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Results

Seborrhea

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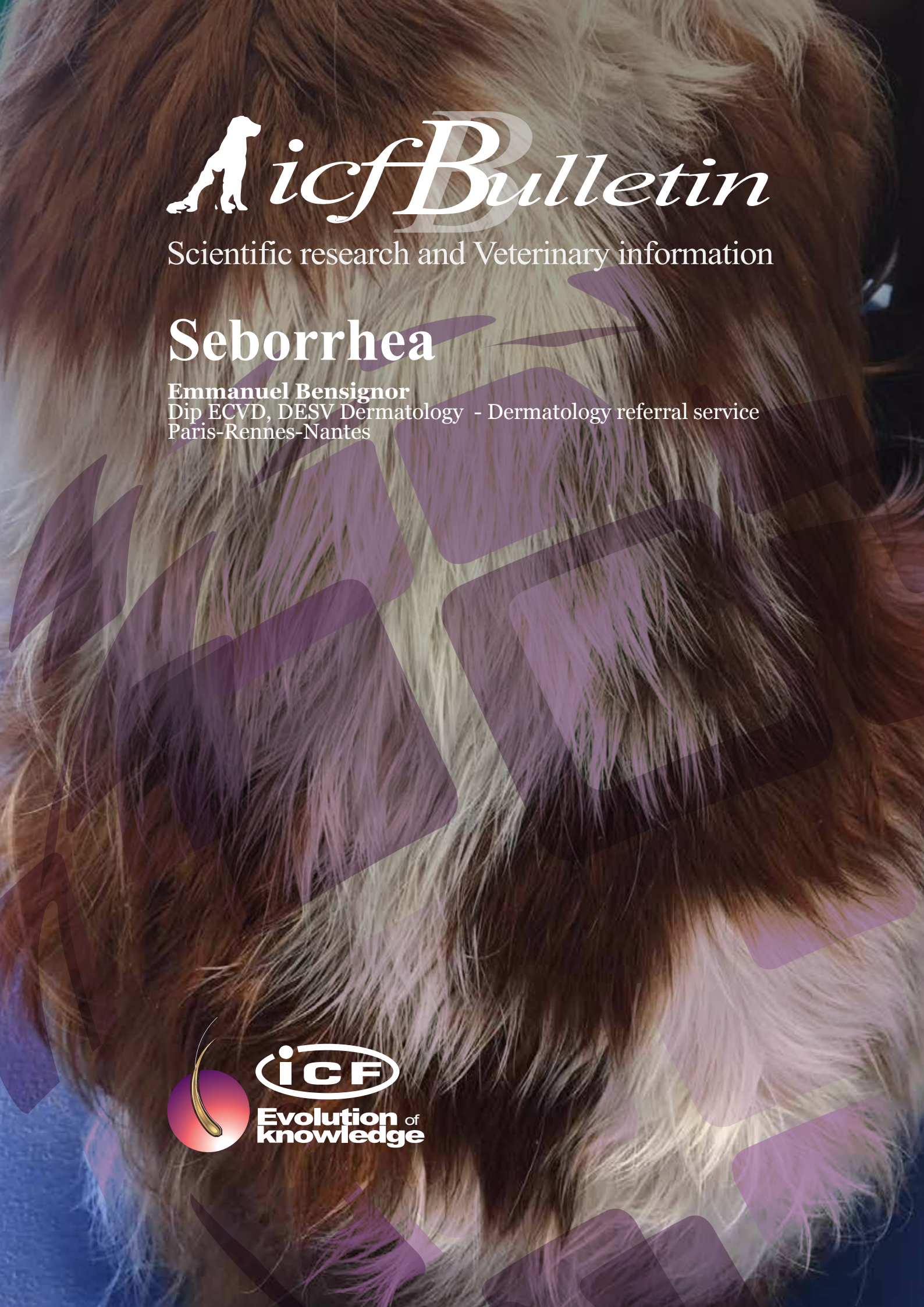

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Seborrhea

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Introduction

Keratinisation disorders are frequent in canine dermatology. They are characterised by alterations in surface lipids (both sebaceous and epidermal) and excessive scale production. Many skin diseases can be associated with scaling and seborrhea, which can be either primary (eg in genetic diseases such as ichtyosis) or, most often, secondary (eg in allergic dermatosis). Even if the recognition and the treatment of the cause responsible for the disease is mandatory, symptomatic topical therapy with antiseborrheic formulations is always needed in order to rehydrate the skin and to alleviate the symptoms.

Epidermal lipid film and keratinization disorders

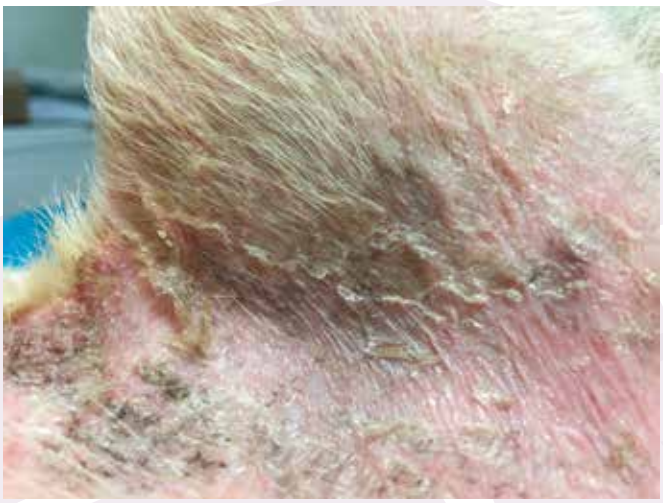
The epidermal film is composed of an emulsion derived from epidermal degradation products and sebaceous gland secretion. Its composition is complex: cholesterol, waxes (diesters) and free fatty acids derived from enzymatic hydrolysis of lipids by surface-living saprophytic bacteria. This superficial lipid film has many roles: protection, cohesion, lubricant, chemical and thermal protection. Good organisation of the cornified layer is ensured thanks to the lipid fraction, its corneocytes and its hydration level.

In kerato-seborrhoeic disorders, a reduction in waxes and a rise in free fatty acids and cholesterol is noted; the pH becomes more alkaline which promotes the proliferation of skin bacteria and explains the frequency of secondary infections. The skin lesions observed are dry and waxy or greasy and oily skin and hair, scaly and lichenified macules, hairs matted in greasy tufts, bad odour with or without pruritus.

Keratinisation disorders are caused by alterations in proliferation, differentiation or desquamation. It is possible to differenciate retention hyperkeratoses, associated with a corneocyte desquamation defect caused by biochemical changes to intercorneocyte cement lipids, following, for example, sebaceous gland destruction (leishmaniosis, granulomatous sebaceous adenitis) and proliferative hyperkeratoses, resulting from an increase in epidermal activity and subsequent defective keratinocyte maturation (differentiation), secondary to any inflammatory dermatosis (allergic skin conditions, ectoparasitic infestations, etc.).

Disturbances in keratinisation have various clinical consequences:

- Rapid, incomplete keratinisation leads to water loss. This makes the coat appear dull and is in part responsible for drying of the cornified layer; barrier function is no longer assured
- Enhanced release of membrane lipid into epidermal intercellular spaces increases disruption to the superficial lipid film
- Very rapidly shed corneocytes are abnormal in size and conformation, leading to grossly visible scales. Scales consist of white to greyish flakes depending on loss of cornified layer elements. Two types of scale have been described, according to size and thickness (psoriasiform– large and relatively thick; pityriasiform– small and thin). Excessive keratin production also takes place within hair follicles. It may be associated with sebaceous hypersecretion and, in some cases, leads to comedone and follicular cast formation. See pictures.



Primary keratinization disorders are linked to various genetic defects. Secondary keratinization defects are caused by an underlying dermatosis. Secondary disorders are far more common than primary disorders (see tables below). Histopathologic examination of skin biopsies is usually needed to differenciate between these diseases, especially in case of primary keratinization defects.

Examples of primary keratoseborrhoeic disorders:

- Primary idiopathic seborrhoea
- Follicular dysplasia (colour dilution alopecia, black hair follicular dysplasia and other dysplasias)
- Vitamin A-responsive dermatosis
- Zinc-responsive dermatosis
- "Epidermal dysplasia" or idiopathic hyperplastic dermatitis
- Sebaceous adenitis
- Lichenoid psoriasiform dermatosis and idiopathic lichenoid dermatitis
- Schnauzer comedone syndrome
- Ichthyosis
- Nasodigital hyperkeratosis
- Ear margin dermatosis
- Acne

Examples of secondary keratoseborrhoeic disorders:

- Ectoparasitic infestation (sarcoptic mange, cheyletiellosis, demodicosis, pediculosis and otodectes infestation)
- Allergic dermatitis (atopic dermatitis, food hypersensitivity, flea allergy dermatitis)
- Pyoderma
- Fungal infection (dermatophytosis, Malassezia dermatitis)
- Endocrinopathy
- Leishmaniosis
- Auto-immune and immune-mediated dermatoses
- Neoplasia (especially mycosis fungoides)

Topical therapy for keratoseborrheic diseases

Topical therapy is mandatory for managing keratoseborrhoeic disorders, because it allows the direct application of active ingredients on the skin. Several formulations are available: shampoos, lotions, sprays, rinces, spot-ons, … They contain keratomodulating and/or antiseborrheic agents. They are active by restoration of normal keratinocyte multiplication and keratinisation and by inhibition of sebum production, with a cytostatic effect reducing the rate of cell division. They may also eliminate excess of corneal cells, by increasing desquamation (ballooning of corneocytes renders the stratum corneum softer and reduction of intercellular cohesion increases their shedding).

Many various substances are useful and are incorporated in medicated products such as salicylic acid, coal tar, sulphur, selenium disulphide, ammonium lactate, phytosphingosine, benzoyl peroxide, zinc gluconate, pyridoxine, essential fatty acids, …

The choice of the product depends on the severity of the dermatitis: the more severe, the more active and potent ingredients shall be selected. For mild and/or pityriasiform keratoseborrhoeic disorders, keratolytic agents should be selected whereas for severe and/or psoriasiform disorders, keratoregulating (keratoplastic) agents will also be used. In all cases but particularly in greasy seborrhoea, antiseborrhoeic agents may be useful. The therapeutic agent often needs to be changed following the development of side effects, rebound effects or change in clinical presentation (e.g. transition from greasy seborrhoea to dry seborrhoea).

Various parameters can be used to assess *in vitro* or *in vivo* the efficacy of the topical medications: clinical improvement using various clinical scales, transepidermal water loss (TEWL) measures, counting of corneocytes (corneometry), skin surface biopsies, electron microscopy etc…

Systemic therapy

In case topical therapy is not effective enough, it is possible to use systemic medications: synthetic retinoids (isotretinoin, acitretine, 1-2 mg/kg/day) regulate proliferation and differenciation of keratinocyte, inhibit epidermal transglutaminases, corneal envelope formation, synthesis of cholesterol and collagenases and modulate keratin expression. They are also active on sebaceous glands by reducing their size and secretions. Their side-effects must be known by the clinician: teratogenicity, mucocutaneous junctional erythema, blepharo-conjunctivitis, pinnal pruritus, diarrhea, vomiting, hepatopathy, pancreatitis. Monitoring is therefore mandatory including blood panels and Schirmer tear tests. Vitamine A (400-600 UI/kg) and essential fatty acids have also been used in keratoseborrheic diseases.

Anecdotally the author has used cyclosporine in some keratoseborrheic disorders because of its effects on the keratinization process.

Conclusion

The diagnosis of the cause of the keratoseborrhoeic disease is mandatory. Once this is done, the clinician can decide of a specific therapy and a topical symptomatic treatment. Choice of an appropriate formulation, judiciously selected active ingredients and an appropriate frequency of application are essential. The prescription varies according to each case and must take into account the nature and extent of the lesions, the concurrent specific treatment, the animal's temperament and willingness of the owner to devote the necessary time. Therefore new formulations such as sprays, lotions or spot-ons are very interesting in addition to classical treatment such as shampoos.

References

Carlotti DN, Bensignor E. Management of keratoseborrhoeic disorders. Eur J Comp Anim Pract 2002: 12: 123-133

Curtis CF. Use and abuse of topical dermatological therapy in dogs and cats. Part 1- shampoo therapy. In Practice 1998: 20: 244-251

Kwochka KW. Overview of normal keratinisation and cutaneous scaling disorders of dogs and primary keratinisation disorders of dogs. In: Current Veterinary Dermatology (Griffin CE, Kwochka KW, Mac Donald JM, Edrs). St Louis: Mosby Year Book; 1993: 167-175 and 176-190.

Kwochka KW. Symptomatic Topical Therapy of Scaling Disorders. In: Current Veterinary Dermatology (Griffin CE, Kwochka KW, Mac Donald JM, Edrs). St Louis: Mosby Year Book; 1993: 191-202

Kwochka KW. Retinoids and Vitamin A therapy. In: Current Veterinary Dermatology (Griffin CE, Kwochka KW, Mac Donald JM, Edrs). St Louis: Mosby Year Book; 1993: 203-210

Scott DW, Miller WH, Griffin CE. Muller and Kirk's Small Animal Dermatology, 6th Edition. Philadelphia: WB Saunders Company, 2001: Sebaceous glands 48-51, Keratinization Defects 1025-1054

CASE 1

A case of sebaceous adenitis in an Akita Inu

A female Akita Inu, 4 years old, is referred at the dermatology service for a chronic skin disease with few improvement after treatment.

The dog used to live in the South of Europe, but recently moved to Paris. She lives in an appartment with high quality food. Monthly flea treatment is realized with afoxolaner. Skin lesions began two years before. Initially they consisted of scales localized on the trunk and then extending to the flanks, face, ears and generalized. A diffuse hair loss is also reported.
The referring vet initially treated with oral antibiotics and steroids with only few improvement. A serology for leishmaniasis was negative. Skin biopsies were performed and showed a granulomatous inflammation centered on sebaceous glands compatible with a sebaceous adenitis. The dog was then treated with a topical shampoo twice a week but as lesions did not improve, ciclosporine was added at the dose of 5 mg/kg/day with only a mild improvement.

At clinical examination the dog is in good general health. Skin lesions are generalized. They consist of a diffuse alopecia **(photo 1)**, seborrheic dermatosis with scaling **(photo 2)**, erythema and few papules and erosions **(photo 3)**. Tufts of hairs are easily epilated and shows keratin aggregates compatible with follicular casts **(photo 4)**.

The most likely differential diagnosis due to previous complementary examinations was a bacterial pyoderma complicating the sebaceous adenitis. Less likely secondary demodicosis or dermatophytosis were considered.

Photo 1

Photo 2

Photo 3

Photo 4

Skin scrapings did not show any mite. Cytological examination showed numerous neutrophils and intracellular bacteria confirming the bacterial pyoderma **(photo 5)**.

The dog was treated systemically with oral antibiotics (cefalexin, 30 mg/kg/day) for one month and ciclosporine was pursued. A reinforced topical treatment was advised with twice a week shampooing and once daily application of a spray containing antiseborrheic and keratoregulating agents (Zincoseb® spray). At follow-up after one month skin lesions were markedly improved. Pruritus disappeared, bacterial lesions were gone, hair began to regrow and scaling was decreased by about 50%. Ciclosporine was continued as the only systemic medication and topical therapy with both the shampoo and the spray were carried on as previously. After three months, the dog was considered stabilized with good hair regrowth and nearly no scaling nor seborrhea **(photos 6 &7)**.

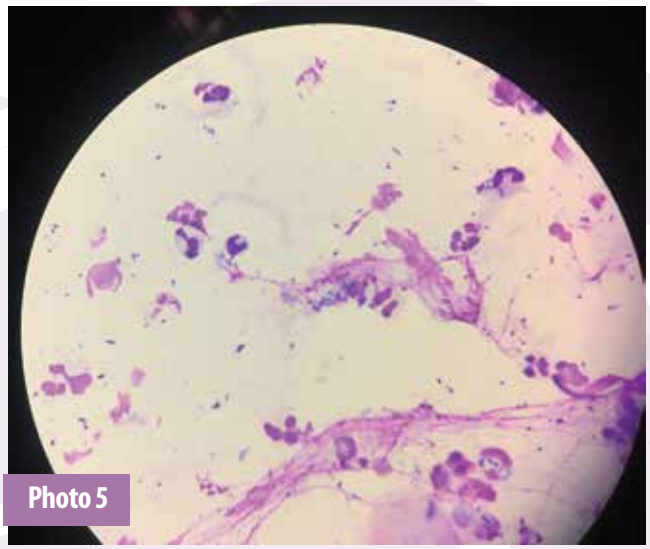


Photo 5

Photo 6

Photo 7

CASE 2

A localized pruritic and seborrheic dermatitis of the neck

A 2 years old, male intact Chinese sharpei is presented at the dermatology referral service for a pruritic disease localized on the neck.

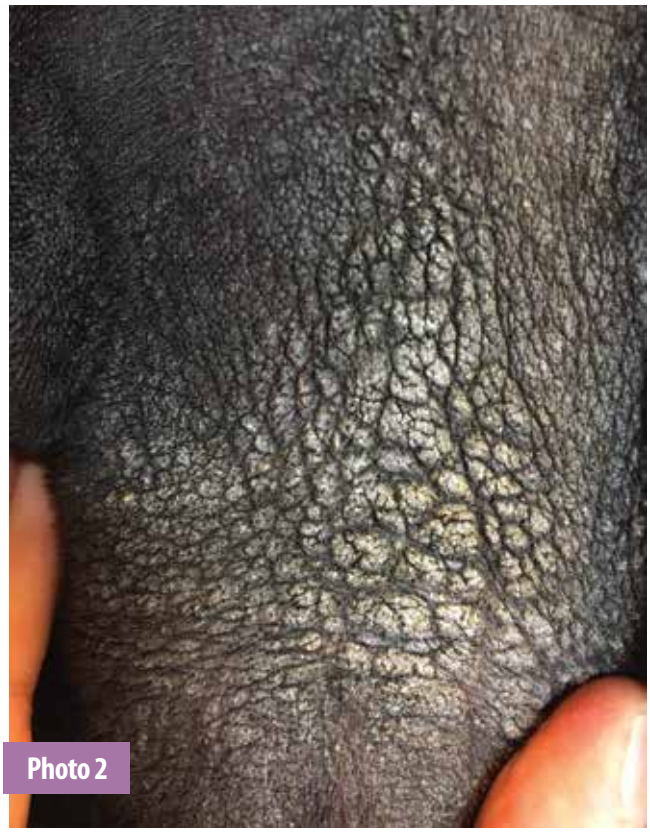
The dog lives in an appartement with another sharpei, is fed high quality food and vaccinations and deworming are regularly performed. Antiflea treatment consists of a montly application of a fipronil containing spot-on on both dogs.

Dermatosis was first noticed three months prior to referral. The owner reports initially a bad smell and pruritus localized on the neck. Skin lesions tended to extend and motivated a consultation to a general veterinarian who prescribed topical shampoo with 2% miconazole and 2% chlorhexidine and 10 days of oral antibiotic (amoxycillin) without any improvement. A short course of injectable steroids was then decided, which allowed an improvement of the pruritus but lesions were still present and dry seborrhea developped.

At clinical presentation, skin lesions were localized to the ventral part of the neck. There was a slight erythema, marked alopecia, a dry seborrhea with lichenification and large adherent scales **(photos 1&2)**.

Differential diagnosis included : localized sarcoptic mange, demodicosis, bacterial overgrowth syndrome, *Malassezia* overgrowth syndrome, « head and neck dermatitis » (a localized form of atopic dermatitis) and less likely contact dermatitis due to the collar (as no lesions were noticed on the lateral and dorsal part of the neck).

Photo 1



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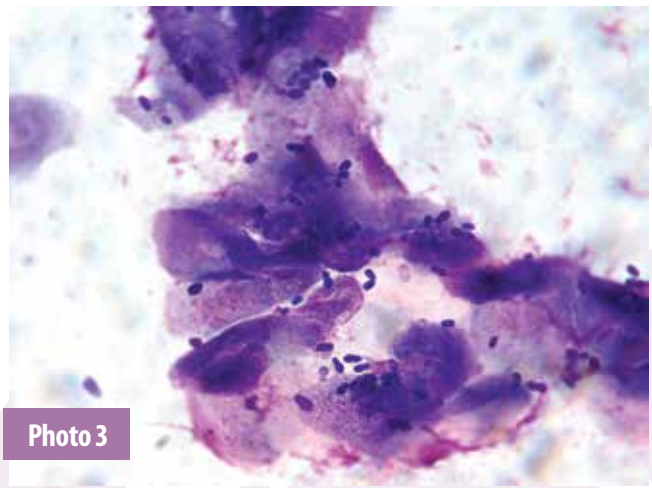


Photo 3

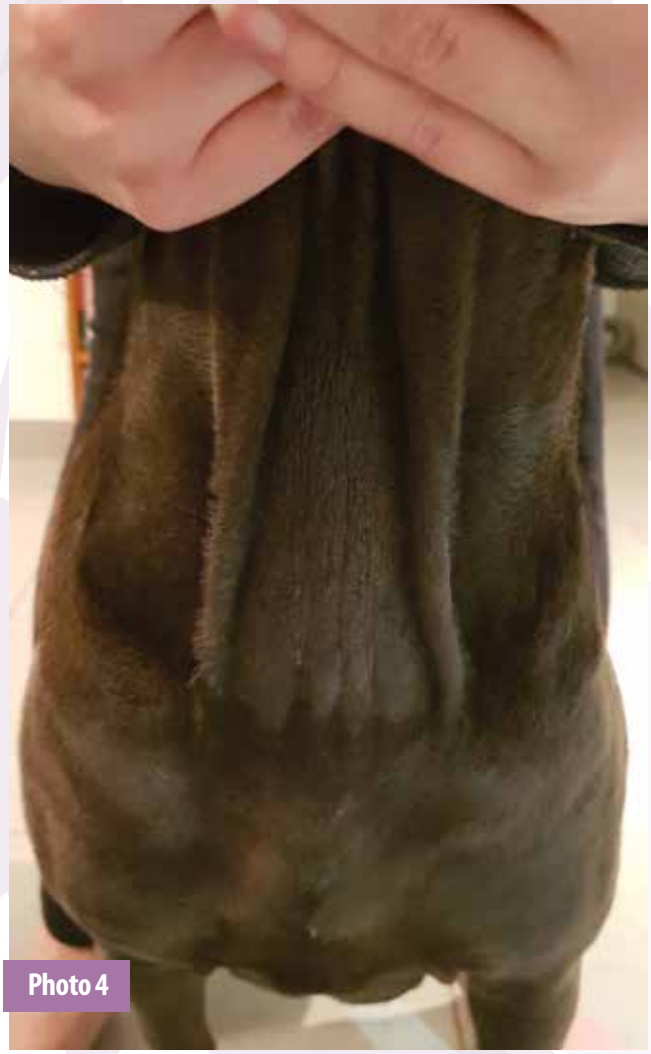


Photo 4

Photo 5

Skin scrapings showed no parasite. Serology for specific anti-Sarcoptes IgG was negative. Cytological examination using the tape strip technique showed no bacteria but a marked proliferation of *Malassezia pachydermatis* yeasts **(photo 3)**.

A systemic treatment with itraconazole (5 mg/kg/day) was decided associated with the topical every other day application of a spray containing anti-seborrheic and antifungal agents (Zincoseb® spray). After one week the owner reported only a mild improvement and he was advised to apply the spray every day instead of every other day. One week later he reported that the pruritus was less important and that the skin was smoother with the bad smell gone. At follow-up visit after one month, no pruritus was noted and the skin was considered as nearly normal **(photos 4 & 5)**. Systemic treatment was stopped and the spray was continued twice a week. One month later, as no lesion nor pruritus relapsed, the owner declined further investigation and prefered to continue the topical treatment as a preventive measure. Three months later the dog was still considered under control.

CASE 3

A localized pruritic and seborrheic dermatitis of the face

A 4 years old, male intact Chinese sharpei is presented at the dermatology referral service for a pruritic disease localized on the face.

The dog has been acquired at three months of age, and no previous skin disease is reported. He is living in an urban environnement, with an in contact cat (with no skin disease). Vaccination, anti-flea and tick treatments and deworming are regularly realized.
Skin lesions appeared two months previously. The owner reports initially a blepharitis and then an extension of the lesions on the folds of the face. Pruritus was initially mild but tended to increase with time and face rubbing developed which was the reason for the consultation to the dermatologist.

Previous therapies consisted only of a topical application of an antiseptic solution, without any improvement.

Lesions are confined on the face. Erythematous and seborrheic lesions, associated with alopecia are noted on the eyelids. Furthermore, a marked and severe intertrigo is observed : on the folds, one can note alopecia, lichenification, hyperpigmentation, scales and a purulent and malodorous exudate **(photos 1 & 2)**. Pruritus is severe with face rubbing during the clinical examination.

Main differentials diagnosis included demodicosis, bacterial overgrowth syndrome, *Malassezia* overgrowth syndrome, bacterial pyoderma, allergic dermatitis (eg atopic dermatitis), and anatomic defect (intertrigo).
Complementary examinations included skin scrapings (no parasite), cytological examination (numerous cocci adherent to corneocytes, no *Malassezia pachydermatis*, no inflammatory cells **(photo 3)**. A Schirmer test was considered as normal (tear production of 12).
A tentative diagnosis of intertrigo complicated by bacterial overgrowth syndrome was made.

Treatment was decided with systemic antibiotics (cefalexine, 20 mg/kg BID) and topical cleaning of the lesions with a gentle soap (Zincoseb® shampoo) followed by the topical application of a spray containing antiseborrheic, rehydrating and antiseptic agents (Zincoseb® spray) every other day for 3 weeks.

At follow-up, a marked improvement was noted with disappearance of the scales, less hyperpigmentation and lichenification **(photo 4)**. Topical treatment was continued alone with twice a week application of the spray. No relapse was noted after two months.

Photo 1

Photo 2

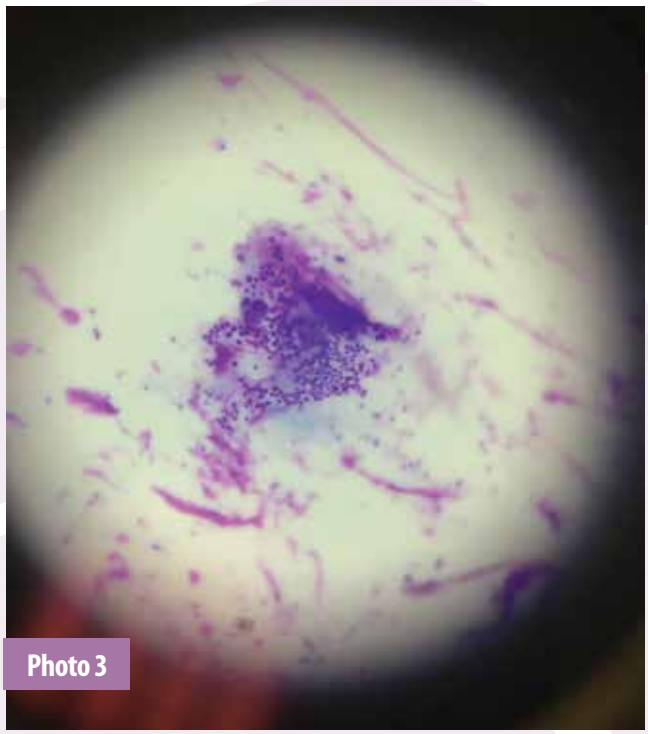


Photo 3

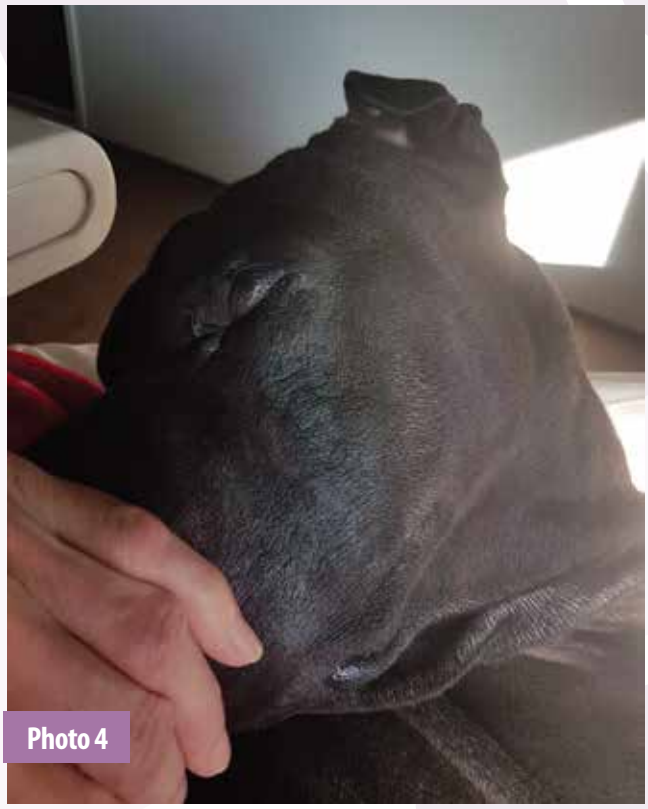


Photo 4

CASE 4

A localized pruritic seborrhea of the ear pinnae

A 3 years old, male intact Border Colley is presented for a refractory pruritic dermatitis localized on the right ear pinnae.

The dog lives in a suburban area in a house with a garden, is correctly vaccinated and dewormed. Antiparasitic treatment consists of a deltamethrin impregnated collar to prevent leishmaniosis. He is fed with high quality dry food.

The dermatosis began suddenly, 3 months prior to referral. Lesions initially consisted of a slight erythema and pruritus, rapidly complicated by scaling and seborrhea confined to the right ear pinnae. A short course of oral corticosteroids markedly improved the pruritus but the scaling and keratoseborrheic disorder increased. 15 days of cefalexin (30 mg/kg/d) was tried without success and the dog was referred for a secondary advice.

On clinical presentation skin lesions are exclusively located on the right ear pinnae. They consist of a focal alopecia on the external part of the pinnae, associated with a marked seborrhea and both large and small scales. On the inner part, a slight erythema and some papules are observed **(photos 1,2&3)**.

Main differential diagnosis for this localized pruritic dermatitis is sarcoptic mange. Therefore multiple skin scrapings were realized and allowed the visualization of a Sarcoptes scabiei var. canis mite **(photo 4)**.

Photo 1

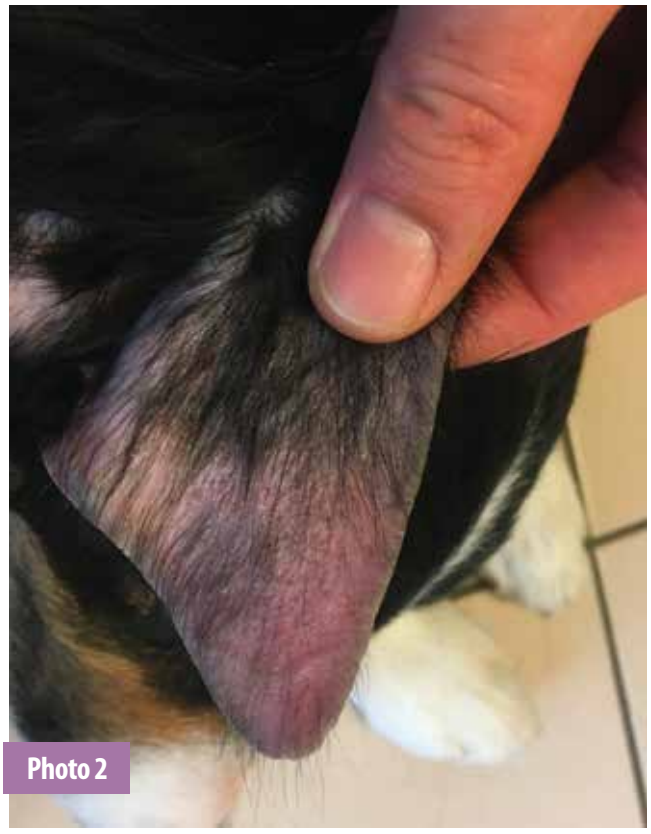


Photo 2

Photo 3

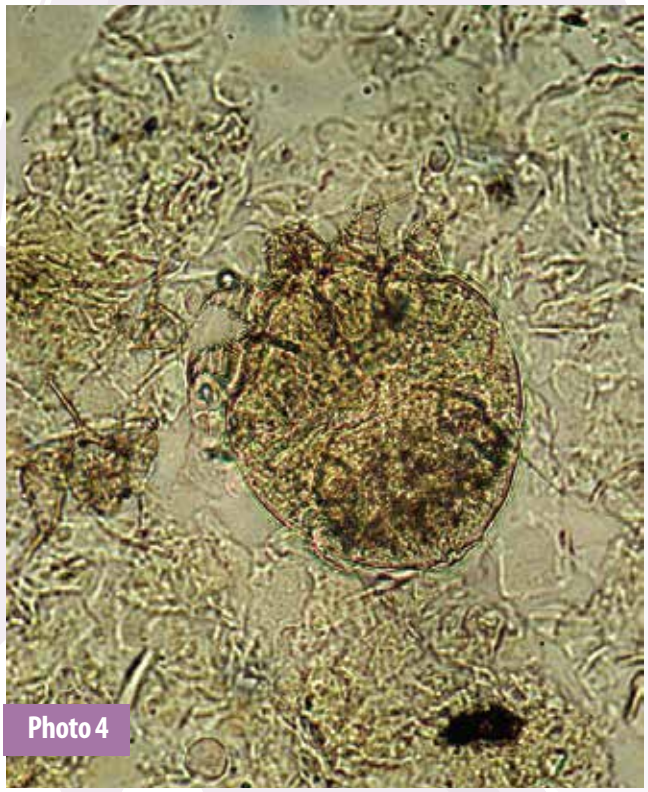


Photo 4

Dog was treated systemically with a combination of sarolaner (one administration with food) and oclacitinib (0.4 mg/kg BID for one week), associated with the topical application once a day of a spray containing antiseborrheic and hydrating agents (Zincoseb® spray).

After one week, the owner reported a complete normalization of the pruritus and a moderate improvement in the scaling. After two weeks, hair began to regrow and scaling was markedly improved. At follow-up after 4 weeks, a complete healing was observed with good hair regrowth and no pruritus **(photos 5, 6 & 7)**. A second administration of sarolaner was prescribed and the topical spray was applied twice a week for one more month then stopped. No relapse was reported after 6 months.

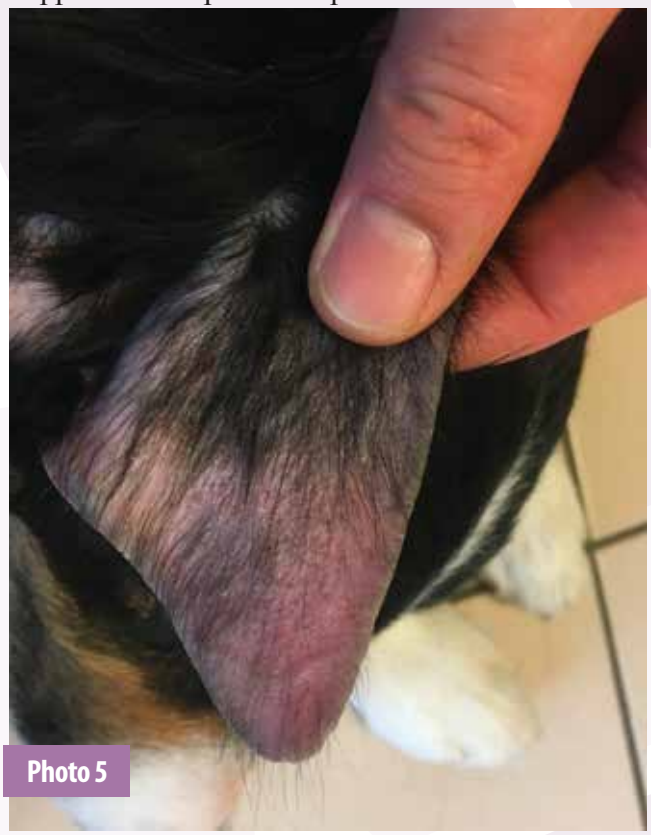


Photo 5

Photo 6

Photo 7

CASE 5

Lichenoid dermatosis in a dog

A male Cavalier King Charles, 7 years old, is presented for localized lesions on the abdominal area associated with pruritus.

The dog is vaccinated, fed with high quality dry food, and regularly treated against fleas by the monthly application of a spot-on containing permethrin and imidacloprid. He lives in a house with free access to a garden.

The dermatosis began 3 weeks earlier. Skin lesions were initially located on the ventral abdomen and appeared suddenly. No pruritus was noted at beginning but as the lesions extended, some licking appeared. No treatment except gentle cleansing with a disinfectant (povidone iodine solution) was done prior to the consultation.

Clinical examination revealed 3 lesions (average size 2/3 centimeters in diameter) localized on the abdominal area. They consisted in a peripheral erythematous border with scales and crusts and a central hyperpigmented and lichenified zone **(photo 1)**.

Complementary examinations included scrapings and scotch tests to exclude demodicosis, fungal culture to exclude dermatophytosis, cytological examination to exclude bacterial pyoderma (no neutrophils observed and only a few coccis), biopsies for histopathological examination which showed orthokeratotic hyperplasia, and a dense subepidermal infiltration with lymphocytes and plasma cells without neutrophils nor interface interface dermatitis, compatible with a lichenoid "psoriasiform" dermatosis **(photo 2)**.

Photo 1

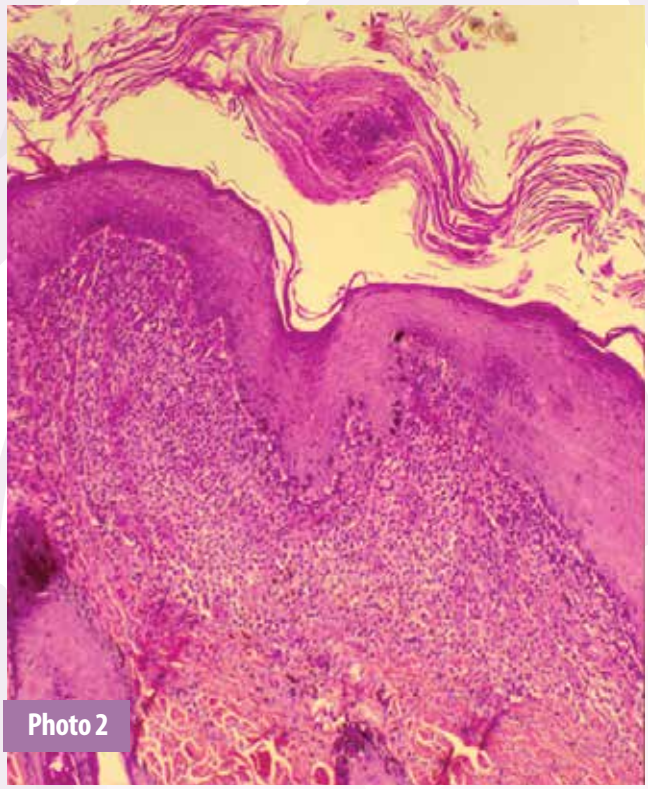


Photo 2

Treatment was realised with Zincoseb® spray used alone, once daily application on the lesion. Owners were asked to brush the area gently and then spray the product. One week later, at the time when the histopathological results were available, they mentioned a good improvement. At follow-up visit after one month a complete healing was observed **(photo 3)**.

Photo 3

CASE 6

A case of canine acne

A cross-breed, 7 years old, neutered female, is presented for a chronic disease affecting the chin.

No other dermatological condition is described. The condition evolves since 3 months. Initially the lesions were not pruritic but the owner mentioned that face rubbing began a few weeks before and increased slightly, which motivated the consultation.

The dog is vaccinated, fed an home made diet, and correctly treated against fleas and ticks with a monthly application of a spot-on product.
A treatment was previously performed with a 10 days course of antibiotics (amoxycillin- calvulanic acid 25 mg/kg/day for 10 days) without improvement. No other therapy was performed before the referral.

Lesions are localized to the chin. They consist of a slight erythema, lichenification, hyperpigmentation,

Results

scaling, and a keratoseborrheic dermatosis with follicular plugs and some comedones **(photo 1)**. The dog is otherwise healthy.

Differential diagnosis includes chin acne, demodicosis, bacterial pyoderma, less likely *Malassezia dermatitis*, zinc-responsive dermatosis or dermatophytosis.

Skin scrapings did not show any *Demodex* mites. Cytological examinations using the scotch-tape method did not reveal any proliferation of bacterial or fungal micro-organisms (coccis and *Malassezia pachydermatis* were indeed observed but considered to be in the normal range). A fungal culture was negative. A presumptive diagnosis of chin acne was made and the owner declined biopsies for histopathological examination.

A topical treatment alone was decided using once a day topical application of a spray containing antiseborrheic and keratoregulating agents (Zincoseb® spray). The owner was instructed to apply the product and to gently rub it onto the skin. No other treatment was allowed.

At follow-up after 14 days, a marked improvement was observed : skin smoothened, scaling was completely resolved and hair began to regrow **(photo 2)**. The owner mentionned that pruritus was improved by about 50%.

A secondary follow-up after 28 days showed a complete resolution of the scaling and the seborrheic disorder, lichenification was less pronounced and hair continued to regrow **(photo 3)**.

The owner was instructed to continue with a pro-active treatment using the spray twice a day locally. After 3 months, no relapse was noted.

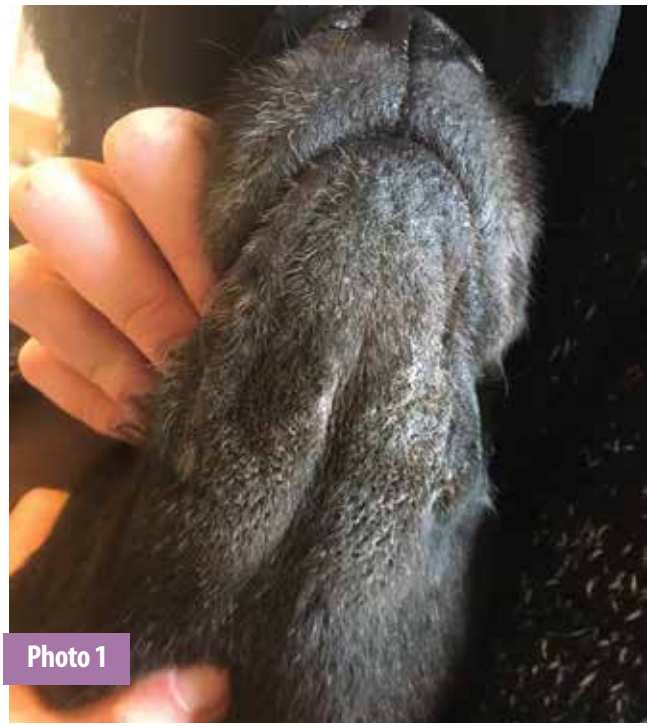


Photo 1

Photo 2

Photo 3

CASE 7

A male, 12 years old, crossed German sheperd dog, is presented for the acute development of pruritic plaques.

The dog lives in a house with a free access to a garden. He is vaccinated, correctly dewormed and treated regularly against external parasite with the monthly application of a spot-on product containing fipronil. He suffered in the past from various episodes of diarrhea, diagnosed previously as an inflammatory bowel disease and is treated with an hypoallergenic food and the daily administration of prednisolone at a dosage of 0,5 mg/kg.

Skin lesions began one month before referral. The owner reports the appearance of multiple plaques, initially located on the abdomen, associated with a pruritus. Polyuro-polydipsia is associated.

At clinical examination, the dog is pot bellied and various skin lesions are noted : scaling, numerous pinkish plaques located on the abdomen and on perianal area, atrophy of the skin and some phlebectasia **(photos 1, 2 & 3)**.

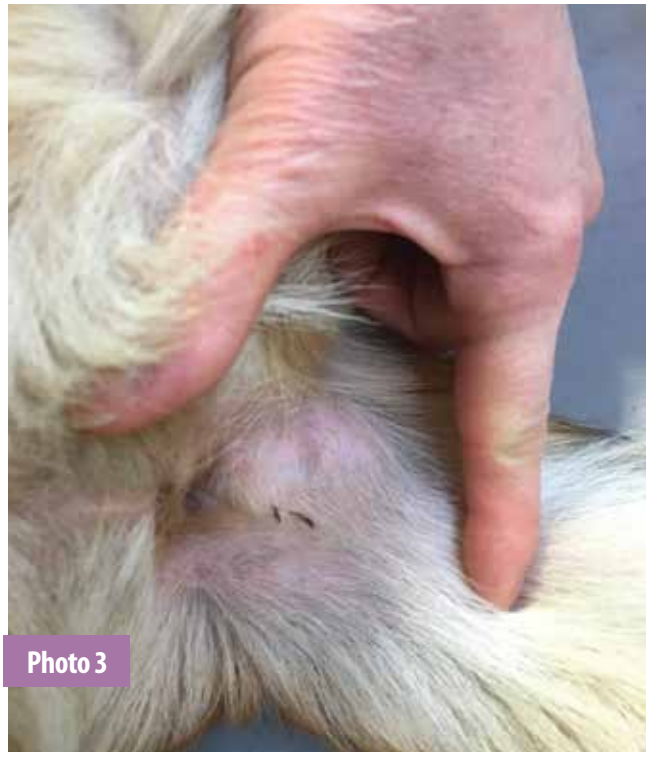
Main differential diagnosis consists in calcinosis cutis secondary to iatrogenic Cushing syndrome. Less likely a neoplasm or pseudoneoplasm may be considered.

Cytological examination by fine needle aspiration of one plaque did not show any inflammatory cell, but some refringent and very basophilic material compatible with calcium salts **(photo 4)**. An ACTH stimulation test confirmed an hypocortisolemia before and after stimulation.

Prednisolone was stopped and the dog was treated with the daily application of a spray containing rehydrating and antiseborrheic agents (Zincoseb® spray). After one week, a marked improvement was noted with less plaques and a better looking skin **(photos 5 & 6)**. Two weeks later the improvement continued and the spray was continued twice a week **(photos 7 & 8)**.

Photo 1

Photo 2



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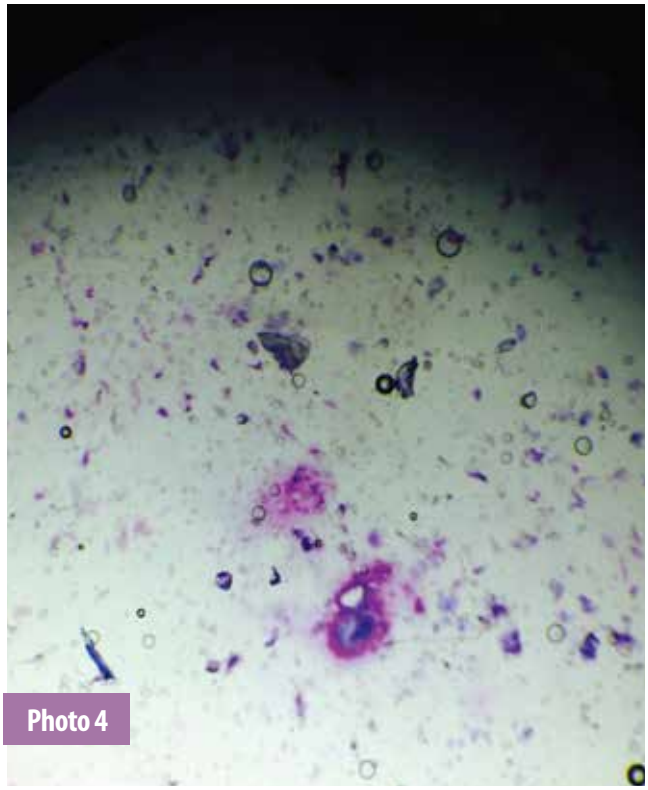

Photo 4

Photo 5

Photo 7

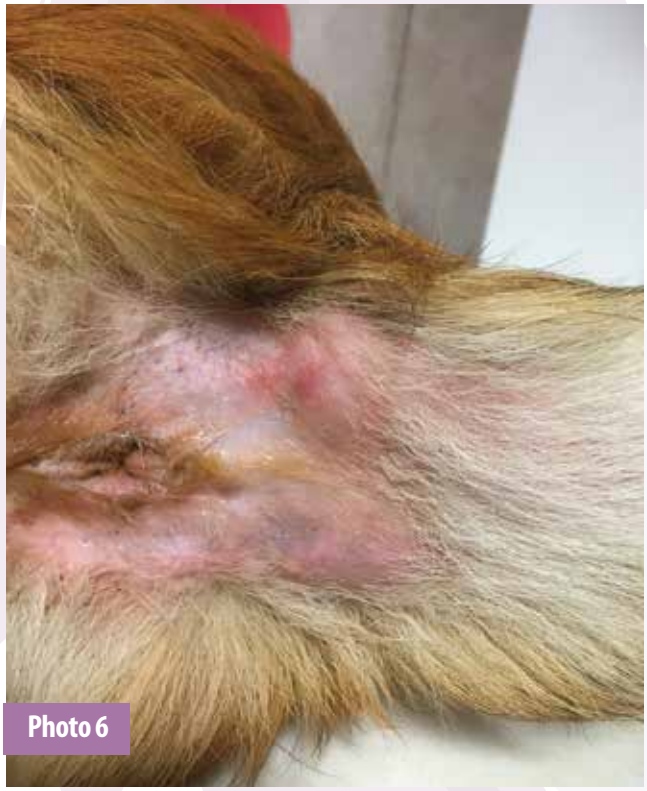

Photo 6

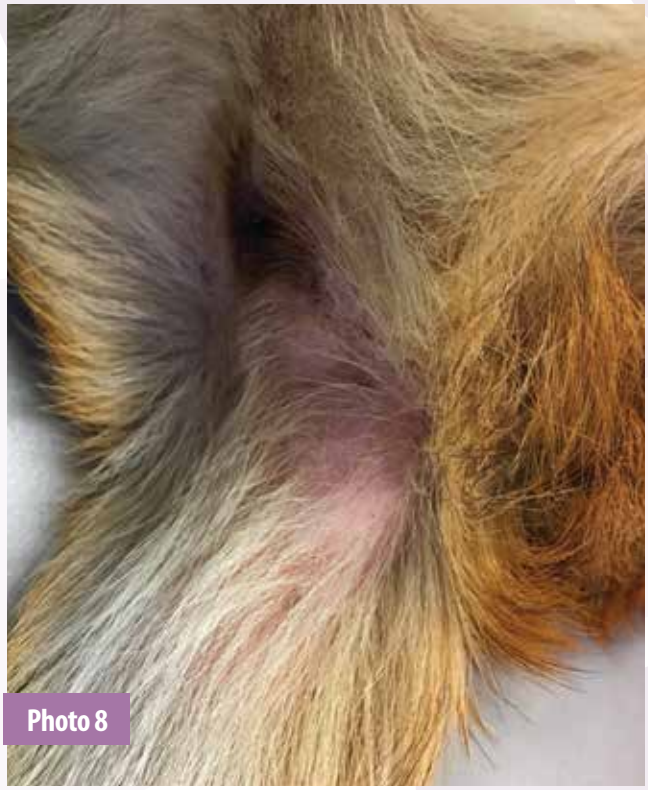

Photo 8

CASE 8

A 2 years old, female Cavalier King Charles is referred for a scaling and seborrheic dermatitis without pruritus.

The dog lives in an appartement in french Brittany. She is vaccinated, dewormed and treated against ectoparasites with the administration of fluralaner every three months orally. Skin lesions were noticed as soon as 4 months of age, and consisted initially in a mild scaling. No pruritus was observed. As the condition worsened and the scaling increased, the owners consulted their veterinarian who suspected a flea allergy dermatitis and prescribed an oral antiflea therapy and a short course of oral antibiotics without any improvement.

At clinical examination the dog is in good general health. Skin lesions consist of dry and dull hair and a generalized scaling, with pityriasiform scales, and a diffuse hypotrichosis. Some scales are whitish, very superficial and others are brown, larger and more adherent to the skin and to the hairs **(photos 1 to 3)**. Hyperkeratosis of the footpad margins is associated on the 4 legs.

Differential diagnosis included cheyletiellosis, leishmaniasis and an hereditary cornification disorder such as an ichtyosis. Allergic dermatitis was not considered as the condition was not pruritic. Endocrinopathy such as hypothyroidism was unlikely as no systemic signs were associated.

Skin scrapings and scotch tests did not reveal any mite. Trichoscopy showed large and small adherent scales **(photo 4)**. Leishmaniasis serology was negative.

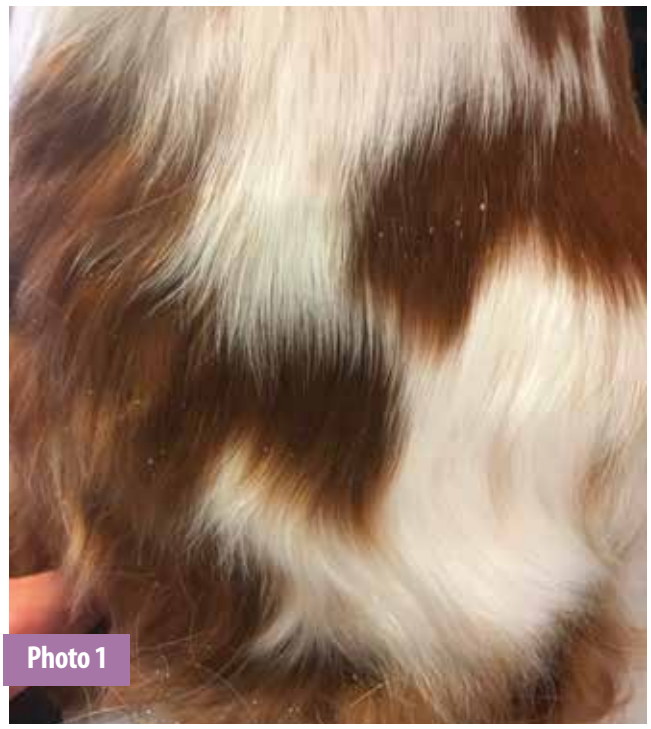

Photo 1

Photo 2

Photo 3

Photo 4

Dermatoscopy confirmed that the scales were very adherent to both the skin and hair **(photos 5 & 6)**. Biopsies were advised but the owner declined due to financial reasons. A tentative diagnosis of ichtyosis was made based on the history, clinical presentation and dermatoscopy findings.

The dog was treated with a combination of an antiseborrheic shampoo (Zincoseb® shampoo) once a week and the every other day application of a spray containing antiseborrheic agents (Zincoseb® spray). A follow-up telephone call after 3 weeks was scheduled and the owner reported that the scales were less prominent and the hair appeared more shiny. At follow-up visit after 6 weeks, the dog presented a nice and shiny coat, with nearly no scaling **(photos 7 & 8)**. Topical treatment was continued with once a week application of the shampoo and twice a week application of the spray and no relapse was reported after 3 months.

Photo 5

Photo 6

Photo 7

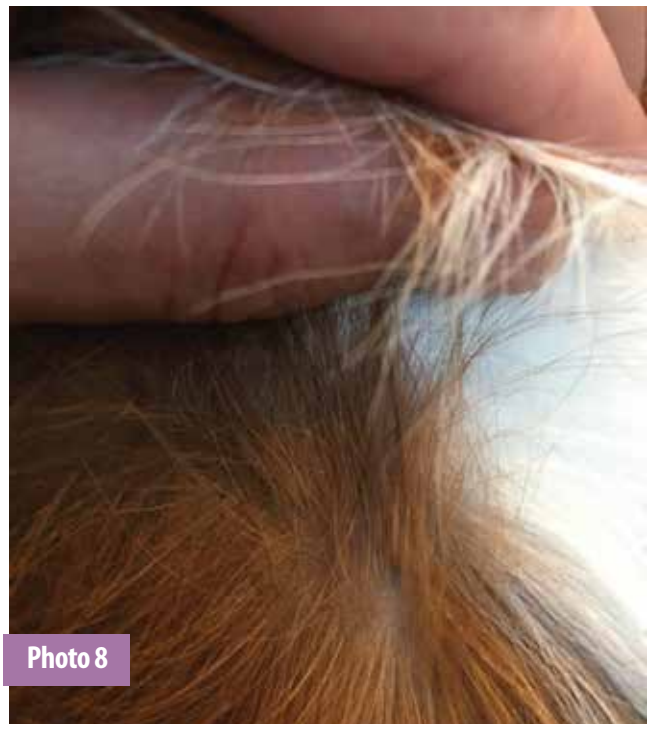

Photo 8

CASE 9

A case of pemphigus foliaceus in a dog

An Australian shepherd, male, intact, 2 years old, is presented for a severe pruritic chronic generalized dermatosis.

The dog lives in an house with a garden, is vaccinated, correctly treated against fleas (monthly administration of afoxolaner), and fed with a high quality dry food.
The dermatosis began 6 months before referral, initially on the face with further worsening and extension to the thorax, abdomen and the feet. Various treatments have been previously administered (antibiotics, steroids, oclacitinib) without any improvement.

Physical examination revealed hyperthermia, lethargy, and severe skin lesions : multifocal alopecia, disseminated pustules and a severe erosive, scaling and crusting dermatosis on the face **(photo 1)**. Scrapings were negative. Cytology of intact pustules revealed non degenerated neutrophils and many acantholytic keratinocytes **(photo 2)**. No bacteria nor fungal organisms were observed. A fungal culture was negative. Histopathologic examination showed subcorneal acantholytic pustules **(photo 3)** and a diagnosis of pemphigus foliaceus was made.
Treatment included discontinuation of afoxolaner (which has been associated to drug-triggered pemphigus in one recent case), immunusuppressive therapy with prednisolone at 1 mg/kg BID and additional topical treatment with keratolytic and antiseborrheic shampoo on the body twice a week and an antiseborrheic spray on the face (Zincoseb® spray) as shampooing was unpracticle in this location.

Photo 1

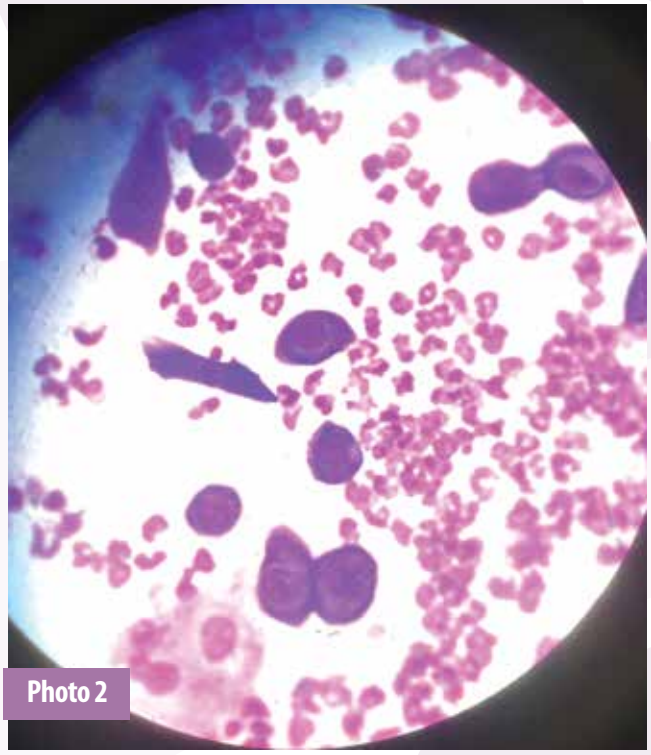

Photo 2

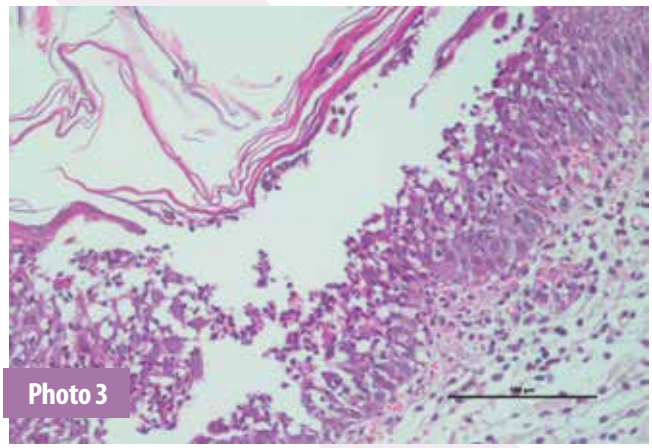

Photo 3

Follow-up after 15 days showed a marked improvement of skin lesions and pruritus **(photo 4)**. The dog was more alert, fever completely resolved. Treatment was continued unchanged. At a second follow-up 15 days later, no further pustules were noted and all inflammatory lesions were completely resolved. Scaling was markedely improved **(photo 5)**. Dosage of prednisolone was tapered to every other day then twice a week and topical therapies were pursued.

Photo 4

At the time of this writing, the dog is maintained in remission with these medications **(photo 6)**.

Photo 5

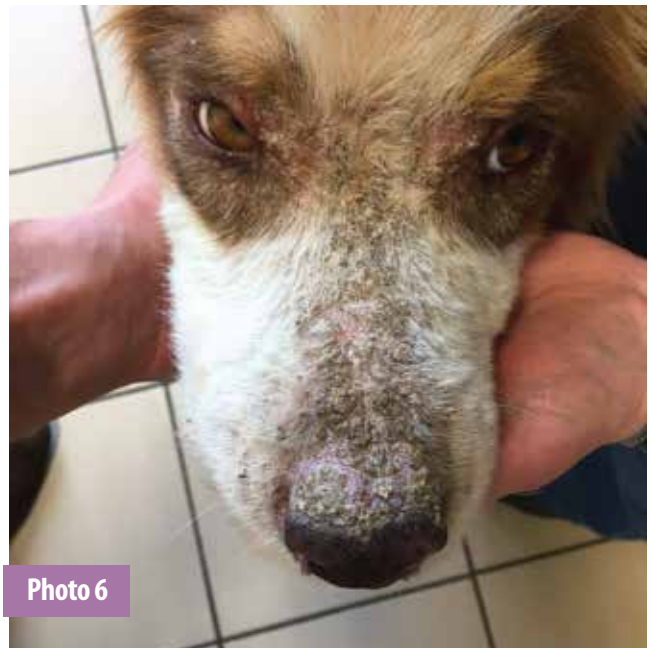

Photo 6

NOTES

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