Certain skin diseases universally present diagnostic and/or treatment dilemmas for general practitioners and specialists alike, and opinions abound on the “best” way to handle these conditions. This uncertainty largely reflects our lack of knowledge about these skin diseases and our relative inability to make evidence-based statements.

This article presents 5 dermatologic conditions that raise plentiful questions, inspire a variety of opinions, and result in contentious discussion; often there is no one correct answer. The aim of the following presentations is to help you formulate your own approach to these dilemmas, and they are accompanied by key points on each topic, questions to be considered, and useful references for further study.

DILEMMA 1. I’ve diagnosed methicillin-resistant staphylococcal pyoderma. Now what?

Staphylococcal pyoderma is one of the most common dermatologic conditions in dogs. In addition, over the past 10 years, prevalence of antibiotic resistance in canine staphylococcal infections has substantially increased.1

If methicillin-resistant Staphylococcus (MRS) is present, the laboratory report will indicate that the organism is resistant to all beta-lactam antibiotics, including all penicillins and cephalosporins. Many strains of MRS are resistant to multiple classes of antibiotics, leaving the practitioner with few good options for treatment.

Clinical Pearls
The clinical pearl here is topical antiseptic therapy. We’re used to thinking of topical products as adjunct treatments (ie, used in addition to systemic antibiotics), but the thought process has changed. Now dermatologists are advocating that topical therapy be used instead of systemic antibiotic therapy in both resistant and nonresistant strains of Staphylococcus when possible.

Reducing systemic antibiotic use helps minimize risk for antimicrobial resistance. Topical antiseptic products kill even highly antibiotic-resistant strains. Most research in this area has been done with chlorhexidine, which is an excellent choice for topical therapy.2,3

Mild to moderate superficial pyoderma responds very well to daily topical application of 2% to 4% chlorhexidine (the percentage does not seem to matter), generally for 3 to 4 weeks (Figure 1). Key points are:
• Medication must be applied daily.
• To ensure daily application, provide the client with a spray, mousse, or other “leave-on” product to make frequent application easy for the client; whole-body chlorhexidine shampooing can still be done weekly.

More severe cases, or cases of deep pyoderma, still require systemic antibiotic treatment; choice of antibiotic is based on

FIGURE 1. Abdomen of a dog with papules, pustules, and epidermal collarettes indicative of superficial pyoderma. The treatment of choice for this localized disease is 2% to 4% chlorhexidine, applied once daily by spray, mousse, or other formulation, for 3 weeks or longer.
susceptibility results. It is particularly important to continue treatment until at least 2 weeks past complete clinical remission. Here, topical antiseptic treatment is a valuable adjunct therapy.

Pitfalls & Challenges
And the pitfalls? Among them, terminology and client education.

In dogs, MRS is usually *Staphylococcus pseudintermedius* (MRSP, an animal strain), but occasionally *S schleiferi* (MRSS, also of animal origin) or *S aureus* (MRSA, a human strain) is the culprit. It is important to get the species terminology and abbreviations correct; do not refer to these infections generically as “MRSA.”

A client using “MRSA” as an Internet search term will quickly become frightened by horror stories of human contagion. Instead:

- Reassure clients that MRSP and MRSS are animal pathogens that are uncommonly transmitted to humans.
- Point the client to websites with accurate, up-to-date information about MRS; one of the best is wormsandgermsblog.com.

For the practitioner interested in additional information about treating canine pyoderma, excellent reviews have been published recently.4,5

DILEMMA 2. What’s the best way to diagnose canine atopic dermatitis?
According to the current definition, canine atopic dermatitis (AD) is a genetically predisposed inflammatory and pruritic allergic skin disease with characteristic clinical features, associated with immunoglobulin E (IgE) most commonly directed against environmental allergens.6

Pitfalls & Challenges
These environmental allergens include grass, tree, and weed pollens as well as molds and dust mites. This most recent definition allows that, in some individuals, the dermatitis may be triggered or exacerbated by other factors, such as food items. Additionally, it appears that some individuals with “atopic-like” clinical signs do not produce detectable allergen-specific IgE, illustrating the multifactorial pathogenesis of this condition.

For dogs with distinctly seasonal signs, diagnosis is not particularly difficult; the main rule out is seasonal occurrence of external parasites. For dogs with nonseasonal signs, however, making an accurate diagnosis can be challenging mainly because the most common clinical signs of AD are not pathognomonic. Numerous other skin conditions have overlapping signs. In addition, some dogs suffer from more than one allergic condition.

Clinical Pearls
In 2010, a prospective study that evaluated over 1000 pruritic dogs from 15 countries was published.7 In this study, AD was defined as cases with clinical features of atopic dermatitis irrespective of the offending agent. Clinical signs in dogs with AD were compared with signs observed in a group of dogs with pruritus from other causes, including fleas, scabies, other parasites, primary skin infections, and miscellaneous causes.

As a result of this study, new diagnostic criteria for AD were proposed (Table). A dog satisfying at least 5 of the 8 criteria has about an 80% chance of having AD. These criteria provide a useful and rapid screening method to identify potentially atopic dogs but must be used with some caution.

1. The criteria are only an initial step; dogs fulfilling the criteria still must undergo diagnostic testing to rule out other conditions that can mimic AD.
2. The criteria are focused on dogs with typical clinical signs of AD, and some dogs have atypical presentations. An 80% chance of being right also means a 20% chance of being wrong.
3. Finally, 20% of dogs with clinical signs consistent with AD had food-induced signs that were indistinguishable from those of non–food-induced AD.

Most recently, subgroups of the International Committee on Allergic Diseases of Animals have published practical “white paper” reviews

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If 5 or more of these criteria are present, there is an 80% chance that AD is the cause of pruritus.

- Age of onset < 3 years
- Mostly indoors
- Corticosteroid-responsive pruritus
- Chronic or recurrent yeast infections
- Affected front feet
- Affected pinnae
- Nonaffected front feet
- Nonaffected ear margins
- Nonaffected dorsolumbar area

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DILEMMA 3. What is the best way to diagnose food allergy in dogs and cats?

Asking this question may be the single easiest way to start an argument among dermatologists. Food hypersensitivity is considered the third most common allergic condition diagnosed in dogs and cats after flea allergy and AD. It can be seen alone or in combination with these other allergies.

The controversies surrounding diagnosis of food allergy illustrate our inability to make evidence-based statements about this disease.

Pitfalls & Challenges

On clinical examination, there are no specific dermatologic signs that reliably differentiate food allergy from the other types of allergic disease (Figure 2). Clues to the presence of food allergy include:

• History of year-round skin disease that does not always respond to standard anti-itch therapy, such as glucocorticoids
• Presence of concurrent gastrointestinal signs (eg, mild or intermittent diarrhea, vomiting, flatulence)
• Initial occurrence during puppyhood (though food allergy can occur at any age).

Clinical Pearls

The few things that experts are likely to agree on regarding food allergy are:

• Serum allergy tests are not reliable for diagnosis. False-positive and false-negative results are common.
• The only accurate test for food allergy is a carefully performed, strict, dietary restriction–provocation trial.

FIGURE 2. Pruritic abdominal/inguinal region in a dog with erythematous inflammatory allergic skin disease. Note the indication of chronic, low-grade inflammation as evidenced by the “lacy” hyperpigmentation pattern. It is impossible to distinguish via clinical signs whether the important allergens for this dog are environmental, food-related, or a combination thereof.

PEARLS & PITFALLS: Key Points

1. Watch out for methicillin-resistant Staphylococcus (MRS) as a cause of canine pyoderma.
   • To minimize development of antimicrobial resistance, treat dogs with mild to moderate superficial pyoderma with daily topical chlorhexidine, not antibiotics.
   • Steer your clients to good sources of information on MRS, such as wormsandgermsblog.com.

2. Diagnosis of canine atopic dermatitis is made using clinical criteria, not with an allergy test. For a recent review of clinical diagnosis, see reference 8; for up-to-date treatment recommendations, see reference 9.

3. It is typically impossible to differentiate food allergy from other allergies based on clinical criteria alone. Serum testing for food allergy is unreliable; for correct diagnosis, use a dietary restriction–provocation trial.

4. Early reports indicate that the isoxazoline compounds (afoxolaner and fluralaner) may be excellent treatments for canine generalized demodicosis. Stay tuned for more studies.

5. Avoid long-term treatment of idiopathic feline eosinophilic granuloma complex diseases with injectable long-acting corticosteroids. Such use promotes both diabetes mellitus and treatment resistance. Instead, consider protocols using cyclosporine or chlorambucil.
A diet trial must be performed for up to 3 months, although some animals experience relief much sooner. Diet trials should never be performed with “pet store brand” diets, as these diets often contain trace amounts of important allergens not listed on the label. The most frequently asked question—“Which diet is the best to use?”—is the one that generates the most controversy. No convincing evidence exists regarding whether a home-cooked or commercially prepared diet is better for a given patient. Regardless of the diet chosen, compliance is paramount and strict adherence to the prescribed food is necessary.

DILEMMA 4. What’s best for treating canine generalized demodicosis?

Pitfalls & Challenges

Treatment options for canine demodicosis have been limited by lack of available products for treatment and potential adverse effects:

• Amitraz is still a viable treatment option, although frequent dipping presents formidable compliance problems.
• Oral ivermectin (0.4–0.6 mg/kg Q 24 H) is preferred by many, but adverse reactions (typically neurologic signs) can occur.
• Milbemycin (1–2 mg/kg PO Q 24 H) is very effective and has recently reappeared on the U.S. market, but daily use makes it rather costly.
• Topical once-weekly moxidectin + imidacloprid has met with variable success in the U.S.
• Off-label use of doramectin (0.6 mg/kg SC once weekly) is sometimes considered, but may also produce adverse effects similar to those caused by ivermectin.

It is important for clients to understand that, regardless of the regimen selected, treatment must continue for a very long time—until repeated skin scrapings are negative—and, if treatment stops prematurely, the disease will surely return.

Clinical Pearls

This dilemma appears to be resolving, however, with the recent buzz about use of the isoxazoline compounds (afoxolaner and fluralaner) as off-label treatments for canine generalized demodicosis. There is only one published study of such use, but dermatologists are anecdotally reporting tremendous success with these drugs—even at the usual label-indicated, flea-and-tick prevention doses (Figure 3). More studies are sure to follow, and we may be on our way to an easy method for long-term control of this serious dermatosis.

DILEMMA 5. How should I treat feline eosinophilic granuloma complex?

Feline eosinophilic granuloma complex is a clinical syndrome that classically manifests as eosinophilic ulceration, eosinophilic plaque, or collagenolytic granuloma (Figure 4, page 75).

This syndrome should not be viewed as a specific diagnosis but rather as a reaction pattern in feline skin that may be induced by many different definable underlying causes, such as parasites, infection, or allergy. However, it is clear that feline eosinophilic granuloma complex can exist as an idiopathic condition on its own, without a
2. The disease is, or becomes, glucocorticoid resistant; however, and it appears that this dilemma may be advisable in some cases.

Definable cause. In such cases, initial treatment has often centered on long-acting injectable glucocorticoids, such as methylprednisolone acetate. When the disease is idiopathic and recurrent, problems arise when:

1. Each glucocorticoid injection makes development of diabetes mellitus more likely or
2. The disease is, or becomes, glucocorticoid resistant.

Other treatment protocols include those based on:

- **Cyclosporine**: May need to be used at doses up to 13.3 mg/kg PO Q 24 H or
- **Chlorambucil**: 0.1–0.2 mg/kg PO Q 24 H or Q 48 H.

Exercise caution with these protocols: this dose of cyclosporine is nearly twice the feline label dose of 7 mg/kg, and possibility of adverse effects is increased, while chlorambucil is a potent and potentially myelosuppressive drug. Monitoring cyclosporine levels and complete blood counts may be advisable in some cases.

The treatments outlined are not always effective, however, and it appears that this dilemma may persist for some time to come.

**References**


**FIGURE 4.** Eosinophilic granuloma disease (in this case, collagenolytic or “linear” granuloma) in a young cat. Although this pattern is usually thought to be a reaction to a parasite or allergen, some cases are idiopathic and may be treatment resistant.