CASEBOOK

CERTIFICATE IN VETERINARY DERMATOLOGY
**CONTENTS:**

<table>
<thead>
<tr>
<th>Case Number</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Canine Pyotraumatic Dermatitis</td>
<td>1</td>
</tr>
<tr>
<td>2. Laprine Psoroptic Mange</td>
<td>12</td>
</tr>
<tr>
<td>3. Canine Generalised Demodicosis</td>
<td>24</td>
</tr>
<tr>
<td>4. Feline Plasma Cell Pododermatitis</td>
<td>41</td>
</tr>
<tr>
<td>5. Canine Pemphigus Foliaceus</td>
<td>58</td>
</tr>
<tr>
<td>6. Canine Juvenile Cellulitis</td>
<td>76</td>
</tr>
<tr>
<td>7. Canine Atopic Dermatitis</td>
<td>90</td>
</tr>
<tr>
<td>8. Canine Sarcotic Mange</td>
<td>112</td>
</tr>
<tr>
<td>9. Porcine Dermatitis and Nephropathy Syndrome</td>
<td>126</td>
</tr>
<tr>
<td>10. Canine Hypothyroidism</td>
<td>143</td>
</tr>
</tbody>
</table>

**Total word count:** 9988
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>ELISA</td>
<td>enzyme-linked immunosorbant assay</td>
</tr>
<tr>
<td>FcεRIα</td>
<td>high affinity IgE receptor alpha-subunit</td>
</tr>
<tr>
<td>DHPPiL</td>
<td>Distemper, adenovirus type 2 (hepatitis), parvovirus, parainfluenza virus type 2, Leptospira canicola, Leptospira icterohaemorrhagiae</td>
</tr>
</tbody>
</table>
CANINE PYOTRAUMATIC DERMATITIS

BREED: Cocker Spaniel  
AGE: Four years  
SEX: Entire male  
WEIGHT: 15.6 kilograms

OWNER’S COMPLAINT:  
Intense pruritus affecting the right caudal metatarsus.

HISTORY:  
The dog presented with a 24-hour history of incessant chewing and resultant self-trauma of the right metatarsal area. There was no previous history of a dermatological problem and no flea control had been used on the dog.

PHYSICAL EXAMINATION:  
On general examination the anal sacs were full but not infected or impacted. Dermatological examination revealed a large amount of flea faeces in the coat (Figure 1.3). The caudal metatarsal area appeared to be painful and the hair in that area was matted with a suppurative discharge. On close examination of the affected site there was erythema, exudation and ulceration. After clipping it could be seen that the lesion had well demarcated margins (Figure 1.1,1.2).
DIFFERENTIAL DIAGNOSIS:

- Demodicosis
- Dermatophytosis
- Hypersensitivity
  - Atopy
  - Food hypersensitivity
  - Flea bite hypersensitivity
- Allergic contact dermatitis
- Irritant contact dermatitis
- Pyotraumatic dermatitis
- Pyotraumatic folliculitis
- Bacterial folliculitis
- Anal sac impaction/infection

LABORATORY AND OTHER DIAGNOSTIC TESTS:

Fleas and flea faeces were seen on coat brushings taken on a piece of damp white paper. Microscopic examination of skin scrapings taken in liquid paraffin was negative for ectoparasites. An acetate tape preparation was taken from the skin of the affected area and stained with a modified Wright’s stain (Diff Quik, Dado Behring AG). Microscopic examination revealed coccoid bacteria and neutrophils with intracellular bacteria.

DIAGNOSIS AND PROGNOSIS:

Pyotraumatic dermatitis associated with flea infestation. A good prognosis was given provided that flea control was maintained.
**TREATMENT:**

The area was clipped and cleaned with chlorhexidine 4% solution (Hibiscrub, SSL International PLC). An Elizabethan collar was used to prevent further self-trauma (Figure 1.4) and 0.5% fusidic acid/0.1% betamethasone gel (Fuciderm, Leo) was applied to the area. Cephalexin (Rilexine 300mg, Virbac) was given at a dose of 20mg/kg twice daily for two weeks. Fipronil spray (Frontline, Merial) was used for flea control and applied at a dose of 6mls/kg (4 sprays/kg).

**REINSPECTIONS AND FINAL OUTCOME:**

Three days later the dog was no longer pruritic and only a small area of erythema and crusting was present on the caudal metatarsus (Figure 1.5). Treatment was continued with the antibiotic/steroid gel and cephalexin for a further ten days.

On examination three weeks later there was complete resolution of clinical signs and no evidence of fleas (Figure 1.6). There has been no recurrence of the condition in the eighteen months following treatment.

**DISCUSSION:**

The clinical signs in this case are compatible with previous reviews of pyotraumatic dermatitis (PD) (Kunkle 1979, Mason 1991, Rosencrantz 2000). Long and densely coated breeds appear to be predisposed (Kunkle 1979, Ihrke 1996).

Diagnosis of PD is often based on history and clinical signs. Scrapings and cytology are used to rule out other causative factors (Rosencrantz 2000) as in
this instance. Histopathology was not performed, as this was the first occurrence of PD in this patient. However, Rienke and others (1987) have recognised a deep form of PD with suppurative, necrotising folliculitis. Histopathology should therefore be performed in a recurrent case.

PD may occur at flea feeding sites (Curtis 2001) and flea-bite hypersensitivity is a common cause of this condition (Scheidt 1988, Sousa and Medleau 1992). No further diagnostic tests to confirm flea bite hypersensitivity were conducted as most cases of PD respond to clipping, cleaning and topical treatment irrespective of the underlying cause (Kunkle 1979, Mason 1991), as was seen here.

Elimination of the underlying cause is important in recovery and prevention of recurrence (Ihrke 1987). In this case there was rapid improvement with no recurrence after eighteen months, hence it is likely that flea-bite hypersensitivity was the initiating cause.

Topical glucocorticoids have been recommended for treatment of PD (Ihrke 1980). Schroeder and others (1996) demonstrated that in cases of PD a topical antimicrobial or topical anti-inflammatory alone gave significantly poorer results when compared with a topical antimicrobial/anti-inflammatory combination.

Systemic antibiotics, although not always indicated in treatment of surface pyodermas (Mason 1991, Ihrke 1996), may be useful in some cases (Rosenkrantz 2000). *Staphylococcus intermedius* is the most common isolate.
in cases of canine pyoderma (Mueller and others 1998, Holm and others 2002). The presence of coccoid bacteria would suggest that *S. intermedius* was involved in this case. Cephalexin was selected on the basis of its efficacy against *S. intermedius* (Phillips and Williams 1984, Medleau and others 1986),

Systemic glucocorticoids have been recommended in cases of PD to break the "itch-scratch cycle" (Kunkle 1979, Ihrke 1996), however, the use of topical glucocorticoids, an Elizabethan collar and flea control was successful in this case and further treatment was not required.
REFERENCES:

*Veterinary Medicine* **83**, 984 – 1004


Figure 1.1: Erythema, exudation and ulceration of the right caudal metatarsus at initial presentation – post clipping

Figure 1.2: The same lesion as Figure 1.1 showing very well demarcated margins
Figure 1.3: Flea faeces present in the coat of the dog at initial examination

Figure 1.4: Elizabethan collar fitted to the dog to prevent further self trauma of affected area
Figure 1.5: Right hind leg after three days of topical and systemic treatment showing a reduction in erythema and exudation

Figure 1.6: Resolution of clinical signs with new hair growth, three weeks after cessation of topical and systemic treatment
**LAPRINE PSOROPTIC MANGE**

**BREED:**  Lop rabbit

**AGE:**  Three years nine months

**SEX:**  Entire male

**OWNER’S COMPLAINT**

Pruritus of both ears accompanied by a large amount of debris.

**HISTORY:**

There had been pruritus of both ears a week prior to presentation. Crusting had been evident in both ears for the past two weeks. No lesions were noticed on in-contact humans.

**PHYSICAL EXAMINATION:**

On examination there were dry, haemorrhagic crusts on the concave surface of both pinnae (Figure 2.1, 2.2). The convex pinnae were unaffected. The skin underlying the crusts was ulcerated and erythematous and the rabbit resented close examination of the ears. On close examination of the affected areas with a hand lens, mites could be seen. No other abnormalities were found on general or dermatological examination.
DIFFERENTIAL DIAGNOSIS:

- Psoroptic mange – *Psoroptes cuniculi*
- Cheyletiellosis- *Cheyletiella parasitivorax*
- Sarcoptic mange – *Sarcoptes scabiei*
- Fur mite infestation – *Leporacarus gibbus*
- Dermatophytosis
- Otitis externa

LABORATORY AND OTHER DIAGNOSTIC TESTS:
Microscopic examination of crusts from both ears, mounted in liquid paraffin, revealed a large number of *Psoroptes cuniculi* mites and eggs (Figure 2.3, 2.4). There was no evidence of *P. cuniculi* or other mites on microscopic examination of acetate tape preparations taken from a number of areas including abdomen, dorsum and neck.

DIAGNOSIS AND PROGNOSIS:
Otitis externa caused by *Psoroptes cuniculi*. A good prognosis was given.

TREATMENT:
The ears were gently cleaned with saline and some of the crusts were removed. A topical treatment using a combination of diethanolamine fusidate, framycetin sulphate, nystatin and prednisolone (Canaural, Leo Laboratories Limited) was applied to provide relief from the pruritus.
Ivermectin (Panomec, Merial) was administered by sub-cutaneous injection at a dose of 400micrograms/kg bodyweight every ten days on two occasions.
Informed consent was obtained from the owner for the use of the unlicensed drug, ivermectin.

The hutch was sprayed with a combination permethrin and pyriproxyfen spray (Indorex, Virbac).

**REINSPECTIONS AND FINAL OUTCOME:**

Three days later the pruritus had ceased. After ten days there was a marked improvement with reduction in the crusts (Figure 2.5, 2.6). After three weeks there was no evidence of mites or eggs on skin scrapings taken from the ears and no crusts were present in either ear (Figure 2.7, 2.8).

**DISCUSSION:**

Heavy crusting of the concave pinnae and pruritus as seen in this case are consistent with previous reports of ear mite infestation (Brokis 1979, Saunders 1979, Scarff 2000, Jenkins 2001, Harcourt Brown 2002). The condition is usually bilateral, however, unilateral disease has been reported (Scarff 1991, Harcourt-Brown 2002). Clinical signs are believed to be the result of a hypersensitivity reaction to mite antigens hence infestation in some animals may not cause clinical disease (Pruett and others 1996, Stromberg and Fisher 1996, Uhlíř 1991).

Although infestation with this mite is usually confined to the ear, as in this case, they can infest other parts of the body including perineal skin folds (Yeatts 1994), ventral abdomen (Cutler 1998, Brokis 1979) and dewlap (Jenkins 2001) possibly during grooming (Cutler 1998).
Diagnosis of this condition is not difficult as these mites are quite large (0.7mm long) and can often be seen with the naked eye, as in this case. Microscopic examination will allow confirmation of the species (Wall and Shearer 2001).

Ivermectin, although not licensed for use in rabbits, has been found to be a safe and effective treatment for ectoparasites in this species (Curtis and others 1990, Cutler 1998). Recommended doses vary from between 100-400 micrograms/kg for one to three treatments at intervals of ten days to two weeks (Pandey 1989, Bowman and others 1992, Wright and Riner 1985, Curtis 1990, Scarff 2000). In this case the highest recommended dose on two occasions proved effective.

Fipronil (Frontline spray, Merial) has been used for the treatment of this condition (Cutler 1998), however, studies indicate that this drug is toxic in rabbits (Cooper and Penaliggon 1997). A recent unpublished study demonstrated the safety and effectiveness of a single dose of selamectin at 6mg/kg and 18mg/kg in rabbits¹.

Complete removal of the crusts should not be attempted as it is painful (Scarff 2000, Jenkins 2001), however, it may be useful to remove the most severe crusts and scabs to prevent secondary bacterial infection and fly strike (Cutler 2001).

1998) as was done here. Use of a combination product, as in this case, can reduce inflammation and loosen the crusts (Jenkins 2001).

Environmental control is important as the mite can survive for up to 21 days off the host especially in conditions of low temperature and high humidity (Arlian and others 1981). Environmental insecticides are recommended for removal of all life stages (Wall and Shearer 2001) and were used in this case to spray the hutch and surrounding area.
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Figure 2.1: Haemorrhagic crusts and erythema of the concave aspect of the left pinna at initial presentation

Figure 2.2: Concave aspect of the right pinna of affected rabbit at initial presentation showing haemorrhagic crusts and erythema
Figure 2.3: Photomicrograph of adult *Psoroptes cuniculi* found in the crust taken from the pinna of affected rabbit.

Figure 2.4: Photomicrograph of crust from the pinna of affected rabbit showing *Psoroptes cuniculi* eggs.
Figure 2.5: The left ear, ten days after starting treatment with ivermectin, showing reduction in crusts and erythema.

Figure 2.6: Reduction in crusts and erythema of the right pinna ten days after starting treatment with ivermectin.
Figure 2.7: Resolution of lesions of the left pinna one week after second ivermectin treatment

Figure 2.8: Resolution of lesions of right pinna one week after second ivermectin treatment
CANINE GENERALISED DEMODICOSIS

**BREED:** Border collie  
**AGE:** Eighteen months  
**SEX:** Entire female  
**WEIGHT:** 14 kilograms

**OWNER’S COMPLAINT:**
Hair loss around the mouth and scabs on the feet. Intense pruritus of the face, feet and groin.

**HISTORY:**
Alopecia and erythema around the mouth had been present for twelve months. Four months prior to presentation the alopecia and erythema had become more extensive, involving the feet and abdomen. Treatment with methyl prednisolone acetate injections and oral cephalexin had been given intermittently for three months. During that time there had been a worsening of the clinical signs with crusting, exudation and pruritus of the feet and abdomen.

**PHYSICAL EXAMINATION:**
General physical examination revealed a generalised peripheral lymphadenopathy. The dog was depressed and reluctant to have the feet examined.

On dermatological examination there was patchy alopecia and erythema of the lips (Figure 3.1). Papules, pustules and comedones were present on the
abdomen (Figure 3.2). Areas of alopecia, crusts, exudation and erythema were present on all feet (Figure 3.3, 3.4).

**DIFFERENTIAL DIAGNOSIS:**

- Deep pyoderma secondary to:
  - Demodicosis
  - Dermatophytosis
  - Food hypersensitivity
  - Atopy
  - Endocrine disease e.g. hypothyroidism, hyperadrenocorticism

- Idiopathic pyoderma

- Pemphigus complex

- Systemic Lupus erythematosus

- Deep fungal infection

**LABORATORY AND OTHER DIAGNOSTIC TESTS:**

Microscopic examination of skin scrapings and hair plucks taken from the feet and face and mounted in liquid paraffin revealed *Demodex canis* mites and eggs (Figure 3.5-3.7). A needle aspirate was taken from an intact abdominal pustule and stained with a modified Wright’s stain (Diff Quik, Dado Behring AG). Coccoid bacteria and neutrophils with intracellular bacteria were found on microscopic examination (Figure 3.8). Coccoid bacteria, degenerate neutrophils and *D. canis* mites were found on microscopic examination of
acetate tape preparations taken from the feet and stained as above. A fungal culture was negative after 14 days.

**DIAGNOSIS AND PROGNOSIS:**
Generalised demodicosis (GD). The prognosis in the case, provided that no further glucocorticoids were given, was fair.

**TREATMENT:**
The dog was bathed weekly in 2.5 % benzoyl peroxide shampoo (Paxcutol, Virbac). Once the coat was dry a 0.05 % solution of amitraz (Aludex, Intervet) was applied all over the dog. Cephalexin (Ceporex 250mg, Schering-Plough Animal Health), at a dose of 20mg/kg twice daily was given for five weeks.

**REINSPECTIONS AND FINAL OUTCOME:**
After five weeks of treatment there was a marked improvement. The dog’s feet seemed to be less painful and there was a reduction in exudation and crusting (Figure 3.9). There were no pustules on the abdomen (Figure 3.10). On microscopic examination of skin scrapings taken from the mouth and mounted in liquid paraffin, *D. canis* mites were still present. After a further six weeks of treatment with amitraz there was good hair growth around the mouth and on the feet. Skin scraping at that time yielded only fragments of mites. A microscopic examination of skin scrapings performed after a further four weeks of weekly amitraz showed no mites. All hair had regrown and there was
no evidence of pyoderma (Figure 3.11 – 3.13). Eighteen months after cessation of treatment there has been no recurrence of the condition.

**DISCUSSION:**

Canine demodicosis can present as a generalised or localised condition. It may be either juvenile or adult in onset. Juvenile onset demodicosis, as in this case, has a better prognosis than adult onset demodicosis, which is often associated with other primary systemic diseases such as hyperadrenocorticism (Henfrey 1990, Lemarié 1996).

Localised demodicosis is a mild clinical disease, most cases resolving without treatment. However, the generalised form is a severe disease with a guarded prognosis (Scott 1979b, Folz 1983, Henfrey 1990, Lemairé 1996). The history in this case would suggest that in the twelve months prior to presentation the localised form was present around the mouth. Examination of skin scrapings of the lips at initial presentation yielded *D. canis* mites.

Demodicosis has been induced in dogs and horses following the use of glucocorticoids (Scott and White 1983, Sundberg and others 1994). In a retrospective study of adult onset demodicosis (Lemarié and others 1996) 24% of the dogs had been treated with glucocorticoids.

Evaluation of cellular immunity of dogs with GD has shown T cell suppression, which may be partly due to secondary pyoderma (Hirsh and others 1975, Barriga and others 1992, Lemairé and Horohov 1996). Treatment with glucocorticoids, which will suppress T-cell function (Papich and Davies 1998),
may, in this case, have predisposed the localised demodicosis to become generalised. Scott and others (1976) have described several dogs with localised demodicosis, which have converted to severe generalised demodicosis following glucocorticoid therapy.

Cytology indicated a secondary pyoderma. In a retrospective study of dogs with generalised demodicosis 60 – 70% of cases had pustular disease (Lemarié and others 1996). *Staphylococcus intermedius* is the most common bacterium isolated from cases of pyoderma associated with demodicosis (Kwochka and others 1985, Lemarié and others 1996).

Treatment in this case was directed at eradication of the mites and resolution of the secondary bacterial infection. Amitraz is the only licensed treatment for canine demodicosis. Various studies report widely varying rates of cure, using amitraz, from zero to 99% successful (Folz 1983, Scott and Walton 1984, Kwochka and others 1985). However, weekly treatment using a 0.05% solution, the concentration licensed for use in Europe, treatment success rates of up to 75% have been reported (Bussieras and Chermette 1986). Weekly application for twelve weeks was successful in this case.

Daily oral ivermectin has been used for treatment of generalised demodicosis (Ristic and others 1995, Guaguère 1998, Mueller and others 1999) with clinical cures of between 80-90%. It was not used here due to the good response to amitraz and the breed of the dog. Collies and their crosses are susceptible to the toxic effects of ivermectin due to a gene mutation (Paul and others 1987, Hopper and others 2002). This mutation results in a lack of P-glycoprotein,
which is needed for normal function of the blood-brain barrier (Pullion and others 1985, Mealey and others 2002).

Benzoyl peroxide shampoo has been reported to have a follicular flushing action, which enhances the removal of scale and reduces bacterial counts in hair follicles (Guaguère 1996). It is an effective antibacterial agent and has been used in deep pyodermas (Scott 1979a, Guaguère 1996). Thompson and Mandy (1976) saw a ‘dramatic’ improvement in the secondary pyoderma associated with GD and a decrease in mite numbers in dogs treated with benzoyl peroxide shampoo.

Evaluation of skin scrapings was used to determine the end point of treatment. Use of visual clinical improvement is an unreliable indicator of cure as some animals can be clinically normal for some time before elimination of mites, as was seen here (Paradis and Laperriere 1992, Miller and others 1993). Treatment should continue for four weeks after negative skin scrapings are obtained, as in this case (Ristic and others 1995, Medleau and others 1996).
REFERENCES:


Figure 3.1: Alopecia and erythema around the mouth of the dog at initial presentation

Figure 3.2: Close up of the lesions on the abdomen at initial presentation showing pustules, papules and comedones
**Figure 3.3:** Lesions present on one affected foot at initial presentation showing erythema, alopecia, crusts and exudation

**Figure 3.4:** Lesions present on one affected foot at initial presentation showing erythema, alopecia and crusts
Figure 3.5: Photomicrograph of adult *Demodex canis* mite seen on skin scraping taken from the dog (low power)

Figure 3.6: Photomicrograph of adult *Demodex canis* seen on skin scraping (high power)

Figure 3.7: Photomicrograph of *D. canis* egg seen on skin scraping taken from the dog (high power)
Figure 3.8: Photomicrograph of cytology of an aspirate taken from an abdominal pustule showing neutrophils and intracellular bacteria (modified Wright’s stain, high power)

Figure 3.9: The feet of the dog after five weeks of treatment with amitraz showing reduction of erythema, exudation and crusts
Figure 3.10: Early resolution of lesions on the abdomen after five weeks of treatment with amitraz.

Figure 3.11: Resolution of lesions around the mouth after four months of treatment with amitraz.
Figure 3.12: Resolution of lesions and hair regrowth on one affected foot after four months of treatment with amitraz.

Figure 3.13: General appearance of the dog after four months of treatment with amitraz showing resolution of the lesions.
BREED: Domestic shorthair

AGE: Eight years

SEX: Spayed female

OWNER'S COMPLAINT:
Swollen pads and apparent discomfort on walking. A mass of tissue was protruding from the central pad of the left front foot.

HISTORY:
For the past four years there had been intermittent swelling of the central pads of all feet and the cat had been reluctant to walk on hard surfaces. The swelling of the footpads seems to wax and wane unpredictably but the condition becomes worse after feeding fish. No flea control has been used. Previous treatment with prednisolone was ineffective.

PHYSICAL EXAMINATION:
Dermatological examination revealed swelling of all four central pads. The central pad of the left front foot was ulcerated and reddish granulation tissue was protruding from it (Figure 4.1a). The other pads had a bluish appearance with fine lines on the surface (Figure 4.2a, 4.3a). On both hind feet there was swelling of the digital pads (Figure 4.3a, 4.4a). On palpation the pads were soft but did not appear painful. On general examination the dorsum of the muzzle was swollen (Figure 4.5a).
DIFFERENTIAL DIAGNOSIS:

- Plasma cell pododermatitis
- Immune mediated disease: pemphigus foliaceus, pemphigus vulgaris
  - bullous pemphigoid
  - systemic lupus erythematosus
- Vasculitis
- Neoplasia (e.g. squamous cell carcinoma, mastocytoma, lymphosarcoma)
- Chemical or physical trauma
- Eosinophilic, bacterial or fungal granulomas

LABORATORY AND OTHER DIAGNOSTIC TESTS:

Microscopic examination of fine needle aspirates taken from a central pad and the nose and stained with a modified Wright’s stain (Diff Quik, Dado Behring AG) revealed large numbers of plasma cells (Figure 4.6). On histopathology of a punch biopsy specimen of the pads, taken under general anaesthesia, there was perivascular to diffuse infiltration of the dermis with plasma and lymphocytic cells (Figure 4.7; Appendix 4.1).

Plasma biochemistry and haematology were normal. Tests for feline leukaemia virus and feline immunodeficiency virus (FIV) were negative.

DIAGNOSIS AND PROGNOSIS:

Plasma cell pododermatitis. The prognosis is fair provided the condition is closely monitored and treatment given as required.
**TREATMENT:**
While under general anaesthesia for sample collection the left central pad was debrided. Tissue of abnormal appearance was removed and the pad sutured. As previous treatment with prednisolone had not been effective, doxycycline (Ronaxan 20mg, Merial Animal Health) was given for four weeks at a dose of 25mg once daily crushed and mixed with food.

**REINSPECTIONS AND FINAL OUTCOME:**
After four weeks all pads were smaller than at initial examination and the ulcerated pad was completely healed following debridement (Figure 4.1b). The owners reported that the cat seemed much more comfortable and was venturing outside for the first time in four years.

After a further four weeks of treatment most pads had returned to a normal size although there remained moderate swelling of some pads (Figure 4.1b-4.4b). The nose was markedly decreased in size (Figure 4.5b). In the year since the cessation of the doxycycline there has been no recurrence of the condition.

**DISCUSSION:**
Plasma cell pododermatitis is an uncommon disease, which presents as a soft spongy swelling of multiple footpads of cats (Taylor and Schmeitzel 1990, Nuttall 1998). The central metacarpal and metatarsal pads are most commonly involved but occasionally digital pads may be affected as seen here (Nuttall 1998).
Biochemistry and haematology are usually normal, as in this case, however, a normocytic anaemia has been seen due to ulceration and haemorrhage of the pads (Taylor and Schmeitzel 1990). Plasmacytic stomatitis has been associated with this condition (Scott 1984) although plasma cell infiltration of the nose, as seen in this case, has not been reported. Although not present here, FIV infection was reported in nearly 50% of a series of cases of plasma cell pododermatitis (Guaguère and others 1992, Simon and others 1993).

The pathogenesis of this condition is unknown. The presence of a plasma cell infiltration and elevated serum globulin levels reported in some cases (Medleau and others 1982, Taylor and Schmeitzel 1990), would suggest an underlying immune mechanism (Guaguère and others 1992). The condition is reported to respond to glucocorticoids and other immunosuppressive drugs (Gruffydd-Jones and others 1980, Drolet and Bernard 1984, Foil 1995). A contact irritant may be involved as White (2003) has seen the condition resolve within four weeks of changing the type of material used in the litter tray.

Treatment may not be necessary in all cases as this condition has been reported to spontaneously regress (Gruffydd-Jones and others 1980) or wax and wane (Drolet and Bernard 1984, Taylor and Schmeitzel 1990). In some cases the clinical signs may not lead to pain or lameness but in this case the large ulcerated pad was causing discomfort and was therefore surgically debrided. Taylor and Schmeitzel (1990) found a good response to

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surgical debridement while Guaguère and others (1992) have recommended total surgical removal of affected pads, noting that the pads appear to regrow normally.

Oral prednisolone is commonly recommended for treatment of this condition. However, responses have been variable and inconsistent (Gruffydd and others 1980, Medleau and others 1982, Taylor and Schmeitzel 1990). One unpublished study\(^3\) compared a group of cases treated with prednisolone with a non-treated control group. Prednisolone did not result in an increase in the frequency of clinical improvement. Treatment with aurothioglucose induced remission in one cat (Medleau and others 1982). Chlorambucil and azathioprine have also been suggested for treatment (Nuttall 1998, Scott and others 2000) but there are no published reports of their use.

It was decided to treat this case with doxycycline as one unpublished study\(^4\) indicated that 89% of cats improved either completely or partially on 25mg of doxycycline orally once daily for up to twelve weeks. Doxycycline is a modified tetracycline, which has some anti-inflammatory effects. Various studies in humans and animals have demonstrated this effect in rheumatoid arthritis, osteoarthritis, periodontal disease and cranial cruciate rupture (Greenwald and others 1987, Yu and others 1991, Greenwald and others 1992, Jauernig and others 2001). Tetracyclines appear to inhibit neutrophil chemotaxis (Esterly and others 1978, Esterly and others 1984) and have been used


Doxycycline has been reported to cause oesophagitis if administered as a whole tablet without food (McGrotty and Knottenbelt 2002) so in this case the tablet was crushed and mixed with the food.

In this case the clinical signs have been seen to wax and wane, however, there has been no recurrence of the condition in the twelve months since cessation of treatment. This may be due to the doxycycline but may also be the normal waxing and waning of this condition. The spontaneous resolution of clinical signs could not be excluded as an explanation for the current remission of the disease.
REFERENCES:


APPENDIX 4.1

HISTOPATHOLOGY REPORT:

Diagnosis: Plasma Cell Pododermatitis

Prognosis: Fair with continued clinical care.

4X pad samples:
There is moderate epidermal hyperplasia, extensive ulceration and a marked perivascular to diffuse infiltration of the dermis with plasmalymphocytic cells. Many plasma cells contain Russell bodies. There is an acute inflammatory infiltrate associated with the ulceration.

Comments:
This is consistent with plasma cell pododermatitis. This is an uncommon idiopathic cutaneous disorder which may also affect the oral mucosa, and which is of undetermined origin. Many cases are asymptomatic and regress spontaneously. Those requiring treatment may respond to prednisolone or chrysotherapy. Recently doxycycline has been suggested as a possible therapy.
Figure 4.1a: Left front foot at initial presentation showing swollen ulcerated central pad

Figure 4.1b: Left front foot after surgical debridement and four weeks of treatment with doxycycline showing resolution of lesions
Figure 4.2a: Right front foot at initial presentation showing swollen bluish central pad

Figure 4.2b: Appearance of the right front foot after four weeks of treatment with doxycycline
Figure 4.3a: Right hind foot at initial presentation showing swollen, bluish central and digital pads with fine lines on the surface. (iatrogenic haemorrhage due to fine needle aspirate)

Figure 4.3b: Appearance of right hind foot after four weeks treatment with doxycycline
Figure 4.4a:
Left hind foot at initial presentation showing swollen central and digital pads with fine white lines and bluish appearance.

Figure 4.4b:
Resolution of lesions on left hind foot after four weeks of treatment with doxycycline.
Figure 4.5a: Swollen dorsum of the muzzle at initial presentation

Figure 4.5b: The dorsum of the muzzle after four weeks of treatment with doxycycline showing resolution of the swelling
Figure 4.6: Photomicrograph of cytology of aspirate from central pad hind foot showing two plasma cells and a neutrophil. (modified Wright’s stain, high power)

Figure 4.7: Histopathological section showing numerous plasma cells containing Russell bodies (Haematoxylin and eosin, high power)
CANINE PEMPHIGUS FOLIACEUS

BREED: Jack Russell Terrier
AGE: Thirteen years
SEX: Entire male
WEIGHT: Eight kilograms

OWNER’S COMPLAINT
Scabs on head and ears, which have progressed to the dorsum and legs.

HISTORY:
In the last three months lumps and crusts have developed on the head and ears. During the last two months the lesions have progressed to involve the lumbar area and the legs. The lesions were not pruritic but the dog was lethargic. Treatment with oral enrofloxacin and an antifungal/antibacterial shampoo (Malaseb, Leo Pharmaceuticals) were ineffective. The companion dog, a littermate, was clinically normal.

PHYSICAL EXAMINATION:
On general examination there was a mild generalised peripheral lymphadenopathy.
Dermatological examination revealed a patchy alopecia and crusted papules of the head, ears, dorsum, flanks and distal limbs (Figure 5.1-5.6). The ventral abdomen and the black haired areas on the dorsum were normal. No pustules were seen.
DIFFERENTIAL DIAGNOSIS:

- Immune mediated disease  
  - Pemphigus foliaceus
  - Pemphigus erythematosus
  - Discoid and Systemic lupus erythematosus
- Bacterial folliculitis
- Dermatophytosis (*Tricophyton mentagrophytes*)
- Demodicosis
- Cutaneous drug eruption
- Zinc responsive dermatosis
- Subcorneal pustular dermatosis

LABORATORY AND OTHER DIAGNOSTIC TESTS:
Microscopic examination of skin scrapings and hair plucks mounted in liquid paraffin were negative for ectoparasites. Wood’s lamp examination was negative for fungal organisms and fungal culture of hairs and crusts was negative. Microscopic examination of acetate tape preparations taken from crusted areas and stained with a modified Wright’s stain (Diff Quik, Dado Behring AG) revealed coccoid bacteria. After a three-week course of oral cephalexin (Rilexine 300mg, Virbac) at a dose of 20mg/kg twice daily the lesions were unchanged.

Histopathological examination of punch biopsy specimens, taken from affected areas, revealed intra epidermal pustules with neutrophils and acanthocytes (Figure 5.7,5.8; Appendix 5.1).

Haematology and plasma biochemistry were normal.
**DIAGNOSIS AND PROGNOSIS:**

Pemphigus foliaceus. The prognosis is guarded.

**TREATMENT:**

Treatment was initiated at immunosuppressive doses of oral prednisolone 2mg/kg once daily for three weeks.

**REINSPECTIONS AND FINAL OUTCOME:**

After two weeks of treatment there were fewer crusts present and the owner reported that the dog was more active and alert. There was no evidence of new lesions developing. Over a period of eight weeks the prednisolone was tapered to 1mg/kg every other day. A week later there was recurrence of the lesions. Prednisolone was increased to 2mg/kg daily and azathioprine (Imuran 25mg, Glaxo Wellcome) at a dose of 2mg/kg daily was commenced. After two weeks there was no evidence of lesions developing so the prednisolone and the azathioprine were tapered over a further eight weeks to 1mg/kg every other day.

On examination one month later there was good hair growth and no lesions were evident (Figure 5.9-5.12). There has been no recurrence of the clinical signs in the past four months while being maintained on prednisolone and azathioprine.

**DISCUSSION:**

Pemphigus foliaceus, the most commonly seen bullous immune mediated skin disease of dogs (Werner and others 1983, Ihrke and others 1985, Delmage
1992), is an autoimmune disease in which antibodies bind to desmoglein I, a transmembrane desmosomal protein that mediates intercellular adhesion (Kuhl and others 1994). The antigen has been characterised in humans (Rubinstein and Stanley 1987, Razsi and others 1990) and in dogs (Suter and others 1993). Although not seen in this case, there have been reports of familial occurrence in dogs (Noxon and Myers 1989) and in humans (Voelter and others 1973).

The clinical signs of crusts and alopecia initially involving the head and ears and slowly progressing to become generalised with no involvement of mucocutaneous junctions, are consistent with previous reports (Werner and others 1983, August and Chickering 1985, Ihrke and others 1985, Suter and others 1998). The lack of lesions on the black haired areas seen in this case has not been reported.

Footpad hyperkeratosis, although not present in this case, is commonly seen (Ihrke and others 1985, August and Chickering 1985) and may be the only lesions found (Scott and others 1987). The primary lesions of vesicular pustules are very short lived and are rarely seen (Ihrke and others 1985, Carlotti 1989). Systemic signs of illness, including the lymphadenopathy and lethargy seen in this case are not a consistent finding (Halliwell 1980, Werner and others 1983, Ihrke and others 1985, Scott and others 1987).

Pemphigus foliaceus may be clinically and histopathologically indistinguishable from superficial folliculitis (Werner and others 1983, Kuhl and others 1994, Suter and others 1998) and dermatophytosis (Parker and Yager
The use of antibiotics to rule out bacterial pyoderma (Marsella 2000, Bond 2002) can be useful, as in this case. Jack Russell terriers, perhaps as a result of their hunting and investigative behaviour, appear to be predisposed to infection with *Trichophyton mentagrophytes* (Bond 2002).

The diagnosis in this case was based on the histopathology findings of intra-epidermal pustules with acantholytic keratinocytes, which is considered pathognomonic for this condition (Suter and others 1998, Werner and others 1983).

Direct immunofluorescence is positive in 60-80% of cases and can be used to support the diagnosis of pemphigus foliaceus especially in those with non-diagnostic histopathology (Scott and others 1987, Marsella 2000).

Prednisolone, at doses of 2-4mg/kg once daily, is the most common immunosuppressive agent used to induce remission in this condition (Halliwell 1980, Rosenkrantz 1993, Marsalla 2000). Once remission is obtained the prednisolone should be tapered to 0.5-1.0mg/kg every other day (Rosenkrantz 1993) and in around 50% of cases this will maintain remission (Ihrke and others 1985, Scott and others 1987). In those cases which cannot be maintained on low doses of glucocorticoids, as in this instance, other medications such as azathioprine and chlorambucil can be used. These drugs act as steroid sparing agents to minimize the side effects of long-term immunosuppressive glucocorticoids (Ihrke and others 1985, Rosenkrantz...
Azathioprine in combination with prednisolone was able to maintain remission in this case.

Cyclosporin, dapsone, tetracycline/niacinamide, chrysotherapy and cyclophosphamide have all been used with varying success (Manning and others 1980, Ihrke and others 1985, Rosenkrantz and others 1989, Scott 1990, Kristensen and Mehl 1992, White and others 1992).
REFERENCES:


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APPENDIX 5.1

HISTOPATHOLOGY REPORT:

Diagnosis: Pemphigus foliaceus
Prognosis: Guarded

5X skin biopsies:
There is mild to moderate irregular epidermal hyperplasia and an intra-epidermal pustule containing mainly, non-degenerate polymorphonuclear cells and acantholytic cells. There is mixed superficial perivascular dermatitis. There is no evidence of bacteria, mites and dermatophytes in the sections examined. PAS staining was negative for dermatophytes.

Comments:
This is consistent with a diagnosis of pemphigus foliaceus. Pemphigus foliaceus is an autoimmune disease involving deposition of antibodies on keratinocytes within the stratum spinosum, activation of enzymes and interference with desmosome formation leading to clefting. Although the aetiology is unknown it is thought to occur either as a spontaneous disease, drug-induced disease of a sequel to chronic inflammatory disease. Adequate control may be possible with immunosuppressive therapy.
Figure 5.1: The appearance of the head at initial presentation showing patchy alopecia and crusting.

Figure 5.2: Close-up of the crusting on the dorsum of the head seen at initial presentation.
Figure 5.3:  
The right hind leg at initial presentation showing crusting and alopecia

Figure 5.4:  
Close up of the crusting on the right front leg seen at initial presentation
Figure 5.5: Crusting and patchy alopecia seen on the dorsum at initial presentation.

Figure 5.6: Crusting and patchy alopecia of head and legs seen at the time of biopsy specimen collection (sampling site on the right side of head).
Figure 5.7: Histopathological section showing an intraepidermal pustule (Haematoxylin and eosin, low power)

Figure 5.8: Histopathological section – close up of intraepidermal pustule showing acantholytic cells and neutrophils (Haematoxylin and eosin, high power)
Figure 5.9: Resolution of lesions on the head after four months treatment with prednisolone and azathioprine

Figure 5.10: Resolution of lesions on the head after four months of treatment with prednisolone and azathioprine
Figure 5.11: Appearance of right front leg after four months of treatment with prednisolone and azathioprine

Figure 5.12: Resolution of lesions on the dorsum after four months of treatment with prednisolone and azathioprine
CANINE JUVENILE CELLULITIS

BREED: Jack Russell Terrier
AGE: Ten weeks
SEX: Entire male

OWNER’S COMPLAINT:
Lethargy, discharge from eyes and swelling around the lower jaw.

HISTORY:
For the last week there had been a purulent discharge from both eyes. In the past four days swelling had developed around the lower jaw and crusts were present around the muzzle. The lesions were not pruritic. The appetite was normal, however, there was lethargy and depression. The littermates were all clinically normal. A combined vaccine, DHPPiL (Quantum 7, Schering Plough Animal Health) had been administered five days prior to presentation.

PHYSICAL EXAMINATION:
On general examination the submandibular lymph nodes were visibly enlarged (Figure 6.1). The prescapular lymph nodes were palpably enlarged. The temperature was 39.5°C. There was a bilateral mucopurulent ocular discharge.

On dermatological examination crusts, exudation, erythema and oedema were present around the muzzle, eyelids, prepuce and anus (Figure 6.2-6.5). There was erythema, crusts and scale in both ears (Figure 6.7a). Pustules and papules were present on the abdomen (Figure 6.6).
DIFFERENTIAL DIAGNOSIS:

- Juvenile cellulitis
- Demodicosis
- Dermatophytosis
- Canine acne
- Deep pyoderma
- Angioedema
- Cutaneous drug eruption

LABORATORY AND OTHER DIAGNOSTIC TESTS:

No mites were found on microscopic examination of skin scrapings taken from the muzzle and abdomen and mounted in liquid paraffin. Wood’s lamp examination and fungal culture were negative. Culture of an aspirate from a pustule and a lymph node was negative. An aspirate was taken from an affected lymph node and a pustule and stained with a modified Wright’s stain (Diff-Quik, Dado Behring AG). No bacteria were found on microscopic examination but a number of non-degenerate neutrophils were seen (Figure 6.8, 6.9).

DIAGNOSIS AND PROGNOSIS:

Juvenile cellulitis (puppy strangles). The prognosis is excellent if treatment is given early. Scarring can result if treatment is delayed.
TREATMENT
Prednisolone (Prednisolone Tablets BP (Vet) 5mg, Millpledge Pharmaceuticals) was commenced at a dose of 2mg/kg orally once daily. Cephalexin (Ceporex 50mg, Schering-Plough Animal Health) was given at a dose of 20mg/kg orally twice daily for fourteen days.

REINSPECTIONS AND FINAL OUTCOME:
After a week the lesions were markedly improved. The lymph nodes were reduced to about half their initial size and the owner reported that the puppy was much brighter. Crusts were still present around the muzzle but the oedema had resolved (Figure 6.10). The temperature was within normal range. Prednisolone was continued at the same dose for a further week then reduced to every other day treatment. On examination one week later the lymph nodes were considered normal size on palpation. The lesions had resolved around the eyes, muzzle, anus and ears (Figure 6.7, 6.11, 6.12). There were no crusts or papules evident on the abdomen or prepuce (Figure 6.13).

DISCUSSION:
The age of onset, three months in this case, is consistent with previous reports (White and others 1989, Mason and Jones 1989, Scott and others 2001). There is only one report of a juvenile cellulitis-like condition in an adult dog (Jeffers and others 1995). Although not seen in this case multiple puppies in a litter may be affected (Mason and Jones 1989, Reimann and others 1989).
Oedema and crusting of the eyelids, lips and muzzle, and submandibular lymphadenopathy, seen in this case, are common clinical signs (Mason and Jones 1989, White and others 1989, Scott and others 2001). Although not seen here lymphatic lesions may fistulate and drain pus, which can lead to scarring (Scott and others 2001). The perineal and inguinal regions are less commonly affected. Anorexia and joint pain, which was not seen in this case, have been noted in around 50% of cases (White and others 1989). Lethargy and depression with pyrexia, as seen here, is a variable clinical sign (White and others 1989).

Diagnosis of this condition is usually based on clinical signs and the use of scrapings, culture and cytology to rule out other differentials, as in this case. Suppurative lymphadenitis with neutrophils seen on cytology of a pustule and affected lymph node in this case is a common finding (White and others 1989). Bacterial culture of pustules was negative in this case and in other reports of this condition (Reimann and others 1989, White and others 1989). Biopsy specimens were not taken due to the rapid response to treatment. However, histopathology shows similar findings to cytology (Reimann and others 1989).

The pathogenesis of this condition is not understood and treatment has been based on historically excellent responses to glucocorticoids (White and others 1989, Malik and others 1995, Scott and others 2001). It has been suggested that this is a systemic disease with dermatological signs and is possibly an exaggerated immune response to a foreign antigen perhaps of viral origin (Reimann and others 1989). Malik and others (1995) have suggested a link
between distemper vaccination and this condition. Evidence of metaphyseal osteopathy, which is reportedly associated with distemper virus (Grøndalen 1979, Mee 2000), was found in a series of five cases of juvenile cellulitis (Malik and others 1995). Clinical signs developed in this case 24 hours post vaccination.

Immunosuppressive doses of oral glucocorticoids at 2mg/kg daily are recommended (Scott and others 2001) and were successful in this case. This treatment is combined with antibiotics, where there is evidence of secondary bacterial infection (Mason and Jones 1989, White and others 1989). Cephalexin was used in this case as the pyrexia and lethargy indicated possible secondary bacterial infection.

Relapses are rarely reported (White and others 1989, Scott and others 2001) and have not occurred in this case in the year since treatment concluded.
REFERENCES


Figure 6.1: Enlarged submandibular lymph nodes visible at initial examination

Figure 6.2: Oedema, crusting and erythema of the eyelids and muzzle seen at initial presentation
Figure 6.3: Crusting and oedema of the eyelids and muzzle, and erythema of the concave pinnae seen at initial presentation.

Figure 6.4: Close up of crusting, and oedema of the eyelids seen at initial presentation.
Figure 6.5: Crusting of the ventral anus seen at initial presentation.

Figure 6.6: Erythema, papules and pustules of the ventral abdomen and erythema of the prepuce seen at initial presentation. The presence of blood is iatrogenic due to an aspirate sample taken of an intact pustule.
Figure 6.7a: Appearance of ear at initial presentation showing erythema, scale and crusting.

Figure 6.7b: Appearance of the ear after three weeks of treatment with prednisolone.
Figure 6.8: Photomicrograph of aspirate of an abdominal pustule at initial presentation showing non-degenerate neutrophils with vacuoles (modified Wright’s stain, high power)

Figure 6.9: Photomicrograph of aspirate of submandibular lymph node at initial presentation showing non-degenerate neutrophils – (modified Wright’s stain, high power)
Figure 6.10: Appearance of the head after one week of treatment with prednisolone and cephalaxin showing reduction in crust and oedema

Figure 6.11: Resolution of lesions of the head after three weeks of treatment
Figure 6.12: Resolution of lesions on the head after three weeks treatment with prednisolone

Figure 6.13: Resolution of lesions on the abdomen and prepuce after three weeks of treatment with prednisolone
CANINE ATOPIC DERMATITIS

BREED: Dalmatian
AGE: Four years
SEX: Entire male
WEIGHT: 30 kilograms

OWNER’S COMPLAINT:
Chewing feet continuously for the past eight months.

HISTORY:
The dog had exhibited a seasonal pruritus of the feet for the past two years but in the last eight months this had become continuous. The clinical signs were more severe when out on grass. Otitis externa had occurred twice in the past two years. Treatment with glucocorticoids had resulted in temporary improvement of the pruritus. There were no lesions on in-contact humans. The sire of this dog had a history of persistent otitis externa. A prescription diet to control cystine uroliths was used. Selamectin was used monthly for flea control.

PHYSICAL EXAMINATION:
Dermatological examination revealed erythema of the concave pinnae and between the pads on the ventral surface of all feet. On the caudal aspect of the metatarsal and metacarpal pads there was alopecia, erythema and crusts (Figure 7.1-7.4). On general examination the popliteal lymph nodes were slightly enlarged.
DIFFERENTIAL DIAGNOSIS:

- Hypersensitivity
  - Atopic dermatitis
  - Food hypersensitivity
  - Flea bite hypersensitivity
  - Contact hypersensitivity

- Ectoparasites - fleas
  - *Sarcoptes scabiei*,
  - *Demodex canis*
  - Cheyletiellosis

- Dermatophytosis.
- Bacterial folliculitis
- Malassezia dermatitis.

LABORATORY AND OTHER DIAGNOSTIC TESTS:

No evidence of ectoparasites was found on microscopic examination of hair plucks, skin scrapings and coat brushings. Fungal culture was negative. Acetate tape preparations taken from affected areas and stained with a modified Wright’s stain (Diff Quik, Dado Behring AG) showed coccoid bacteria only. Oral cephalexin at a dose of 20mg/kg twice daily was given for three weeks for pyoderma. This resulted in mild improvement of lesions on the feet. A diet of pork and potato was fed exclusively for eight weeks, by the end of which the pruritus and erythema were unchanged. Intradermal testing with commercial aqueous allergens (ARTU Biologicals) was performed using standard methods under sedation with medetomidine.
(Domitor, Pfizer Limited). Significant reactions were seen to a number of allergens (Figure 7.5, Appendix 7.1).

A serum allergy test was performed (Heska allercept indoor panel and general pollen screen) using Fc receptor antibodies. There were a number of significant reactions (Appendix 7.2).

**DIAGNOSIS AND PROGNOSIS:**
Atopic dermatitis (AD). The prognosis is guarded.

**TREATMENT:**
With the owners informed consent immunotherapy was commenced using an alum-precipitated vaccine (ARTU Biologicals). Only eight allergens or allergen groups can be used for a single vaccine. Therefore only those allergens demonstrating a positive reaction that correlated with the history and clinical signs were included (Appendix 7.3). The treatment has continued to the present following the manufacturer’s schedule (Appendix 7.4).

At the start of immunotherapy other treatments were given to relieve the pruritus. Clemestine (Tavigal, Novartis) at a dose of 1mg/kg twice daily was given for ten days with no improvement. Prednisolone (Prednisolone BP Vet 5mg, Millpledge Pharmaceuticals) given orally at 0.5mg/kg daily for five days reducing to 0.25mg/kg every other day over the next three weeks, resulted in resolution of the pruritus.

An essential fatty acid supplement containing evening primrose oil and marine fish oil (Efavet regular, Schering-Plough) at a dose of 15 capsules daily was
given. Allergen avoidance was attempted. This involved not walking the dog on grass and washing the feet with an oatmeal shampoo (Episothe, Virbac) after contact with grass. Dust mite reduction and avoidance measures were used in the house.

REINSPECTIONS AND FINAL OUTCOME:
After three months of immunotherapy prednisolone was discontinued. There was still erythema of the feet but the dog was markedly less pruritic. After six months there was almost a complete resolution of the clinical signs with no erythema or pruritus of the feet (Figure 7.6-7.8). Nine months after initiation of immunotherapy the essential fatty acid (EFA) supplementation was stopped. In the following six months maintenance immunotherapy injections have continued to control the condition.

DISCUSSION:
The age of onset of clinical signs, two years, is consistent with previous reports (Scott 1981, Nesbitt and others 1984, Nuttall 2001, Zur and others 2002). The Dalmatian is reported to have an increased incidence of atopic dermatitis (AD) (Schick and Fadok 1986, Scott 1981). Familial occurrence of AD has been reported in humans and animals (Schwartzman 1984, Sousa and Marsella 2001). The sire of this case has a history suggestive of allergic skin disease.

AD should be diagnosed by history and clinical signs and elimination of other relevant differential diagnoses. Intradermal and serum allergy testing should be used for detection of allergens for immunotherapy (Hillier 2002, DeBoer
and Hillier 2001, Nuttall 2001). The clinical signs in this case were compared with the criteria established by Willemse (1986) and after intradermal and blood testing, four major and three minor criteria were present.

Despite the now widespread use of serum allergy tests the intradermal test is still regarded as the ‘gold standard’ (Codner and Tinker 1995, DeBoer 1996, Hillier 2002). However, false positives and false negatives can occur (Codner and Tinker 1995, Halliwell and others 1996, Bourdeau and others 1996, Hillier and others 2000, Hillier 2002).

A serum allergy test, an FcεRIα-based assay for detection of allergen reactive IgE, was used in this case to supplement the data from the intradermal test. This test has been shown to have a high sensitivity (86%) and specificity (92%) when compared with intradermal tests (Wassom and Grieve 1998). In this case there was some correlation between the blood test and the intradermal test.

Treatment of AD should include a combination of allergen avoidance, anti-inflammatory agents, allergen specific immunotherapy and antimicrobial drugs (Olivry and Sousa 2001). Allergen avoidance has proved successful in humans (Platts-Mills and others 2000, Tan and others 1996) and was helpful in this case, however, total elimination of exposure to allergens is difficult (DeBoer 1989, Kunkle 1989, Hillier 2002).

Response to immunotherapy is variable with studies reporting up to 70% of cases showing some clinical improvement (Zur and others 2002b, DeBoer...
One double-blinded study claimed clinical improvement in 59% of cases (Willemse and others 1984). Scott and others 1993) reported no difference in response whether blood or intradermal test results were used as a basis for immunotherapy.

Clinical improvement in this case was seen within six months of starting immunotherapy although in some cases this may take up to 12 months (Angarano and MacDonald 1992).

EFAs have been reported to be useful in the management of atopic dermatitis (Scaff and Lloyd 1992, Bond and Lloyd 1992, Logas and Kunkle 1994) and may have improved the clinical signs in this case prior to response to the immunotherapy.

Antihistamines, although unsuccessful in this case, can be useful in the management of pruritus (Paradis and others 1991a, Miller and others 1992, Scott and others 1999). Glucocorticoids are very effective in reducing pruritus, as in this case (Paradis and others 1991b, Olivry and others 2002a) but long-term use is associated with undesirable side effects. Cyclosporin has been reported to be as successful as glucocorticoids in the treatment of pruritus in AD with good to excellent response in 60-75% of dogs. (Olivry and others 2002a, Olivry and others 2002b).

Other medications have been trialed including herbal treatment (P07p), lipoxygenase inhibitors (zileuton), misoprostal, topical tacrolimus, phosphodiesterase inhibitors and pentoxifylline, with some success, however
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extracts of house dust and house dust mite in healthy dogs and dogs 
suspected of being atopic. Journal of the American Veterinary Medical 
Association 206, 812-816

placebo-controlled, cross-over pilot study on the efficacy of zileuton for canine 
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### APPENDIX 7.1

Wheal reaction 15 minutes post-injection

<table>
<thead>
<tr>
<th>Allergen</th>
<th>Reaction</th>
<th>Allergen</th>
<th>Reaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Histamine – positive control</td>
<td>4+</td>
<td>Poplar</td>
<td>0</td>
</tr>
<tr>
<td>Saline – negative control</td>
<td>0</td>
<td>Oak</td>
<td>0</td>
</tr>
<tr>
<td>Flea extract</td>
<td>0</td>
<td>Willow</td>
<td>0</td>
</tr>
<tr>
<td><em>Tyrophagus putrescentiae</em></td>
<td>2+</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Acarus siro</em></td>
<td>2+</td>
<td>Red top grass</td>
<td>4+</td>
</tr>
<tr>
<td><em>Dermatophagoidespteronyssinus</em></td>
<td>2+</td>
<td>Fescue</td>
<td>3+</td>
</tr>
<tr>
<td><em>Dermatophagoides farinae</em></td>
<td>3+</td>
<td>Orchard grass</td>
<td>3+</td>
</tr>
<tr>
<td>Housedust</td>
<td>2+</td>
<td>Kentucky blue grass</td>
<td>3+</td>
</tr>
<tr>
<td>Cat dander</td>
<td>1+</td>
<td>Rye grass</td>
<td>2+</td>
</tr>
<tr>
<td>Sheep wool</td>
<td>0</td>
<td>Timothy</td>
<td>3+</td>
</tr>
<tr>
<td>Parakeet feathers</td>
<td>0</td>
<td>Mugwort</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rapeseed</td>
<td>0</td>
</tr>
<tr>
<td>Birch</td>
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<td>Lamb’s quarter</td>
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</tr>
<tr>
<td>Alder</td>
<td>0</td>
<td>Daisy</td>
<td>0</td>
</tr>
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<td>Hazel</td>
<td>0</td>
<td>Plantain</td>
<td>3+</td>
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<tr>
<td>Beech</td>
<td>2+</td>
<td>Sheep sorrel</td>
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</tr>
<tr>
<td>Ash</td>
<td>0</td>
<td>Dandelion</td>
<td>0</td>
</tr>
<tr>
<td>Sycamore</td>
<td>0</td>
<td>Nettle</td>
<td>0</td>
</tr>
</tbody>
</table>

(4+ - wheal size equal to positive control, 0- wheal size equal to negative control.)
## APPENDIX 7.2

Results serum allergy test: Fc Receptor Antibodies:

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<tr>
<th>Allergen</th>
<th>Antibodies</th>
<th>Positive</th>
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<tbody>
<tr>
<td>Timothy</td>
<td>H234</td>
<td>Yes</td>
</tr>
<tr>
<td>Cocksfoot</td>
<td>86</td>
<td>No</td>
</tr>
<tr>
<td>Meadow Grass</td>
<td>H170</td>
<td>Yes</td>
</tr>
<tr>
<td>Rye Grass</td>
<td>H207</td>
<td>Yes</td>
</tr>
<tr>
<td>Sheep Sorrel</td>
<td>149</td>
<td>No</td>
</tr>
<tr>
<td>English Plantain</td>
<td>42</td>
<td>No</td>
</tr>
<tr>
<td>Nettle</td>
<td>0</td>
<td>No</td>
</tr>
<tr>
<td>Lambs Quarter</td>
<td>101</td>
<td>No</td>
</tr>
<tr>
<td>Mugwort</td>
<td>0</td>
<td>No</td>
</tr>
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<td>Alder</td>
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<td>No</td>
</tr>
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<tr>
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<tr>
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<td>Cat epithelium</td>
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<tr>
<td>Guinea Pig epithelium</td>
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</tr>
<tr>
<td>Flea Saliva</td>
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</tr>
</tbody>
</table>

Comment: Values greater than 150 are considered POSITIVE.
APPENDIX 7.3

Allergens (for immunotherapy vaccine):

Tyrophagus putrescentiae
Dermatophagoides farinae
Dermatophagoides pteronyssinus
Grass Pollen mix (Bermuda, Orchard, Sweet Vernal, Timothy, Velvet grass)
Agrostis gigantea (red top grass)
Festuca pratensis (fescue, meadow)
Poa pratensis (Kentucky blue grass)
Plantago lanceolata (English Plantain)

(All 4+/3+ reactions on the intradermal test and selected positive reactions on the blood test were included in the vaccine)
APPENDIX 7.4

Immunotherapy protocol:

(Given as subcutaneous injection)

Day 1  0.2mls
Week 2  0.4mls
Week 4  0.6mls
Week 6  0.8mls
Week 9  1.0mls
Week 12 1.0mls

Maintenance:

Week 16  1.0mls
Week 20  1.0mls

After week 20, the maintenance dose of 1.0mls continued at four week intervals.
Figure 7.1: Appearance of hind foot at initial presentation showing erythema between the pads and crusting of the metatarsal and digital pads.

Figure 7.2: Different view of Figure 7.1 showing erythema between the pads and crusting of the pads.
Figure 7.3:
Appearance of front foot at initial presentation showing alopecia, erythema and crusting of the dorsal metacarpal and digital pads

Figure 7.4:
Different view of Figure 7.3 showing erythema and crusting of metacarpal and digital pads
Figure 7.5: Intradermal skin test showing erythema and oedema (wheals) of the positive control and a number of other sites (arrows mark the positive reactions)

Figure 7.6: Appearance of hind foot after six months of immunotherapy and EFA supplementation
Figure 7.7: Resolution of lesions on foot after six months of immunotherapy and EFA supplementation

Figure 7.8: Resolution of lesions on the foot after six months of immunotherapy and EFA supplementation
CANINE SARCOPTIC MANGE

BREED: Schnauzer
AGE: Two years and nine months
SEX: Entire male
WEIGHT: 19 kilograms

OWNER’S COMPLAINT:
Swelling and pruritus of the right pinna.

HISTORY:
For the past three months there had been pruritus and swelling of the right pinna. Treatment with oral cephalexin and betamethasone injections had no effect on the clinical signs. There were no lesions on the in-contact humans or the companion dog. Routine flea control was maintained on both dogs with selamectin every three months (Stronghold, Pfizer Limited). The affected dog commonly has contact with fox inhabited areas in the woods where he is walked. Both dogs have only infrequent contact with other dogs.

PHYSICAL EXAMINATION:
Dermatological examination of the right concave pinna showed it to be thickened with crusts, excoriations, patchy alopecia and erythema (Figure. 8.1-8.3). The left pinna was normal. A pinnal-pedal scratch reflex was induced by rubbing the tip of the right pinna. There were papules and pustules in the axilla and groin (Figure 8.4).
On general examination the submandibular lymph nodes were moderately enlarged.

**DIFFERENTIAL DIAGNOSIS:**

- Ectoparasite infestation:  - *Sarcoptes scabiei*
  - Demodicosis
  - Cheyletiellosis
  - Fleas
  - *Otodectes cynotis*
  - Pediculosis

- Atopy
- Food hypersensitivity
- Contact dermatitis
- Dermatophytosis
- Pyoderma
- Malassezia dermatitis

**LABORATORY AND OTHER DIAGNOSTIC TESTS:**

Skin scrapings were taken from the right pinna and mounted in liquid paraffin. Microscopic examination revealed *Sarcoptes scabiei* adults and eggs (Figure 8.5, 8.6). Cytology of a pustule revealed neutrophils with intracellular coccoid bacteria.

**DIAGNOSIS AND PROGNOSIS:**

Sarcoptic mange. The prognosis is excellent.
TREATMENT:
Selamectin (Stronghold, Pfizer) was applied monthly for three months to both the affected and the in-contact dog, using the 120mg formulated product, equivalent to 6mg/kg in this case. A household insecticide, pyriproxyfen and permethrin (Indorex, Virbac), was sprayed in the areas of the house to which the dogs have access.
Cephalexin (Rilexine 600mg, Virbac) was given at a dose of 20mg/kg orally twice daily for three weeks for the secondary pyoderma.

REINSPECTIONS AND FINAL OUTCOME:
On examination one month later, no pustules or papules were evident in the axilla or groin. There was some swelling of the pinna and crusts were still present. The dog was less pruritic and the submandibular lymph nodes were reduced in size.
Treatment was continued using selamectin and on examination, two months later, there were no lesions present on the right pinna (Figure 8.7-8.9). It was not possible to elicit the pinnal-pedal scratch reflex.

DISCUSSION:
The clinical signs, in this case, are consistent with previous reports (Griffin 1993, Folz 1984). Lesions usually become generalised although the occasional case may have lesions that remain localised (Anderson 1979), as in seen in this case. Secondary superficial pyoderma, as seen here, is common (Griffin 1993).
Scabies has been reported in 10-50% of humans after contact with infected dogs (Charlesworth and Johnson 1974, Baker and Stannard 1974, Ruiz-Maldonado and others 1977, Griffin 1993). The infection is usually self-limiting (Estes and others 1983). Spread to in-contact dogs has been reported in 75% of cases (Baker and Stannard 1974). In this case the in-contact dog and humans were not clinically affected by *Sarcoptes scabiei*. It is possible that the infrequent use of selamectin in both dogs may have limited the spread of the mite.

Given the lack of contact with other dogs, the presence of foxes and the affected dog’s habit of close investigation of fox inhabited areas it is likely that the infection was contracted from this species. It is not possible to differentiate morphologically between *S. scabiei* var. *vulpis* and var. *canis* (Zahler and others 1999) and cross-infestivity has been demonstrated between foxes and domestic dogs (Arlian and others 1984a, Bornstein 1991). A recent study documented the presence of sarcoptic mange in foxes in the south of England (Bates 2003), the same geographical location as this case.

Diagnosis of sarcoptic mange can be difficult with 50-65% of skin scrapings being negative (Folz 1984, Bornstein and others 1996). Scraping was diagnostic in this case but other methods can be used to aid diagnosis including faecal flotation for egg detection (Folz 1984). ELISA testing for scabies antibody is useful in the diagnosis of this condition with a sensitivity of 84-92% and specificity of 89-96% (Bornstein and others 1996, Curtis 2001, Lower and others 2002). Antibodies are not detectable until three to five weeks post infection (Bornstein and Zakrisson 1993).
Histopathology can be diagnostic but only if mites can be found (Morris and Dunstan 1996). The pinnal-pedal reflex, as seen in this case, is a useful tool in the diagnosis of scabies as it can be elicited in between 75-90% of dogs (Griffin 1993, Mueller and others 2001). However up to 7% of normal dogs will exhibit the reflex.

Amitraz and selamectin are the only licensed treatments for sarcoptic mange in the dog. Although amitraz is an effective treatment (Folz and others 1984) selamectin, a semi-synthetic avermectin (Bishop and others 2000) was used due to ease of treatment. It has been shown to be safe and 100% effective after two treatments and was successful in this case (Shanks and others 2000, Six and others 2000).

Other unlicensed drugs including fipronil, milbemycin and ivermectin are reported to be effective (Scheidt and others 1984, Curtis 1996, Miller and others 1996).

Environmental treatment was carried out as it has been shown that all life stages of *S. scabiei* can survive off the host for up to three weeks if conditions are favourable (Arlian and others 1984b, Arlian and others 1989).
REFERENCES:

ANDERSON, R.K (1979) Canine scabies. *Compendium on Continuing Education for the Practicing Veterinarian* 1, 687-693


BATES, P (2003) Sarcoptic mange (Sarcoptes scabiei var vulpis) in a red fox (Vulpes vulpes) population in north-west Surrey. *Veterinary Record* 152, 112-114


CURTIS, C.F. (1996) Use of 0.25 per cent fipronil spray to treat sarcoptic mange in a litter of five-week-old puppies. *Veterinary Record* **139**, 43-44


*International Journal for Parasitology* **29**, 759-766
Figure 8.1: Appearance of the right concave pinna at initial presentation showing crust, excoriations, erythema and patchy alopecia.

Figure 8.2: The tip of the right concave pinna at initial presentation showing crust, erythema and patchy alopecia.
Figure 8.3: Close up of the distal right pinna at initial presentation showing crusting, patchy alopecia, erythema and excoriations

Figure 8.4: Pustule in axilla seen at initial presentation
Figure 8.5: Photomicrograph of Adult *S. scabiei* seen on skin scraping taken from the right pinna (low power)

Figure 8.6: Photomicrograph of *S. scabiei* egg seen on examination of skin scraping taken from the right pinna (low power)
Figure 8.7: Appearance of right pinna showing resolution of lesions after three months treatment with selamectin.

Figure 8.8: General appearance of right pinna after three months treatment with selamectin showing resolution of lesions.
PORCINE DERMATITIS AND NEPHROPATHY SYNDROME

**BREED:** Landrace

**AGE:** Sixteen weeks

**SEX:** Castrated male

**OWNER'S COMPLAINT:**
One pig rejected at slaughter due to dermatological and renal lesions. Other pigs in the herd were showing similar dermatological signs. One pig was not gaining weight and was lethargic with generalised haemorrhagic macules (purpura).

**HISTORY:**
For the past 18 months, around 10% of weaned pigs between eight and 16 weeks of age had been developing papules and purpura of the pinnae, trunk and hind limbs (Figure 9.1). In most pigs these lesions resolved but in some the purpura coalesced and became necrotic. Two of the worst affected pigs had been slaughtered and rejected at the freezing works due to the presence of renal and skin lesions. One 16-week-old pig was lethargic and thin and had skin lesions that had become progressively worse with generalised haemorrhagic patches.

The general husbandry of the unit involves weaning at eight weeks of age and keeping individual litter groups together, separated by a solid barrier. The pens are cleaned daily using Virkon-S (Antec International). The pigs are kept at a low stocking rate. The pens are partly open and birds are able to nest in the roof. The sows are brought from one local unit.
For the past five years there has been a 5% rate of occurrence of a wasting disease seen just after weaning. Ninety percent recover but they remain separated from the other pigs. There have been no spontaneous deaths reported associated with the skin condition.

**PHYSICAL EXAMINATION:**

On general examination the worst affected pig was smaller than its littermates and appeared depressed and lethargic. On dermatological examination there were a number of haemorrhagic macules and patches, some showing necrosis, on the dorsum, ventral abdomen, hindquarters and pinnae (Figure 9.2-9.5). On the scrotum there was a large haemorrhagic crust (Figure 9.6).

**DIFFERENTIAL DIAGNOSIS:**

- Porcine Dermatitis and Nephropathy Syndrome (PDNS)
- African Swine Fever (ASF)
- Classical Swine Fever (CSF)
- Swine erysipelas
- *Staphylococcal* infection
- Salmonellosis
- Septicemia
- *Actinobacillus* infection
LABORATORY AND OTHER DIAGNOSTIC TESTS:
Microscopic examination of skin scrapings taken from the papular lesions and mounted in liquid paraffin were negative for ectoparasites. *Staphylococcus xylosus*, a non-pathogenic serotype, was found on culture of an aspirate sample from a papule. The pig was euthanised and a post mortem performed (Appendix 9.1). Histopathology of the skin revealed a necrotising vasculitis and superficial pustular dermatitis (Figure 9.7-9.9). The histopathological findings in the kidney, liver and skin were consistent with a diagnosis of PDNS (Appendix 9.2).

DIAGNOSIS AND PROGNOSIS:
Porcine Dermatitis and Nephropathy Syndrome. The prognosis for herd control is guarded.

TREATMENT:
The remaining pigs with the most severe skin lesions were treated with intramuscular lincomycin (Lincocin 100mg/ml, Pharmacia and Upjohn Animal Health) at 10mg/kg daily for ten days.

Modifications in the pens were made to limit contact with birds. A more rigorous disinfection procedure has been implemented using Virkon-S. As the lesions do not appear to occur before the pigs are around 65 kgs an attempt has been made to have the pigs slaughtered at this lower bodyweight.
REINSPECTIONS AND FINAL OUTCOME:
The lincomycin had no effect on the skin lesions and was discontinued. In the past six months the pigs have been sent for slaughter at a lower weight and none have been rejected. At the time of writing there had been no further severe cases of PDNS. Papules and purpura have continued to appear in around 10% of weaned pigs but the clinical signs are mild and resolve rapidly.

DISCUSSION:
PDNS is a recently described disease in the UK (Smith and others 1993). PDNS usually occurs between the ages of eight to 20 weeks as was seen here (Higgins 1993, Cook and others 2001).

The clinical signs of PDNS are the result of a type III hypersensitivity reaction affecting the lymph nodes, kidney and skin (Thibault and others 1998, Rosell and others 2000, Done and others 2001). A necrotising vasculitis is the predominant finding on histopathology and was seen in this case (Done and others 2001, Thomson and others 2001b).

Morbidity is between 0.5-14%, as in this case (Higgins 1993, Done 2001). Of those pigs showing cutaneous purpura mortality is reported to be between 60 – 100% (Ramos-Vara and others 1997, Done and others 2001). There have, however, been no mortalities at this holding associated with this condition.

It is important to differentiate PDNS from ASF and CSF (Done 2001, Gresham and others 2001b). A recent outbreak of CSF illustrated that it can occur concurrently with PDNS (Gresham and Thomson 2001, Sharpe and others
This case was therefore referred to the divisional veterinary manager after slaughter.

The aetiology of PDNS is unknown but cases have been seen in association with porcine multisystemic and wasting disease (PMWS) (Spillane 2000, Cook and others 2001, Gresham and others 2001a). PMWS appears to be associated with porcine circovirus type 2 (PCV-2) (Ellis and others 2000, Kennedy and others 2000, Allan and Ellis 2000) and in one study PCV-2 was found in 93% of PDNS cases (Rosell and others 2000). It has been suggested that the immune dysfunction caused by PMWS enables other antigens to precipitate the immune complex vasculitis that presents as PDNS (Done and others 2001). The wasting disease seen in recently weaned piglets at this holding is possibly PMWS but this has not been confirmed.

Other infectious agents such as *Pasturella multocida* and PRRSV (porcine reproductive and respiratory syndrome virus) may be associated with PDNS (Thomson and others 2001, Segalés and others 1998) and may worsen the disease. In this case culture of lesions did not detect pathogens but the histopathology of the skin indicated possible bacterial involvement.

Peritogianni (2001) reported poor response to antibiotic treatment, as seen in this case. However, there is one report of the use of antibiotics in the month prior to the onset of PDNS reducing the number of clinical cases (Thomson and others 2001a). Other suggested treatments include ‘serum therapy’, the use of serum from recovered older pigs on younger pigs, (Dean 2002) but there are no published reports of its success.
Management of PDNS involves control of PCV-2 infection and minimising stress (Done and others 2001). A recent survey of herds in the UK found 95% of pigs with no clinical signs were seropositive for PCV-2 (Thomson and others 2000). Hence elimination of PCV-2 may be difficult. The virus is resistant to most disinfectants (Allan 2000), however, Virkon-S, a virucidal disinfectant, used in this unit, claims to be active against PCV-2 (Done and others 2001).

Limiting bird to pig contact, as in this case, is recommended as the virus may be mechanically spread by birds (Done and others 2001). Vertical transmission may occur (Done and others 2001) hence identification of infected sows may be useful in elimination of PCV-2.

Stress appears to trigger clinical disease and minimising stresses is important in the control of PDNS. Recommendations include weaning later, not mixing litter groups, low stocking rates, isolating sick pigs and adequate nutrition, all measures currently used here (Done and others 2001).
REFERENCES:


DEAN, S. (2002) 'Serum therapy' in pigs as a treatment for PMWS/PDNS. *Veterinary Record* 150, 222


APPENDIX 9.1

Report 1:

The carcass of a 36kg castrated male pig was received for examination. The pig had been shot and there was blood exuding from the nostrils and mouth. There were numerous brown raised scabs particularly prominent over the bell, posterior aspects of the hind leg and a continuous black scab approximately 10cm in diameters covering the scrotum. In the buccal cavity there were haemorrhages on the larynx and epiglottis. The submandibular and retropharyngeal lymph nodes were markedly enlarged and congested. The thoracic lymph nodes were enlarged. The stomach contained scant bile stained contents. There were no abnormalities in the gastric mucosa. Small and large intestines were grossly normal apart from the markedly enlarged mesenteric lymph nodes. The kidneys were markedly enlarged, the right kidney measuring 145mm, the left kidney 140mm and weighing 211 and 206 grams respectively. There were ecchymotic haemorrhages on the cortex and throughout the cortical tissue. The cortex was pale and remarkably firm. The renal lymph nodes were greatly enlarged and congested.

Report 2:

Tests carried out on the Staphylococcus spp recovered from the skin lesions have shown that this was not Staphylococcus hyicus.
APPENDIX 9.2

Histopathology: report 1

(Histopathology carried out at local ministry of agriculture laboratory)

Histopathology has been carried out on the tissues from this pig and the findings were as follows:

Kidney: severe subacute to chronic exudative glomerulonephropathy with granulomatous interstitial nephritis.

Lymph node: Mild granulomatous lymphadenitis (multinucleated giant cells in many germinal centres).
The histological features are consistent with PDNS

Skin: Severe subacute to chronic necrotising dermatitis with dermal necrotising vasculitis.
Histopathology report 2:

(histopathology from a second laboratory on skin samples taken from the pig at the time of slaughter)

Skin: there is a marked irregular epidermal hyperplasia with rete ridge formations, spongiosis, exocytosis of inflammatory cells and superficial pustules, which contain degenerate polymorphonuclear cells and large numbers of coccoid bacterial. There are areas of epidermal necrosis and superficial dermal necrosis with overlying inflammatory crust and pustules formations. The latter contain degenerate polymorphonuclear cells and deep perivasuclar dermatitis and perifolliculitis. There is degeneration and inflammation of blood vessel walls. The adnexal glands appear normal. There are no yeasts or dermatophytes in the sections examined.

Comments: The changes are not diagnostic however the pattern is consistent with a severe bacterial pyoderma with evidence of vasculitis within deep dermal blood vessels. The latter could represent a type 3 hypersensitivity involving bacterial antigen-antibody complex deposition however I am unable to confirm this from the histological sections.
Figure 9.1: Crusted papules seen on one of the healthy pigs. These are the earliest lesions seen on many of the affected pigs.

Figure 9.2: Haemorrhagic macules/patches on the back of the worst affected pig seen at initial presentation.
Figure 9.3: Haemorrhagic macules/patches with necrosis seen on the dorsum of the affected pig at initial presentation

Figure 9.4: Focal areas of haemorrhagic macules and papules with a large necrotic area over the scrotal area seen on hindquarters of affected pig at initial presentation
Figure 9.5: Haemorrhagic macules/patches on the pinna, neck and head seen on worst affected pig at initial presentation

Figure 9.6: Large haemorrhagic crust over scrotum seen on worst affected pig at initial presentation
Figure 9.7: Histopathological section showing superficial dermal necrosis with inflammatory crust (Haematoxylin & eosin, low power)

Figure 9.8: Histopathological section showing acute vasculitis (Haematoxylin & eosin, high power)

Figure 9.9: Histopathological section showing acute vasculitis (Haematoxylin & eosin, high power)
CANINE HYPOTHYROIDISM

**BREED:** Italian Spinone

**AGE:** Seven years

**SEX:** Spayed female

**WEIGHT:** 41.6 kilograms

**OWNER’S COMPLAINT:**
Dull and dry coat with scale accompanied by lethargy and weight gain.

**HISTORY:**
In the past six months the dog had been gaining weight and had become lethargic and reluctant to exercise. There was alopecia of the neck where the collar normally sits. Over the last four months the coat has become dull and scale has become obvious. No pruritus was evident.

**PHYSICAL EXAMINATION:**
General examination revealed obesity and bradycardia (72 beats per minute). On dermatological examination the hair coat was dull and generalised scale was present (Figure 10.1, 10.2). There was poor hair growth around the neck at the location of the collar (Figure 10.3, 10.4).
DIFFERENTIAL DIAGNOSIS:

- Endocrine disease
  - Hypothyroidism
  - Hyperadrenocorticism
  - Sex-hormone dermatosis (alopecia X)
- Demodicosis
- Dermatophytosis
- Nutritionally deficient diet
- Intestinal malabsorption/digestion
- Sebaceous adenitis

LABORATORY AND OTHER DIAGNOSTIC TESTS:

Microscopic examination of hair plucks and skin scrapings mounted in liquid paraffin was negative for ectoparasites and dermatophytes. Wood’s lamp examination was negative and fungal culture of hairs and scale was negative. Plasma biochemistry, on a fasted sample, revealed high cholesterol (Appendix 10.1). Haematology was normal. Total thyroxine (TT4) and free T4 (fT4) were below reference range. Canine thyroid stimulating hormone (cTSH) was elevated (Appendix 10.2).

DIAGNOSIS AND PROGNOSIS:

Hypothyroidism. Prognosis is good provided replacement treatment is maintained.
**TREATMENT:**
L-thyroxine sodium (Soloxine, Arnolds) was given at a dose of 0.02mg/kg bodyweight once daily in the morning.

**REINSPECTIONS AND FINAL OUTCOME:**
After one month the dog had lost 2.4kgs and the heart rate was normal at 90 beats per minute. The owners commented that the dog was more alert and less tired after exercise. A blood sample taken six hours after administration of L-thyroxine showed the TT4 to be just above the low end of the reference range. The cTSH level was suppressed (Appendix 10.2).

The dose was increased to 0.025mg/kg once daily and the TT4 and cTSH was measured after a further fortnight of treatment. The TT4 was just above the upper reference range (Appendix 10.2). The dog was maintained at this dose and on examination four months later the weight had reduced to 33.5kg. The hair and skin appeared normal with no scale present and the hair had regrown around the neck (Figure 10.5-10.7). The dog has been maintained on this dose of L-thyroxine for the past four months with no recurrence of dermatological or systemic signs.

**DISCUSSION:**
Hypothyroidism is reported to occur more frequently in middle aged, medium to large purebred dogs (Kaelin and others 1986, Panciera 1994) as in this case. Spayed female dogs have been reported to have a higher incidence of the disease (Milne and Hayes 1981, Panciera 1994).
The classical clinical signs observed in hypothyroidism include dermatological signs, lethargy and obesity (Kaelin and others 1986, Panciera 1994, Dixon and others 1999, Paradis 2002) as was seen here. Dermatological abnormalities, including dry and dull haircoat, seborrhoea and alopecia especially of friction areas, are seen in 70-80% of cases (Kaelin and others 1986, Panciera 1994, Dixon and others 1999, Paradis 2002). Metabolic signs, including lethargy and obesity, as seen in this case, are reported in up to 80% of cases (Panciera 1999, Dixon and others 1999). Bradycardia is reported in 15% of cases (Panciera 1994).

Haematology and plasma biochemistry may support a diagnosis of hypothyroidism (Paradis 2002). A mild non-regenerative anaemia, although not seen in here, has been seen in 30-40% of cases of hypothyroidism (Dixon and others 1999, Paradis 2002). Hypercholesterolaemia, as seen in this case, has been reported in 70-80% of cases (Panciera 1994, Dixon and others 1999).

The measurement of TT4 is a useful screening test with a sensitivity of up to 100% (Dixon and others 1999). The low specificity of TT4 measurement (74% in one study (Dixon and Mooney 1999)) is due to suppression of TT4 by non-thyroidal illness and drug treatment (Ferguson 1994, Dixon and Mooney 1999). Free T4 measured by equilibrium dialysis has a specificity of 93% and is useful in cases of non-thyroidal illness with a low TT4 (Dixon and Mooney 1999). cTSH is best used in combination with TT4/fT4 levels as it may be normal in some hypothyroid dogs and elevated in some euthyroid dogs (Dixon

Use of TT4 in combination with cTSH will increase the accuracy of diagnosis (Scott-Moncrieff and others 1998) although non-thyroidal illness may show thyroid function tests similar to those found in hypothyroidism (Ramsey and others 1997). In this case, in the absence of non-thyroidal illness and drug therapy and the presence of classical clinical signs, the cTSH and TT4 would be considered to confirm a diagnosis of hypothyroidism (Panciera 1999).

Dynamic testing was not performed in this case due to the clear TT4 and cTSH tests. The TSH stimulation test is considered the ‘gold standard’ for diagnosis of hypothyroidism (Panciera 1999). It is now rarely used due to unavailability of pharmaceutical grade bovine TSH although recombinant human TSH may be a useful alternative (Sauvé and Paradis 2000). The Thyrotropin releasing hormone (TRH) response test shows variable results (Sparkes and others 1995) and is not recommended (Kemppainen and MacDonald 1993).

Treatment with L-thyroxine was initiated at a dose of 0.02mg/kg once daily as a recent study indicated that this is an adequate dose in the majority of hypothyroid dogs (Dixon and others 2002). Observed clinical improvement should not be used to determine treatment success as there is variable individual response to L-thyroxine (Nachreiner and Refsal 1992) Up to 50% of cases require dose adjustment (Dixon and others 2002), as in this case.
Monitoring can be performed two weeks after a dosage adjustment as TT4 and TSH are in their therapeutic range within two weeks of any dosage change (Dixon and others 2002). Good clinical control is associated with TT4 levels around 55nmol/litre (Dixon and others 2002), as in this case.
REFERENCES:


## APPENDIX 10.1

Haematology and plasma biochemistry results:

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<td>66.5g/l</td>
<td>54-77</td>
</tr>
<tr>
<td>Albumin</td>
<td>34.4g/l</td>
<td>25-37</td>
</tr>
<tr>
<td>Total globulin</td>
<td>32.1g/l</td>
<td>23.0-52.0</td>
</tr>
<tr>
<td>Sodium</td>
<td>150mmol/l</td>
<td>139-154</td>
</tr>
<tr>
<td>Potassium</td>
<td>5.50mmol/l</td>
<td>3.60-5.60</td>
</tr>
<tr>
<td>Chloride</td>
<td>106mmol/l</td>
<td>105-122</td>
</tr>
<tr>
<td>Calcium</td>
<td>2.82mmol/l</td>
<td>2.30-3.00</td>
</tr>
<tr>
<td>Phosphate</td>
<td>1.39mmol/l</td>
<td>0.80-1.60</td>
</tr>
<tr>
<td>Urea</td>
<td>7.2mmol/l</td>
<td>1.7-7.4</td>
</tr>
<tr>
<td>Creatinine</td>
<td>90μmol/l</td>
<td>0-106</td>
</tr>
<tr>
<td>Total bilirubin</td>
<td>0.2μmol/l</td>
<td>0.0-16.0</td>
</tr>
<tr>
<td>Alanine aminotransferase</td>
<td>21U/l</td>
<td>0-25</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>20U/l</td>
<td>0-50</td>
</tr>
<tr>
<td>Creatine kinase</td>
<td>160U/l</td>
<td>0-190</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>12.4mmol/l</td>
<td>3.8-7.0</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>2.82mmol/l</td>
<td>0.56-1.69</td>
</tr>
<tr>
<td>Glucose</td>
<td>4.5mmol/l</td>
<td>2.0-5.5</td>
</tr>
<tr>
<td>Amylase</td>
<td>444U/l</td>
<td>100-900</td>
</tr>
</tbody>
</table>
## APPENDIX 10.2

### Results of thyroid testing:

<table>
<thead>
<tr>
<th>Before treatment:</th>
<th>Result</th>
<th>Reference range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Thyroxine (TT4)</td>
<td>&lt; 3.9nmol/l</td>
<td>(15-40)</td>
</tr>
<tr>
<td>Free Thyroxine (fT4)*</td>
<td>&lt;3nmol/l</td>
<td>(6.6-40)</td>
</tr>
<tr>
<td>Thyroid stimulating hormone (TSH)</td>
<td>5.70ng/ml</td>
<td>(0.01-0.60)</td>
</tr>
</tbody>
</table>

**After one month of L- thyroxine supplementation:**

| TT4 | 19.7nmol/l | (15-40) |
| cTSH | 1.65ng/ml | (0.01-0.60) |

**Two weeks after dose of L-thyroxine increased:**

| TT4 | 65.0nmol/l | (15-40) |
| cTSH | 0.05ng/ml | (0.01-0.6) |

*measured by equilibrium dialysis*
Figure 10.1: Appearance at initial presentation showing dull and dry coat with seborrhoea

Figure 10.2: Close up of scale present in coat at initial presentation
Figure 10.3: Appearance of the neck at initial presentation showing poor hair growth at the site where the collar normally rests.

Figure 10.4: Close up of neck at initial presentation showing hypotrichosis at the collar site.
Figure 10.5: Appearance of coat after four months of treatment with L-thyroxine showing resolution of lesions.

Figure 10.6: Close up of neck after four months of treatment with L-thyroxine showing normal hair growth.
Figure 10.7: Appearance of the coat after four months of treatment with L-thyroxine showing resolution of the seborrhoea