# CANINE PYODERMA

D.N. CARLOTTI, Dip ECVD

Héliopolis B3, Avenue de Magudas, F-33700 Bordeaux-Mérignac

I - Introduction

Pyoderma is one of the most common diseases of the dog. Although it is usual to say pyoderma, the

plural pyodermas should be used, since there are a large number of bacterial diseases of the skin in this

species, with different histopathological and clinical aspects. Some are superficial (the basement

membrane is <u>not</u> destroyed by the infectious process) and some are deep (the basement membrane <u>is</u>

destroyed). Pseudopyodermas are not real pyodermas since infection plays only a secondary role and

antiinfectious therapy is not effective.

As many pyodermas can mimic other skin diseases including pruritic diseases such as allergies and above

all can be secondary to other skin diseases, an accurate diagnosis is mandatory.

II - Pathogenesis of canine pyoderma

Cutaneous flora in healthy dogs and dogs with pyoderma A.

The skin surface in animals and humans is colonized by bacteria that are well adapted to the

microenvironment of the superficial stratum corneum and the hair follicles. In this way, the normal flora

contributes to skin immunity (1).

Staphylococcus intermedius, a gram positive coagulase positive bacterium, is the most common infectious

agent cultured in canine pyoderma. One can also culture Staphylococcus aureus and Staphylococcus hyicus.

Some healthy dogs carry these potential microbial pathogens in low number on the skin surface, but it is

likely that they have transient/nomad rather than resident status.

In contrast these coagulase positive Staphylococci are frequently isolated from mucous membranes (anus,

nose, genital tract, mouth, conjunctivae) and are probably resident at these sites. They might be seeded onto the skin and hair by grooming/licking (2).

Studies on isolates of *Staphylococcus intermedius* from both normal dogs and dogs affected with pyoderma have failed to detect any evidence that different strains of this bacterium vary in pathogenicity (3).

## B. Predisposing factors for pyoderma in dogs

The *stratum corneum* is composed of squames (or surface keratinocytes). Squames are compact plates of keratin embedded in an emulsion of sweat with lipids from the epidermis and sebum. It is a physical barrier limiting penetration of microorganisms and their products (4).

In the dog, the *statum corneum* is far thinner and more compact than that of any other species, and there is a paucity of intracellular emulsion. Futhermore, the hair follicule infundibulum of the dog is open, lacking a sebum plug (5,6).

### C. Primary causes of pyoderma

Pathogenic Staphylococci can easily colonize an inflammed and excoriated, seborrhoeic skin.

In fact, inflammed skin has accelerated epidermal proliferation and desquamation and is more humid and warmer. These alterations of the skin surface micro-environment promotes the multiplication of bacteria. Furthermore, self-trauma and excoriations due to pruritus further degrade the epidermal defences and allow inoculation of bacteria into the skin and also allow leakage of serum which is a source of nutrients for bacteria (7). In addition, there is evidence from an experimental model of canine cutaneous type I hypersensitivity that injection of a mast cell degranulator or histamine intradermally renders the overlying epidermis more permeable to bacterial antigens (8). Allergic skin disease is thus probably one of the most common causes of canine pyoderma.

It has been demonstrated that dogs affected with primary seborrhoea have markedly higher cutaneous bacterial counts than normal dogs, with a flora composed primarily of coagulase-positive *Staphylococci* (9). Any seborrhoeic skin disease (including allergic skin disease and endocrinopathies) is therefore a possible cause of canine pyoderma.

So, as HALLIWELL stated in 1989: « it seems likely that the majority of reccurent bacterial skin diseases in the dog result from surface abnormalities, which permit bacterial colonization, and that immunological predisposing causes are relatively infrequent » (10).

However deep pyoderma can be due to primary deficiency of specific cellular immunity. The best examples are juvenile generalized demodicosis and idiopathic deep pyoderma (cellulitis), particularly in German Shepherds. Also secondary (acquired) deficiency of specific immunity can lead to deep pyoderma. Good examples in canine dermatology are endocrine diseases (hypothyroidism, Cushing's disease), leishmaniasis, and adult-onset demodicosis.

A few studies intended to elucidate a suspicion of immunodeficiency responsible for idiopathic deep pyoderma (cellulitis), particularly in German Shepherds. Of course, affected dogs had not received glucocorticoids, had no demodicosis, spontaneously occurring Cushing's disease or hypothyroidism, leishmaniasis, ehrlichiosis or SLE.

Wisselink created the concept of German Shepherd Dog Pyoderma (1989), in 21 dogs with no serum defect of Ig or complement, no anomaly of chemotaxis or bactericidal action of phagocytes (13). The disease could be due to an autosomal recessive gene.

Carlotti and Valensi reported in 1990 8 cases of cellulitis (including 4 German Shepherd dogs) with a normal leucocyte phagocytosis but with a low response to LTT which resolved with clinical healing (14). Miller also observed a lack of response to LTT in 2 dogs in 1991 (15).

Above all, Day suggested in 1994 a severe T-cell deficiency (16) and Chabanne et al showed in 1995 a defect in CD4+ and an increase in CD8+, ie an alteration of helper T-cell functions, as well as an increase in CD21+ in affected German Shepherd dog (17).

There is no relation with FAD in these dogs and pruritus resolves with antibacterial therapy. Prognosis is guarded and long-term antibiotic therapy is required (without glucorticoids) combined with local antimicrobial cleansing.

THE question is the following: are these alterations the cause or the consequence of the cellulitis? Is there a vicious circle, with *Staphylococci* enhancing their own pathogenicity?

# III - Different clinical forms : classification of canine pyoderma

These are 2 examples of classification of canine pyoderma.

- Superficial Pyoderma
  - \* skin fold pyoderma
  - \* impetigo
  - \* folliculitis
- Deep Pyoderma
  - \* furunculosis
  - \* cellulitis
  - \* interdigital pyoderma complex
- Pseudo-Pyoderma
  - \* pyotraumatic dermatitis
  - \* juvenile pyodermas

Classification of pyoderma by P. FOURRIER and D.N. CARLOTTI (11)

- Surface Pyoderma
  - \* pyotraumatic dermatitis
  - \* intertrigo (skin fold pyoderma)
  - \* mucocutaneous pyoderma
- Superficial Pyoderma
  - \*impetigo
  - \*superficial folliculitis
  - \* superficial spreading pyoderma
- Deep Pyoderma
  - \* deep folliculitis and furunculosis
  - \* cellulitis
- Diseases formaly classified as Pyoderma
  - \* juvenile cellulitis
  - \* hidradonitic cunnurativa

Classification of pyoderma by P.J. IHRKE (12)

# IV - Superficial pyoderma

These diseases are usually benign.

#### A. Skin fold pyoderma (intertrigo)

These lesions are seen in anatomical defects where there is an important bacterial colonization: lip, facial, vulvar, caudal, obese and mammary folds. The dermatosis is localized with erythema, exsudation, suppuration and bad odour.

### B. Impetigo

1. Juvenile impetigo: subcorneal pustules are present on the ventral side of the body, with crusting. The

disease is self-limited.

2. Adult impetigo : large pustules (« bullae ») are seen all over the body. The disease is sometimes severe, eventually with prostration. In general, adult impetigo is secondary to an underlying disease (hyperadrenocorticism, glucocorticoid therapy...) or multiple traumas (eg during hunting).

#### C. Folliculitis

- 1. Juvenile folliculitis: numerous follicular pustules are present on the ventral side of the body. The condition oftern heals at puberty.
- 2. Short-haired dog pyoderma : there are generalized follicular pustules, epidermal collarettes and crusts, with a « moth-eaten » hair. The disease is seen in short-haired breeds. Pruritus disappears when the lesion heal.
- 3. Secondary folliculitis: this common disease is characterized by follicular pustules, epidermal collarettes and crusts which are often generalized. Pruritus is still present after lesions healing in case of underlying pruritic dermatosis. The disease may also generate pruritus in a usually non-pruritic dermatosis (in such cases pruritus disappears when lesions heal).
- 4. « Bacterial hypersensitivity » and/or superficial spreading pyoderma : bacterial hypersensitivity is an uncommon disease based on a clinical triad : erythematous follicular pustules, target lesions/seborrhoeic plaques, haemorragic bullae. There is sometimes a severe pruritus. The existence of a real bacterial allergy is presumed and debatable. In superficial spreading pyoderma, nummular areas of alopecia and erythema are centrifugally expanding, with epidermal collarettes and crusts. These lesions are often associated to intact but transient follicular pustules.
- 5. Deep folliculitis: it is the so-called acral lick dermatitis, which is, in the majority of cases if not all a deep follicular bacterial infection with often retrograde hidrosadenitis. A psychogenic and/or an allergic cause should be looked for after antiinfectious treatment.
- 6. Pyotraumatic folliculitis: some cases of folliculitis (eg in Labradors, Retrievers) appear as oozing suppurative plaque with pain. They are surrounded by pustule of folliculitis or even furunculosis, which help to differentiate them from the « classical » pyotraumatic dermatitis (see below).

# V - Deep pyoderma

These diseases are usually severe.

#### A. Furunculosis

- 1. Acne : papulo-pustules and pustules are seen on the face, particularly the chin. The disease occurs mainly in young dogs.
- 2. Secondary furunculosis: localized or generalized pustules are visible. The disease is associated or secondary to a folliculitis and triggered or aggravated by an excessive therapy (eg glucocorticosteroids).
- 3. Nasal pyoderma: pustules and crusts are present on bridge of the nose and the eyelids. The cause is unknown. There may be an unpleasant scaring. This true bacterial nasal pyoderma should be differentiated from the sterile eosinophilic furunculosis possibly due to arthropod bites and not bacterial in origin.

#### B. Cellulitis

## 1. Localized cellulitis

- a. pressure points pyoderma: there are necrotizing lesions of the elbows, the rump, the stifles, the hocks and the lateral digits. They are due to permanent trauma in heavy dogs.
- b. various localized cellulites: These are other localized necrotizing lesions (eg perianal). Their cause is often unknown; they are sometimes secondary to a furunculosis.

#### 2. Generalized cellulitis

- a. pyodemodicosis: There is an extensive necrotizing skin disease, which is secondary to a generalized demodicosis (an immunodeficiency status).
- b. various generalized cellulites: necrotizing lesions are extensive and often secondary to other immunodeficiencies.

# C. The interdigital pyoderma complex

In non infectious pododermatitis (not a pyoderma), there is erythema, oedema, oozing and alopecia. There are very numerous causes of pododermatitis including surface bacterial and/or *Malassezia* overgrowth. The same lesions are present in infectious pododermatitis that is interdigital pyoderma along with others: furunculosis, ulcerations, fistulae and necrosis (cellulitis). Interdigital pyoderma is often secondary to pododermatitis due to other causes.

### VI - Pseudo-pyoderma

#### A. Pyotraumatic dermatitis

The typical lesions have an acute onset and are characterized by alopecia, erythema, oozing, suppuration, pruritus and/or pain. These lesions are common, and most often associated to pruritic skin disease. They are poorly understood (sometimes due to vasculitis?). There is a spontaneous healing in a few days, but a short systemic or topical glucorticoid treatment associated to a topical antiseptic therapy is useful.

# B. Juvenile pyodermas

- 1. Juvenile pyoderma of new-born puppies: crusty lesions are present on the face, thorax and dorsolumbar area. They might be due to trauma. No treatment is required since there is a spontaneous healing.
- 2. Juvenile cellulitis: The aetiology of this disease is unknown. The typical clinical aspect is a facial oedema and furunculosis, with fistulae, crusting and a suppurative otitis externa. Adenopathy and sterile abscesses (cellulitis) are present. The onset of this uncommon disease occurs before 4 months of age in one or several puppies of a litter. There is a spontaneous healing in a few weeks with scaring. However, systemic immunosuppressive therapy is required (prednisolone 2 mg/kg/day), and a control of secondary infection is beneficial.

# VII - Diagnosis of canine pyoderma

Diagnosis of canine pyoderma is based on history, physical examination and complementary examinations. The owner will often describe accurately the evolution of the pustular lesions, which will

be readily seen by the clinician. Three complementary aids can be used to confirm the clinical diagnosis of pyoderma.

## A. Cytology

In **intertrigo** (skin fold pyoderma), images of « bacterial colonization » are essentially observed, i.e. healthy neutrophils, *Cocci* and *Bacilli* in an extracellular position and degenerated neutrophils in a state of phagocytosis. Thus the hostile reaction of the body remains moderate. In **impetigo** and **folliculitis**, impaired (degenerated) neutrophils are only found, i.e. discoloured, swollen, with hypersegmented and pycnotic nucleus. The pictures of *Cocci* phagocytosis are not particularly numerous but always present and must, therefore, be looked for systematically. The extracellular *Cocci* may also be relatively abundant. This is an image of « bacterial invasion », i.e. the penetration of pathogenic germs into the cutaneous tissue.

The significance of the pictures of phagocytosis differs considerably depending on whether they are observed <u>on</u> the surface or <u>in</u> a cutaneous lesion. In effect, when they are observed inside the skin (epidermis, hair follicles, dermis) one might consider that the phagocytosed germs are pathogenic and that there is a real pyoderma, or in other words bacterial pustulosis. On the other hand, phagocytosis observed on the surface indicates superficial multiplication of germs which are not necessarily and probably rarely pathogenic.

Eosinophils are sometimes present in case of impetigo or folliculitis.

In **deep pyoderma** cytology is less likely to reveal the germs and pictures of phagocytosis, although they must be looked for. Often eosinophils and red blood cells are found. Frequently there is a granulomatous reaction.

The pictures observed in **pyotraumatic dermatitis** are identical to those in intertrigo. Here bacterial colonization is not significant and treatment with antibiotic does not result in remission. In **juvenile cellulitis**, the degenerated neutrophils are very numerous, sometimes with a granulomatous reaction. No germs are seen.

### B. Histopathology

This will show typical lesions, but is relatively rarely performed for the diagnosis of canine pyoderma,

except in case of difficult differential diagnosis and in the Sharpei in which pustules are sometimes difficult to see clinically. Subcorneal and follicular pustules are seen respectively in impetigo and folliculitis. Large spongiotic superficial pustules are seen in superficial spreading pyoderma. Nodular and diffuse intradermal pyogranulomatous reactions are observed respectively in furunculosis and cellulitis, with destruction of adnexae.

# C. Bacteriology

This can confirm the bacterial infection.

# VIII - Treatment of canine pyoderma (1,11,12,18-20)

Systemic and topical therapy can be used in canine pyoderma. Systemic antibiotic therapy is essential in both superficial and deep canine pyoderma. It is always indicated except perhaps in some cases of limited juvenile impetigo or non extensive folliculitis of various causes, in which topical antiseptic therapy may be sufficient.

### A. Selection of antibiotics

The criteria for the choice of an antibiotic are as follows:

- · appropriate kinetics and good cutaneous penetration,
- activity against Staphylococci,
- activity in pus and reactive tissues,
- bactericidal activity rather than bacteriostatic activity particularly in severe cases,
- easy administration (oral, q12h or q24h),
- absence of secondary effects,
- reasonnable cost.

The choice can be empirical, particularly in superficial pyoderma, after cytological examination of pus from an intact pustule which shows bacterial invasion. Bacteriology and sensitivity testing must be used in case of deep pyoderma, recurrent pyoderma, when cytology shows a complex flora with rods, and in case of empirical antibiotic therapy failure. They can be repeated during therapy.

#### B. Dosage and duration of treatment

Ideal doses must be used and duration of treatment must be long enough (a few weeks to several months depending of extension and depth of lesions, and always beyond clinical cure). Maintenance pulse treatment (e.g. 2 to 3 days a week) can be used in chronically relapsing pyoderma but it could theoretically select resistant strains as well as the use of subminimal doses. They are both used for economical reasons but the former is preferable.

# C. Antibiotics useable in canine pyoderma

Antibiotics useful in canine pyoderma are included in the following table. They all have a good cutaneous diffusion (because of their liposolubility) and can be given orally, which is useful because of long therapeutic courses (ease of administration). They are all bactericidal except macrolides which are bacteriostatic. Recent studies have confirmed the efficacy of many of them (5).

Class	Characteristics	examples
Macrolides	manually an achurum /Crama	erythromycin: 30 to 50 mg/kg div. bid or tid
Macrondes	narrow spectrum/Gram+	lincomycin: 40 to 50 mg/kg div. bid or tid clindamycin: 5,5 to 11 mg/kg sid or div. bid
		tylosin: 40 mg/kg div. bid
Penicillins M	resistant to penicillinases	tyloom. 10 mg/ kg arv. eta
2 4114111111111111111111111111111111111	narrow spectrum/Gram+	oxacillin: 30 to 50 mg/kg div. bid
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Penicillins A	resistant to penicillinases	amoxicillin-clavulanic acid: 25 mg/kg/ div.
potentiated by	larger spectrum	bid
clavulanic acid		
Cephalosporins	resistant to penicillinases	cephalexin: 30 to 60 mg/kg div. bid
	broad spectrum	cefadroxil : 44 to 70 mg/kg div. bid
Cephalosporin P	resistant to penicillinases	fucidic acid (the only one of this group):
	narrow spectrum/Gram+	60 mg/kg div. tid
	synergy with penicillins	
	and erythromycin	
Sulfonamides-	broad spectrum	trimethroprim-sulfa: 30 mg (i.e. 5 mg
Diaminopyrimidines		trimethoprim)/kg sid or div. bid
		baquiloprim-sulfadimethoxine: 30 mg
		(i.e. 5mg baquiloprim)/kg q.48h
		ormethoprim-sulfadimethoxine: 30 mg
		(i.e. 5mg ormethoprim)/kg sid after a single
		double dose the first day
Fluoroquinolones	broad spectrum	
	excellent tissue penetration	enrofloxacin: 5mg/kg sid of div. bid

(not to be used in puppies of	marbofloxacin: 2 mg/kg sid
giant breeds)	difloxacin : 5 mg/kg sid
	orbifloxacin : 2.5 mg/kg sid

Penicillin G (which is injectable) and A are sensitive to penicillinases. Aminoglycosides have a low cutaneous diffusion (they are hydrosoluble), are injectable and toxic. Chloramphenicol has a bad reputation in humans and the cat (haematologic toxicity). Tetracyclines have a very low activity against *Staphylococci*. These antibiotics are never or rarely used in canine pyoderma.

Rifampicin is effective against *Staphylococci* but, as it is still used to treat human tuberculosis, it should be used when there is no other therapeutical possibility (5 to 10 mg/kg SID). In addition, it should be then associated to a betalactamine to prevent the selection of resistant strains of *Staphylococci*.

Mupirocine, a topically active bactericidal antibiotic, in a polyethylene glycol base is effective against Gram+ *Cocci*, is not systematically absorbed and is not chemically related to other antibiotics. It can be used in localized pyodermas (acne, pressure point pyoderma, interdigital pyoderma).

### D. **Associated treatments**

Topical therapy is always beneficial in canine pyoderma, particularly in superficial staphylococcal disease. Clipping can be useful and is necessary in deep pyoderma such as cellulitis.

The main useful topical products are chlorhexidin (lotion and/or shampoo), povidone-iodine (lotion and/or shampoo), benzoyl-peroxyde (shampoo and eventually gel), ethyl-lactate (shampoo). They should be used frequently, e.g. once a day, at the begining of therapy. Later, frequency of application may decrease. Each shampoo should be followed by the application of an appropriate humectant.

Therapy of an underlying skin disease is mandatory and an appropriate diagnosis should be made. Staphylococcal immunotherapy (Staphage Lysate®) has been demonstrated to be effective in idiopathic superficial pyoderma.

Topical or systemic glucocorticoids should never be used in true canine pyoderma, even in case of pruritus, because they cause severe relapses (« rebound effect »). In contrast they can be used and are effective in pseudo-pyoderma (eg prednisolone: 1 mg/kg/day for pyotraumatic dermatitis and 2 mg/kg/day for juvenile cellulitis).

# IX - Conclusion

Canine pyoderma is a group of various skin diseases and an accurate diagnosis is mandatory. An

appropriate antibacterial therapy is required in most cases of canine pyoderma, in association with topical therapy. Antibiotics must be selected carefully and used with appropriate dosage and duration of treatment.

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