lower breakpoint (reduced from 2.0 to 0.5 g/mL for oxacillin when testing *S. pseudintermedius*) should be used. If culture and sensitivity testing is performed by a laboratory more familiar with human specimens, the laboratory may not be aware of these important differences and changes. To avoid these errors, a veterinary laboratory that uses Clinical and Laboratory Standards Institute (CLSI)\(^1\) or the European Committee on Antimicrobial Susceptibility Testing (EUCAST) guidelines\(^2\) should perform the tests.

Recently the effectiveness of clindamycin against MRSA has been questioned (Rich et al., 2005). Two genes (*msrA* and *erm*) are responsible for the resistance of *S. aureus* to macrolides (e.g. erythromycin). The *msrA* gene accounts for the resistance only to macrolides, while the *erm* gene encodes for macrolide and lincosamide (lincomycin and clindamycin) resistance. The *erm* gene can be expressed constitutively and the culture will report resistance to clindamycin; this gene can also be inducible, in which case the MRSA will be susceptible initially to clindamycin (and therefore reported as sensitive).

When MRSA has the inducible gene, resistance to clindamycin will develop during treatment. Since the pattern of susceptibility to clindamycin of MRSA isolates possessing the

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msrA gene (truly susceptible to clindamycin) or the inducible erm gene (inducibly resistant) are the same, it is important to distinguish between these phenotypes. This is accomplished by an additional culture technique, the double disc diffusion D-test, which will detect the occurrence of the inducible erm gene. Since commercial veterinary laboratories currently are not performing this additional culture, resistance to erythromycin may be used as a surrogate to indicate the presence of this inducible gene. This is because the msrA gene and the erm gene both encode staphylococcal resistance to erythromycin. Therefore, if the Staphylococcus sp. is resistant to erythromycin, there is a potential for the inducible erm gene to be present.

Rich et al. (2005) found that that 97.3% of erythromycin-resistant isolates of MRSA were truly resistant to clindamycin, even though only 25.5% demonstrated clindamycin resistance on routine laboratory testing. On the basis of this study, it would be prudent to avoid clindamycin for all S. aureus infections that report resistance to erythromycin. Concern about this inducible gene being present in MRSP was investigated in a recent report by Faires et al. (2009), in which inducible clindamycin-resistance was present only in MRSA isolates and not, for example, in MRSP. The authors concluded that, since inducible resistance was not identified in any of the MRSP, the use of clindamycin was a reasonable option for MRSP infections. Unfortunately, subsequent work questioned this conclusion, since the inducible clindamycin gene was identified in two strains of MRSP (Perreten et al., 2010). An additional report also found this inducible gene in methicillin susceptible Staphylococcus spp. (MSSP) and methicillin susceptible S. aureus (MSSA) (Rubin et al., 2011). For this reason, it is recommended that clindamycin should be avoided in any Staphylococcus spp. infection, regardless of the species and strain, if the organism is reported to be resistant to erythromycin.
Resistance of *Staphylococcus* spp. to tetracycline is mediated by two genes, *tet(K)* and *tet(M)* (Trzcinski et al., 2000; Schwartz et al., 2009). Resistance to tetracycline, but not doxycycline or minocycline, is mediated by *tet(K)*, while *tet(M)* will confer resistance to all three members of the tetracycline family. However, it is different in the case of MRSA. If an MRSA isolate has the *tet(K)* gene, exposure to either tetracycline or doxycycline can induce doxycycline, but not minocycline, resistance. It is currently unknown whether this is true for members of SIG that are methicillin resistant. This is a problem, because the results of culture will have suggested that the *Staphylococcus* sp. isolate is susceptible to doxycycline, but in vivo resistance will occur. If the isolate is MSSA the *tet(K)* gene will confer resistance only to tetracycline. In cases of MRSA infection the *tet(K)* gene will also induce doxycycline resistance, leading to clinical failure if using doxycycline. This has led to the recommendation that MRSA infections that are resistant to tetracycline should be considered to be resistant to doxycycline regardless of the in vitro test result. In cases of tetracycline resistant MRSA infections, minocycline should be tested since, if the *tet(M)* gene is present, minocycline will be ineffective, but if the *tet(K)* gene is present, minocycline will be effective (Schwartz et al., 2009).

For treatment of canine SBF, an antibacterial agent should be administered for at least 21 days, or 7±14 days past a physical examination that has determined that the infection has resolved, whichever is longer. In SBF, glucocorticoid therapy should be withheld when the pruritus is only at the site of the lesions or when the pruritus in non-lesional areas is only mild. If a dog with a SBF has intense pruritus in non-lesional areas, then a tapering 21 day course of prednisone may be dispensed. Using glucocorticoids in the presence of a pruritic pyoderma makes interpretation of response to therapy impossible, since it is uncertain whether the glucocorticoid or the antibacterial therapy/antifungal therapy was responsible for
the resolution of the pruritus. Treatment with glucocorticoids may also have a negative effect on the response to antibacterial agents.

A consensus statement titled ‘Guidelines for use of antibacterial drugs for superficial bacterial folliculitis in dogs’ will soon be released (Hillier and Lloyd, 2012). In this publication, the steps involved in the proper selection of antibacterial agents for canine SBF will be reviewed. These guidelines, like the previous guidelines published concerning antibiotic use for treating urinary tract infections in dogs and cats (Weese et al., 2011), are the result of a committee consisting of veterinary internists, pharmacologists, microbiologists and dermatologists. The key points are: (1) identify and treat the underlying cause of SPF; (2) perform skin scrapings to determine if Demodex spp. are present; (3) perform cytology to confirm a bacterial component; and (4) if possible, use disinfectants and/or topical antimicrobials as the sole treatment; if this is not possible, at least use topical therapy to shorten the length of time that systemic antibiotics need to be used; and (5) empirical therapy can be used in non-recurrent cases or recurrent cases that have successfully responded to previous treatment; drugs should be selected from the list of first tier medications.

In cases that fail to respond to appropriate treatment (dose and frequency) using a first tier antibiotic, bacterial culture should be performed. When selecting an antibiotic based on a culture result, a sensitive second tier antibiotic should only be used if the organism is resistant to all first tier antibiotics, if the animal cannot tolerate any of the first tier drugs or if the owner is unable to administer them.

In regards to second tier antibiotics, when appropriate, a veterinary fluoroquinolone should be administered rather than ciprofloxacin. This recommendation is based on a study
showing that there is a large variation in absorption of oral ciprofloxacin in dogs; to obtain appropriate blood levels it would be necessary to administer 12-52 mg/kg ciprofloxacin orally (Papich, 2012). Since this dosage was for highly susceptible bacteria (i.e. with low MIC), larger doses may be required for organisms with high MICs.

When administering rifampicin, a dosage of 5-10 mg/kg once daily should be prescribed for a maximum of 21 days due to potential hepatotoxicity (Papich, 2007). To prevent rapid onset of resistance when treating *S. aureus* in human beings, Liu et al. (2011) suggested that rifampicin should not be used as the only systemic therapy. However, this recommendation is contrary to a review of clinical trials in which rifampicin was effective against *S. aureus* whether administered as monotherapy or in combination (Falagas et al., 2007). In dogs with MRSP, resistance to rifampicin may occur during therapy whether used as a monosystemic drug or in combination (Kadlec et al., 2011). The author has successfully used rifampicin as monosystemic therapy.

Concern about using third generation cephalosporins is shared by numerous groups and agencies. The *BSAVA Guide to the Use of Veterinary Medicines* discusses the prudent use of antimicrobial agents; with respect to third generation cephalosporins, and also for any fluoroquinolones, the guide states ‘that in all species fluoroquinolones and third- and fourth-generation cephalosporins should be used judiciously and never considered as first-choice options’.

The European Medicines Agency (EMA) is also concerned about the use of third generation cephalosporins and fluoroquinolones and states that ‘It is prudent to reserve third

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generation cephalosporins for the treatment of clinical conditions, which have responded poorly, or are expected to respond poorly, to other classes of antimicrobials or first generation cephalosporins' and 'use of the product should be based on susceptibility testing and take into account official and local antimicrobial policies'.

In Sweden, guidelines were published in 2009 concerning the use of antimicrobial agents in the treatment of dogs and cats; these guidelines state that third generation cephalosporins should only be used to treat infections where there are no other suitable options. The guidelines also state that injections with long-acting antimicrobial agents should not normally be used to treat a pyoderma and, specifically, that cefovecin should only be used if the treatment is ‘of the utmost importance’ for the animal and where administration of other medications is not possible.

Cefovecin (Convenia, Zoetis) is a parenterally administered third generation cephalosporin that has tremendous value when used properly (selectively). The author reserves this drug for cases where the owner is unable to orally medicate the dog or cat, or the animal cannot tolerate oral antibiotics. The concern about using this antibiotic is that, after the first injection, therapeutic drug concentrations (above MIC) are only maintained for 7-14 days, depending on the infectious agent, while tissue levels persist for up to 65 days. An unanswered question is whether this prolonged subtherapeutic blood (or tissue) level will put selective pressure on the bacteria such that the incidence of MRS will increase. Will there be an increase in the incidence of extended spectrum β-lactamase (ESBL)-producing bacterial


5 See: http://svf.se/Documents/S%C3%A4lskapet/Sm%C3%A5djurssektionen/Policy%20ab%20english%2010b.pdf (accessed 9 November 2013).
infections, as has been reported with other third generation cephalosporins (Colodner et al., 2004)? ESBLs are capable of conferring bacterial resistance to the penicillins, as well as first, second and third generation cephalosporins. Will adverse reactions require prolonged treatment due to the longer systemic drug clearance? What are the long term effects at injection sites, especially in cats? Most of these questions have not been answered.

An additional concern about both fluoroquinolones and third generation cephalosporins is that they are both independent risk factors for development of ESBL-producing bacterial infections (Colodner et al., 2004). ESBLs are a concern in human medicine because they are frequently MDR, not only to β-lactam antibiotics, but also to other antimicrobial agents, such as aminoglycosides, fluoroquinolones, tetracyclines, chloramphenicol and sulfamethoxazole-trimethoprim. This wide ranging resistance greatly limits effective treatment options. The genes encoding this resistance are mediated by plasmids and/or mobile elements, allowing horizontal transfer between the same and different species of bacteria (Vaidya, 2011). In contrast to fluoroquinolones and third generation cephalosporins, first generation cephalosporins have not been reported to be a risk factor for such resistance (Colodner et al., 2004).

Additional concerns about using fluoroquinolones are that, according to information from the Centers for Disease Control and Prevention (CDC) website, ‘none of the fluoroquinolones are Federal Drug Administration approved for the treatment of MRSA infections. A major limitation of fluoroquinolones is that resistant mutants can be selected with relative ease, leading to relapse and treatment failure’. 6 MRSA strains are especially adept at developing fluoroquinolone resistance and such resistance is already found among MRSA isolated from human beings with community-associated MRSA infections. In

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addition, there is a significant association between total fluoroquinolone use in human hospitals and the proportion of *S. aureus* isolates that are MRSA, as well as between total fluoroquinolone use in the community and the percentage of *E. coli* isolates that are fluoroquinolone resistant (MacDougall et al., 2005). There is an association between the use of fluoroquinolones against *mec(A)* positive *S. aureus* and an increase in the resistance index for methicillin resistance (Venezia et al., 2001). There is also an association between the use of fluoroquinolones and the occurrence of clinically significant MRSA (Crowcroft et al., 1999; Polk et al., 2004).

**Topical treatment**

Systemic therapy for canine pyoderma is becoming more problematic because of the increasing incidence of MRS (Morris, et al., 2006; Loeffler et al., 2007; Jones, et al., 2007). To help address this problem, topical therapy, either as monotherapy or as part of polypharmacy, has become an essential component of managing SPF. Topical therapy may decrease the length of time administering, or eliminate the need for, systemic antibiotics. Since dogs with SBF frequently have atopic dermatitis, bathing will also remove problematic allergens, in addition to bacteria, from the skin. The limitations of using topical therapy include time constraints of the owner, patient cooperation (especially with baths) and, if treating a large area, possibly cost.

Shampoo ingredients that are effective for treating bacterial pyoderma include chlorhexidine, benzoil peroxide, ethyl lactate, triclosan and boric acid/acetic acid. Chlorhexidine was found to be the most effective ingredient in shampoos (Loeffler et al., 2012; Young et al., 2012). However, the widespread use of chlorhexidine in human beings has resulted in reports of reduced chlorhexidine susceptibility (Noguchi et al., 2006). This
resistance is of concern, since chlorhexidine is used for decolonizing human beings with MRSA. Adding to this concern is circumstantial evidence of horizontal transfer of plasmids carrying these resistance genes. Whether or not the use of chlorhexidine in veterinary medicine will add additional resistance pressure on methicillin resistant members of SIG is unclear at this time.

Bleach baths have been reported to be effective for the treatment and prevention of MRSA in young children with atopic dermatitis (Huang et al., 2009). The author has seen very good responses in cases of MDR MRSP in dogs using a 0.06-0.12% solution as a soak 2-4 times weekly. These soaks are followed by a moisturizer or humectants.

Topical therapy with mupirocin is effective for treating localized lesions (Mueller et al., 2012). An advantage of mupirocin is its unique mechanism of action; as a consequence, cross-resistance with other antibacterial agents is very uncommon. However, there have been disturbing reports of mupirocin-resistant MRSA strains (Deshpande et al., 2002; Loeffler et al., 2008). As with chlorhexidine, this resistance is plasmid-mediated and spreads horizontally. Since mupirocin is used for decolonizing human beings with MRSA, restricted use has been advised (Simor, 2007). It is currently unknown whether this resistance will occur in methicillin resistant members of SIG.

The anti-fungal agent miconazole also possesses antibacterial properties against some Gram positive bacteria, including MRSA and MRSP in vitro (Weese et al., 2012). Whether miconazole proves to have the same in vivo effect is unknown. Silver sulfadiazine has traditionally been used for infections associated with Gram negative bacteria, especially
Pseudomonas spp. (Hillier, 2006). However, it is also effective against some Gram positive bacteria, including S. aureus. Its effectiveness on S. pseudintermedius infection is unknown.

Conclusions

If a dog is presented with lesions consistent with SBF, such as papules, pustules and/or epidermal collarettes, skin cytology and a deep skin scraping should be performed. A fungal culture and/or bacterial culture should be considered, depending on the history, physical signs and cytological results. In cases of SPF, if cocci are seen on cytology and the lesion is focal, treatment with an antimicrobial shampoo, with or without a residual spray or cream, may be effective. If the infection is more widespread and there has not been a recent history of antibiotic use, a first tier antibiotic may be prescribed. A re-examination should be performed after 14-21 days to assess the response to therapy. Lastly, there should be an attempt to find and treat the underlying primary cause of the SBF.

Treating SBF requires both symptomatic treatment and identifying and treating the primary cause; however, if only symptomatic treatment is given, recurrence is likely to occur. In view of increasingly resistant bacteria involved in SBF, it is becoming essential to include topical therapy as a part of the symptomatic treatment of SBF. In addition it is necessary to be more restrictive in dispensing second tier antibiotics to reduce the selection pressure these drugs apply to bacteria. Even though using antimicrobial agents may not harm an individual animal, excessive usage has led to the spread of resistant bacteria, both in human beings and veterinary species. Hopefully, this disturbing trend can be disrupted with good stewardship of the use of antimicrobial agents.
Conflict of interest statement

The author of this paper has no financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

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