THE UGLY COCKATIEL – A CASE OF CHRONIC DERMATITIS

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A five year old male cockatiel *(Nymphicus hollandicus)*, bred by the owner and with no previous disease history, was first seen in April 2008 for a problem with skin over the face. The cockatiel had successfully bred many offspring and its last clutch 18 months earlier had been successfully raised. The bird had been housed in an aviary with other cockatiels until the last few weeks when the owner quarantined the bird after first noticing the signs. The skin problems had been evident for a few weeks and were getting worse. The owner had been trying to treat the lesions by applying paraffin oil. He also had budgerigars *(Melopsittacus undulatus)* and king quail *(Coturnix chinensis)* in separate aviaries. None of the other birds were showing any signs of illness. He had stopped breeding the parrots for his own personal reasons but had kept several of his favourite birds. He was still breeding the quail some distance from the parrots. Apart from the quail the owner had a closed aviary. Since it has been quarantined, the bird is housed inside in a cage but not in the kitchen or near air-conditioning systems. There are no smokers in the household. The cage is put outside in sunlight most days if the weather is appropriate.

On examination the bird was bright and alert and in good body condition. There were severe lesions on the face and legs resembling those seen with *Cnemidocoptes* spp infection. It is not recorded whether a skin scrape was performed at the time to confirm the diagnosis. The skin on the legs and feet were so inflamed that the closed band was causing constriction of the limb. The band was removed and the bird treated with oral ivermectin (300 µg/kg PO). The owner was advised to treat all other birds in the aviary and to keep the cockatiel isolated. The cockatiel returned 2 weeks later for a second dose of ivermectin, when it was noted that the skin of the legs and face were much improved and that new pin feathers were present over the head, face and ears.

Two months later the bird represented with a suspected relapse of cnemidocoptic mange. A skin scraping was negative for mites but the bird was again treated with oral ivermectin 300 µg/kg and more medication was dispensed for the remaining birds at home and a subsequent dose in two weeks. The cockatiel was also covered with a dose of azithromycin 80 mg/kg PO weekly for two weeks in case of secondary skin infections. One month later the bird was again presented for the same clinical signs and the process repeated and the owner advised again about environmental contamination and re-infection. The owner was also to treat the cockatiel with another two doses of ivermectin 300 µg/kg every two weeks.

The follow-up consult after 5 weeks again showed some improvement with new pin feathers emerging. The skin still showed hyperkeratosis around the face and legs. Mild inflammation of this skin was evident. Skin scrapes were again negative for mites, despite the classical appearance of cnemidocoptic mange. The bird was still very bright, active and in good body condition. At this point there was concern that there was a degree of underlying immunosuppression which might be

delaying recovery. It was also thought that concurrent subclinical psittacosis might be causing immunosuppression. A 6 week course of azithromycin 80 mg/kg weekly was implemented as an off-label treatment for psittacosis and this would also cover most other secondary bacterial infections. Ivermectin was continued fortnightly at 300 µg/kg.

During the next six weeks the owner noticed that the cockatiel was not scratching as much. He did not notice that the bird had been pruritic until it had ceased. The crusting of the skin on the legs and feet appeared to have reduced and the bare skin on top of the head appeared to be less inflamed. Feathers appeared to be re-growing (again). These feathers did not stay. During the subsequent weeks the areas of feather loss over the head increased in size and there was also feather loss over the ears and dorsal wings. The powder down was minimal and there was a patchy feather covering over the body. Although the skin in general had improved, the crusting had not entirely resolved. Cytology of the skin was negative for bacteria and yeasts. The possibility of atypical circovirus was discussed with the owner.

At the next visit 2 weeks later, the cockatiel showed more extensive loss of feathers and new feathers appeared dystrophic. Skin and feather biopsies were taken, as well as blood and pin feathers. The results for PCR, haemagglutination and haemagglutination inhibition, as well as for the skin biopsy, were all negative for circovirus. Despite the continued deterioration of the plumage and increased inflammation of the skin, the biopsy wound healed normally.

The treatment with ivermectin had been discontinued as the skin scrapes had been negative for two months and there was now concern that it may have been causing an unusual allergic reaction leading to dermatitis and feather dystrophy. Antimicrobial coverage was initially continued for another month (total 13 weeks) as some form of immunosuppression was still a concern. Despite this the skin condition of the cockatiel continued to deteriorate. New pin feathers would grow, however they were becoming more dystrophic and easily falling out soon after emerging. The skin continued to be moderately inflamed with hyperkeratosis and scaling present on the head, neck, dorsal wings and feet. Again the bird was still very bright and active and maintaining body condition.

Following one month without treatment, a blood profile was performed and biopsies taken of the affected skin and feather follicles and forwarded for histopathology. The results were as follows:

BIOCHEMISTRY					HAEMATOLOGY				
				Reference					Reference
Glucose (Serum)	13.1		mmol/L	(12.7-24.4)	Platelets	Clumped and Adequate			
Urea	1.3		mmol/L		WCC	11		x 10^9 /L	
AST	**440**	**H**	IU/L	(128-396)	Heterophil %	69		%	(45-72)
CK	**6286**	**H**	IU/L	(160-420)	Heterophil Abs	7.6		x 10^9 /L	(2.0-19.1)
Cholesterol	**8.1**	**H**	mmol/L	(2.3-5.2)	Lymphocyte %	**23**	**L**	%	(26-60)
Amylase	130		IU/L	(113-870)	Lymphocyte Abs	2.5		x 10^9 /L	(0.6-9.4)
GLDH	**5**	**H**	IU/L	(< 3)	Monocyte %	**6**	**H**	%	(< 2)
Uric Acid	0.54		mmol/L	(0.20-0.65)	Monocyte Abs	0.7		x 10^9 /L	(< 1.4)
Sample Appearance	Normal				Eosinophil %	2		%	(< 3)
Bile Acids (Random)	**0**	**L**	umol/L	(44-108)	Eosinophil Abs	0.2		x 10^9 /L	(< 1.0)
					Basophil %	0		%	(< 2)
					Basophil Abs	< 0.1		x 10^9 /L	(< 0.4)
					Red and white cell morphology normal				

The pathologist's report stated: *marked CK increase with mild AST increase support a significant somatic myopathy. Mild AST increase may also reflect mild hepatopathy as it is accompanied by mild GLDH and cholesterol increase. Histopathology showed hyperplastic feather follicles, hyperplasia, hyperkeratosis, cystic dilation, and mural heterophilic infiltration. The perifollicular dermis had infiltration by lymphocytes, plasma cells, macrophages and heterophils, together with fibroplasia. The overlying epidermis was mildly hyperplastic and keratotic. Low numbers of epidermal stromal cells had pale-staining basophilic intranuclear bodies consistent with polyomavirus.*

The changes in the haematology and biochemistry were expected when considering the clinical signs. However, when the age, history and clinical progression of the disease are considered, this was an extraordinarily rare manifestation of polyomavirus. Sections of the histopathology specimens (skin biopsy) were sent to other pathologists for comment (Figs 1 and 2):

Comment 1: In the skin there was widespread acanthosis and orthokeratotic hyperkeratosis along with foci of apoptotic keratinocytes (Civatte bodies) within the basal layers and occasional subcorneal pustules containing mostly mononuclear cells. In the dermis there were multifocal mostly perivascular but also perifollicular nodular to diffuse lymphoplasmacytic rich inflammatory cellular infiltrations and areas of massive orthokeratotic hyperkeratosis and follicular plugging alongside dystrophic developing feathers, some of which had proliferative undifferentiating epithelium. The follicle wall of most of feathers was massively thickened by epithelial hyperplasia. Immunohistochemistry for psittacine beak and feather disease virus antigen was negative. DIAGNOSIS: Chronic dermatitis, folliculitis, pterylitis.

Some of the features in this biopsy are unusual for avian skin but are nevertheless non-specific. The changes are chronic and could be due to chronic infection (bacterial or fungal such as Malassezia) even though no aetiological agents were seen. There is no histological evidence of PBFD or other viral infections. Chronic, severe hypersensitivity (type 1) should be considered and this is the first avian case where I would also consider a mammalian equivalent to discoid lupus erythematosis (DLE) or other immune mediated disease.

Comment 2: There is an impressive amount of inflammation, specifically the lymphoplasmacytic component, and the ballooning degeneration of some of the epithelial cells, the hyperplasia and dysplasia. The lesion most closely resembles a bacterial or fungal dermatitis, but there is no evidence of either type of organism. The dysplasia and ballooning degeneration of the cells could also be viral induced, but inclusion were not seen and it would have to be caused by a virus that did not produce inclusions. Lastly, there is a disease in mammals called Erythema multiformans which can be associated with a drug reaction or viral infections and probably has other causes.

At this stage it was apparent that the inflammatory skin disease was unlikely to be due to an infectious process. Other possible causes of inflammatory skin disease may include nutritional deficiencies, especially as this bird had an all-seed diet, systemic metabolic disease such as liver or gastrointestinal disease, or endocrine disease. It was also possible the cockatiel had developed a form of immune mediated skin disease - either allergy, drug sensitivity or autoimmune disease. Drug sensitivity was unlikely since the bird had been unmedicated for two months prior and it had not improved.

A staged treatment trial was planned to assess the response and hopefully provide an idea of cause. The initial plan consisted of:

1. Vitamin ADEC injections weekly for 4 weeks;
2. Food elimination allergy trial for 6 weeks using only French white millet and fresh green vegetables (the bird would not eat parrot pellets);
3. Biotin supplement daily for 3 weeks;
4. Supplementation with Thyroxin for 3 weeks;
5. Non-steroidal anti-inflammatory medication for 2 weeks; and
6. If there was still no improvement then immunosuppressive therapy such as cyclosporine was to be considered. Cortisone was not considered as a treatment sa birds tend to be extremely sensitive to its adverse effects.

Since the owner had suddenly become ill, the food elimination allergy trial was started first since he could do this easily at home. Halfway through the six week trial the cockatiel grew a few crest and powder down feathers and the remaining feathers appeared healthier for the first time in several months. These however had fallen out by the time the trial was completed. Also during this time three small skin lumps had started to grow on the left lower eyelid, right carpus and on the left side over the crop. The bird had also started to pluck feathers from over the crop area.

The cockatiel had also started to show mild dyspnoea. Testing of previous feather picking cases had prompted us to consider a link between feather plucking and infection or allergic response to aspergillosis (Gill, 2001). It was assumed the cockatiel was immunosuppressed and that mild dyspnoea had now become part of the clinical presentation of this bird, so Itraconazole (*Sporonox*™) was administered at 10 ml/kg BID. The dyspnoea resolved within one month of treatment but the medication was continued for 12 weeks in case subclinical aspergillosis was still causing an allergic skin response. At the end of the course of Itraconizole there was no evidence of clinical improvement which supported the assumption that fungal infection was not a cause of the skin disease.

The treatment trial was resumed and the next phase was supplementation of multivitamins, especially vitamins A, D, E and C. Weekly doses of Vetafarm ADEC® injection were given at a dose of 0.1 ml per 150g bodyweight. This was repeated for 4 weeks. Additional multivitamins were mixed

with the water source daily using Ornithon® according to manufacturer's directions. During this time the three skin lumps mentioned above had reduced to a very small size. There was also an initial improvement in the skin of the feet and legs but this regressed after the second week. The owner now also reported that the cockatiel had become polyphagic and was eating at least twice as much seed as previously. This appeared to be a true polyphagia as there was a corresponding increase in the total volume of the droppings. The droppings were normal on both gross and microscopic examination. The weight of the bird had remained unchanged.

It was decided to continue the Ornithon® in the drinking water daily whilst the next stage of the trial was started. Biotin was added to the regime. Vetafarm Pigeon Liver and Cleansing Tonic® was used as a source of biotin - each tablet contained 91.5 µg of Cyanocobalamin (Vitamin B12), 6 µg Methionine, 91.5 g Biotin and 6 µg Lysine. The cockatiel was given ½ tablet three times a week. The tablets were crushed and dissolved in warm water and given orally. There was no change in the clinical presentation over the next month of treatment. The polyphagia also continued but there was no increase in the weight of the bird. The dose of Vetafarm Pigeon Liver and Cleansing Tonic was increased to ½ tablet once daily. Again there was no obvious response.

Three weeks later the owner reported that the cockatiel started having episodes of 'spasms' every couple of days. These 'spasms' would last for approximately 30 seconds in which the bird would appear to yawn, have mouth spasms and shake his head. From the description by the owner these episodes were likely to be small seizures. This behaviour was never seen at the hospital. Clinically the cockatiel showed no sign of neurological disease. The cockatiel also continued to have polyphagia without weight gain and the skin and feathers were still slowly deteriorating.

At this time it was decided to forego a trial with thyroxin. The signs of polyphagia without any gain in bodyweight and possible seizures were considered to be more indicative of hyperthyroidism rather than hypothyroidism. Alternatively intracranial disease such as a pituitary tumor was also considered as a possibility. If this was a dog, hyperadrenocorticism secondary to a pituitary would also be considered a possibility given the combination of clinical signs exhibited. Ante-mortem diagnosis of such possibilities is difficult in these birds due to the lack of data and testing facilities.

It was also decided to forego the trial with NSAID's as the clinical evidence did not support a disease syndrome that would be responsive to this class of medication.

Treatment was started with oral cyclosporine 5 mg/kg SID. The dose was decided based on the dose recommended for dogs and cats (Plumb, 2008) and that used by other avian veterinarians (Doneley, 2009). The cyclosporine medication was compounded into 'pina-colada' flavoured drops by Bova Compounding Pharmacy. On the third day after starting treatment the cockatiel had an adverse reaction to the cyclosporine oral medication. A few minutes after the medication was administered the skin became hyperaemic, the feathers appeared 'oily' and he was open mouth panting. The owner had him to the veterinary clinic within 20 minutes. On presentation the respiration was still rapid but had settled substantially according to the owner. The skin was still hyperaemic but the feathers seemed unchanged. Over the next 20 minutes the respiration and skin returned to normal (for what this bird usually presented for its skin!).

The owner was advised to withhold treatment for 48 hours, and to then give half the dose of cyclosporine at 2.5 mg/kg the following day. If there were no further abnormal reactions the dose was to be repeated at 2.5 ml/kg/day for 5 days before increasing to 5mg/kg/day. Ten days after the adverse reaction the cockatiel was presented for a follow-up visit. The owner reported that the skin

still went pink after the medication was administered but he did not observe panting. He also noted that he had not observed seizure-like behaviour since staring the cyclosporine. The skin was not as thickened and there was less scale present on the head. The bird's appetite was still ravenous. After discussion with the compounding pharmacist at Bova it was decided that the reaction to the cyclosporine may have been due to the flavouring. The product was remade using and different base to the flavouring - still pina-colada flavoured but this was water-based rather than oil-based. There were no further reactions to the medication.

After 6 weeks of cyclosporine new pin feathers appeared on the head. The skin was less inflamed than it had been prior to staring treatment with cyclosporine. There were now some feather cysts associated with the flight and contour feathers of both wings. During this time the dose of multivitamins was reduced to twice weekly. It was interesting to note that the appetite was now closer to normal. Subsequent changes in the frequency of multivitamins have resulted in a corresponding increase or decrease in the appetite of the cockatiel.

After 3 months of treatment with cyclosporine skin pathology appeared unchanged: the skin was still moderately inflamed and crusting; the head, neck and crop area were still essentially bald; most of the powder down was missing; the remaining feathers were sparse and in poor condition; and there were numerous cysts on the dorsal surface of both wings associated with the feather follicles. Material was removed from some of the cysts and cytology only showed cellular debris present. Bacteria or fungi were not evident. Despite minimal observable improvement, the owner believed that there was some improvement and wanted to continue with the therapy.

Additionally, during the third month of cyclosporine treatment the cockatiel had also started becoming ataxic and sometimes fell off the perch. This was not evident at the time of consultation. There was no seizure activity as described previously. The owner reported that this behaviour became less frequent and eventually stopped over the following 2 months.

Endocrine disease was and still is a possibility in this cockatiel. Serum cortisol and thyroid levels were examined. The results were as follows:

Total T4 = 12 nmol/L. Cortisol, resting < 28 nmol/L . (Please note: Lower limit of detection is 28nmol/L). Please note that there are no available reference ranges for these assays in this species in this laboratory.

From what is available in the literature the Total T_4 level is in the upper normal range and the resting cortisol is probably in the normal range as is does not appear to be elevated and the particular laboratory is unable to detect any lower levels.

Over the next few months the small skin lumps mentioned earlier grew significantly and several other lumps has also grown. These skin lumps were on the head, chest and wings and ranged from 4-12 mm in diameter. The lumps were prone to traumatise as the bird moved around the cage and small bleeds occurred regularly. The cockatiel also picked at one lump 12mm diameter on the left side of the chest. The follicular cysts were also increasing in size. It was decided to removed these small skin lumps and send both these and new skin biopsies for histopathology. A routine blood profile was performed again. The results were as follows:

BIOCHEMISTRY				HAEMATOLOGY			
			Reference				Reference
Glucose (Serum)	19.3	mmol/L	(12.7-24.4)	PCV	0.58	L/L	(0.41-0.59)
Urea	1.2	mmol/L		Platelets	Clumped &	Adequate	
Calcium	2.1		(2.1-2.7)	WCC	**20.2 H**	x 10^9 /L	
Protein Total	26		(21-48)	Heterophil %	**73 H**	%	(45-72)
Albumin	8		(8-18)	Heterophil Abs	14.7	x 10^9 /L	(2.0-19.1)
Globulin	**18 L**		(21-38)	Lymphocyte %	**17 L**	%	(26-60)
AST	301	IU/L	(128-396)	Lymphocyte Abs	3.4	x 10^9 /L	(0.6-9.4)
CK	**126 H**	IU/L	(160-420)	Monocyte %	**6 H**	%	(< 2)
Cholesterol	**8.1 H**	mmol/L	(2.3-5.2)	Monocyte Abs	1.2	x 10^9 /L	(< 1.4)
Amylase	234	IU/L	(113-870)	Eosinophil %	2	%	(< 3)
GLDH	**10 H**	IU/L	(< 3)	Eosinophil Abs	0.4	x 10^9 /L	(< 1.0)
Uric Acid	0.51	mmol/L	(0.20-0.65)	Basophil %	**2 H**	%	(< 2)
Sample Appearance	Normal			Basophil Abs	0.4	x 10^9 /L	(< 0.4)
Bile Acids (Random)	**14 L**	umol/L	(44-108)	Red and White cell morphology normal			

The leukogram suggests a mild inflammatory or stress response. GLDH increase suggests a mild persistent/recurrent hepatopathy although it is not supported by other liver parameters at this time. Mild hypercholesterolaemia is non-specific but may reflect diet, recent meal, hepatopathy or possible endocrinopathies.

Skin biopsy has a feather follicle present. Immediately subepithelial there is a small foci of heterophils and/or lymphocytes and/or plasma cells. Some surface debris. Another biopsy sample has a large keratogenous filled cyst with thick inner layer. No surface feather follicles but small sample. Surface has a small scab with heterophils, debris etc. Dermal small foci of mixed inflammatory cells. The skin 'lump' is a dense mass just under the epithelium composed of dense whorls and bundles of uniform strap-like cells, relatively bland pale moderate sized nuclei with pale pink cytoplasm. Mitoses are rare. The mass is interspersed with clumps of heterophils and/or plasma cells. Another skin 'lump' is a subcutaneous mass of whorls and bundles not as obvious and it is obscured by dense mononuclear foci. There are also several keratogenous cysts near the surface. DIAGNOSIS: Trauma inflammation and keratogenous cysts often from as a consequence of damaged and distorted surface and are consequential. The tumors are probably a smooth muscle tumor i.e. leiomyoma derived from feather attached smooth muscle (Fig. 3).

Leiomyomas in this area are benign tumors derived from smooth muscle, in this case the muscle of the feather follicle. It is likely to be secondary to chronic inflammation and irritation of the feather follicle. No new evidence was revealed with the biopsies (Fig 4).

Again the biochemistry and haematology results were as expected. A mild persistent or recurrent hepatopathy was assumed from the clinical progression of the cockatiel. Although the hepatopathy cannot be ruled out as the cause of the skin disease, higher values would be expected when considering the severity of clinical signs.

Treatment with cyclosporine was discontinued prior to surgery as the clinical signs of the cockatiel did not appear to be improving whilst on the medication. However, over the next three weeks the owner noticed that the cockatiel was scratching much more and staring to pick at his feathers. He also thought that the then bird had become much crankier than usual. The appetite of the cockatiel

was still more than that of the other normal cockatiels that he owned. On examination the skin appeared mildly thicker and redder and inflamed than previously. There was also more crusting apparent of the skin on the feet. The feather cysts on the wings had mildly reduced in size but there were fewer feathers present on the wings in general.

Treatment with cyclosporine was restarted on the observation that the cockatiel was more uncomfortable during the period when he was untreated. Interestingly, during the following 3 weeks the bird stopped scratching and picking at the feathers and was much happier in his general demeanor. The skin on the head, neck and chest appeared less inflamed and smoother to touch. The skin of the feet was still inflamed and crusting. The feather cysts on the wings had remained the same.

The dose of cyclosporine was increased from single daily dosage to 5 mg/kg x BID. After six weeks there appears to be a small amount of further improvement in the skin and some new pin feathers starting to emerge at the tail base and more powder down present. Currently this is the stage of the clinical progression of the bird.

DISCUSSION

Avian dermatology is an important part of clinical practice. Presenting complaints involving feather loss are frequently seen, yet there is very little information on inflammatory skin disease that is not associated with nutritional deficiencies or infection. Much of avian dermatology is still poorly understood, particularly dermatological expressions of hypersensitivities or allergies (Bauck, 1997) as was suspected in this case.

Folliculitis, often associated with *Staphylococcus* spp. infection, is commonly seen in birds. Grossly there is swelling of the perifollicular skin with a variable amount of reddening. This lesion must be differentiated from mycotic infections (Schmidt and Lightfoot, 2006). Folliculitis and dermatitis due to dermatophytes is less common in birds than in mammals. When present, there may be gross swelling of the follicles with variable hyperkeratosis and crust formation. A variable amount of necrotic debris may also be seen (Schmidt and Lightfoot, 2006).

Whilst bacterial infection is not suspected as the primary cause of the skin and feather problems in this bird, its integument is grossly abnormal and makes it susceptible to secondary infection. This is important in the management of this case. Similarly, fungal skin infection does not appear to be the primary cause of disease and fungi were not detected in the biopsies. It is assumed than any deeper or systemic fungal infection would have shown some improvement to the 12 week course of itraconizole. Finally, despite early suspicion of viral skin infection, evidence of viruses could not be demonstrated by histopathology.

A number of specific and non-specific nutritional problems can result in poor feather quality and skin disease (Schmidt and Lightfoot, 2006). The integument is where clinical signs of dietary inadequacy often appear first (Harrison and McDonald, 2006). Nutritional causes of hyperkeratosis are generally associated with dietary deficiencies of vitamin A, however excesses of vitamin A are also correlated with hyperkeratosis. In addition, both vitamins E and A may be required to treat hyperkeratosis due to vitamin A deficiency (Harrison and McDonald, 2006). Malnutrition is a possible factor of the underlying disease processes in this cockatiel as it had been fed and all-seed diet with little supplementation at the time it was initially presented. The bird was given four doses of injectable vitamins A, D, E and C and has been on oral multivitamin supplementation for 18 months. Although

supplementation is not a replacement for proper nutrition, the bird does not eat fresh vegetables offered daily. Changing the diet to one based on parrot pellets rather than seed was not an option as the owner had attempted to feed the cockatiel parrot pellets in the past but it would not eat them.

The choice to trial additional supplementation with biotin was based on that it has been shown to be beneficial in disorders of the skin and nails of other animals and to improve the quality of the coat and hooves of horses. In birds deficiencies of zinc and biotin have been associated with hyperkeratosis. Biotin deficiencies, which can result from excess salt, are correlated with hyperkeratosis on the footpad and the plantar surfaces of the toes (Harrison and McDonald, 2006). It is expected that correction of dietary deficiency is a long term process before it is evident clinically. However, given the length of time that this bird has been on supplements, some improvement in the clinical signs should have been seen if malnutrition was the main underlying disease process.

Hyperkeratosis of the feather sheath may occur as a result of malnutrition or in association with an infectious agent that affects the developing feather. The precise effects that malnutrition and organopathy, particularly hepatopathy, have on the quality of feathers remains undetermined. Their role is suggested clinically by the frequency of abnormal plumage in birds fed marginal diets and who have hepatopathies. Further, many generalized feather abnormalities will resolve when a bird is placed on a proper diet or when organopathy is effectively treated (Cooper and Harrison, 1999). In self-mutilation cases malnutrition appears to play a major role. The reasons for this could be multifactorial and include hepatic lipidosis, poor feather quality, the inability of the skin to resist infection and the predisposition to other illnesses (Gill, 2001).

Metabolic disease can also result from failure of proper metabolism even though nutrition is adequate. Gastrointestinal, hepatic and pancreatic diseases are potential underlying causes (Schmidt and Lightfoot, 2006). Blood results show there is evidence of persistent or recurrent hepatopathy in this bird. It is unknown whether the hepatopathy is primary or secondary to the skin disease. In the blood results only GLDH was elevated and other liver parameters were within the normal range for this species. The effect of the hepatopathy on the skin of this cockatiel is unknown, but the severity of skin abnormalities does not correlate to the mild hepatopathy suggested by biochemistry.

An endocrine problem has to be considered in this case as some types of feather loss may be metabolic in nature. A pituitary tumor is also possible given the neurological signs the bird was showing. The diagnosis of endocrine disorders in birds has proven to be difficult because multiple organ systems may be affected and clinical signs are varied. In addition, good clinical pathology tools for their diagnosis have been scarce and difficult to interpret. The variation between species in normal values has also been a stumbling block (Rae, 2000).

Hypothyroidism and hyperadrenocorticism have been discussed in the literature in association with feather loss, but little definitive evidence for diagnosis is given. Hypothyroidism in pet psittacine birds has been associated with excess fat deposition over the legs and abdomen and a delayed moult, as well as loss of contour feathers. Haematologic and biochemical abnormalities associated with hypothyroidism include a non-regenerative anaemia, mild heterophilia, hypercholesterolaemia and hypoalbuminaemia (Bauck, 1997, Danylyl et al., 2009). Although hypothyroidism is often suspected in psittacine birds, documentation of the condition is poor (Rae, 2000). The normal T4 levels in birds are much lower than those in mammals, and therefore care must be taken to ensure

that the avian resting T4 is measurable with a particular assay and not below the detection limits of the assay. T4 levels in birds may be influenced by increased plasma corticosterone, environmental temperature, handling, bleeding, food intake and some drugs (Rae, 2000). The definitive diagnosis of hypothyroidism should not be based on a single T4 value because multiple factors, including hyperthermia, systemic illness, certain drugs and stress, may cause it to fall within the hypothyroid range. These factors should not reduce the capacity of a normal thyroid to respond to TSH. The diagnosis of hypothyroidism should be confirmed by a TSH stimulation test (Rae, 2000). The baseline range for T4 levels in a cockatiel are 3.0-23.6 nmol/L (data and units converted from ng/ml multiplied by 1.27 to obtain nmol/L) (Rae, 2000). This cockatiel's T4 level was 12 nmol/L at rest and, without confirmation of a TSH stimulation test, is considered euthyroid. Unfortunately, sourcing the TSH for further testing has been unsuccessful. The signs of hyperthyroidism include polydipsia, polyuria, regurgitation, tachycardia, weight-loss, convulsions and death (Rae, 2000). There appear to be no documented cases of hyperthyroidism in birds in the literature. Thyroid neoplasia, both benign adenomas and malignant, invasive carcinomas, has been reported in birds, but unlike in some mammals, these tumors do not appear to be functionally secreting (Rae, 2000).

There are no documented cases of feather abnormalities resulting from hyperadrenocorticism or hypoadrenocorticism in birds although both conditions would be expected to occur (Cooper and Harrison, 1999). Although naturally occurring hyperadrenocorticism has not been reported in birds, the effects of exogenous corticosteroids have been studied. Exogenous corticosteroids increase food intake, increase plasma glucose and increase liver glycogen concentration resulting in liver enlargement. Corticosteroids also increase glomerluar filtration rate and produce glucosuria, which may present clinically as polyuria. In addition, a stress leukogram with lymphopaenia and a proportional increase in granulocyte populations may be seen with corticosteroid administration (Rae, 2000). Hyperkeratotic dermatitis and feather loss were reported in a macaw with histologic evidence of adrenal gland degeneration. This bird was on a poor diet and had numerous abscesses and pododermatitis. It is likely the adrenal gland degeneration in this bird was secondary to other medical problems and was not the primary cause of the lesions (Cooper and Harrison, 1999). Testing procedures for assessing adrenocortical function involving ACTH stimulation have been described in psittacine birds, and measurement of plasma corticosterone by radioimmunoassay, not cortisol, is the most appropriate for evaluating adrenocortical function in birds (Rae, 2000). The mean post-ACTH corticosterone concentrations were 4 to 14 times the mean baseline corticosterone concentrations (Rae ,2000). The baseline range for corticosterone levels for cockatoos range from 4.8-33.0 nmol/L (r cockatoos (data and units converted from ng/ml multiplied by 1.27 to obtain nmol/L) (Rae, 2000). This cockatiel's cortisol level was below the detectable limits of 28nmol/L at rest. This result gives no further information on the function of the adrenal glands in the patient and further testing using corticosterone is required further evaluation. This has not been possible as sourcing a laboratory capable of performing the test has proven unsuccessful.

Expected advances in diagnostic tools are anticipated to make the recognition of endocrine diseases in birds more definitive and their successful management attainable (Rae, 2000). Confirmation of endocrine disorders can also result from finding appropriate endocrine gland lesions at post-mortem (Schmidt and Lightfoot, 2006).

Allergic skin disease in birds is occasionally reported, but not well documented, and confirmation can be difficult. Gross changes include feather loss (often self induced), reddening and occasionally surface exudates (Schmidt and Lightfoot, 2006). Definitive diagnosis of allergic skin disease is difficult. Food elimination has led to improvement in some cases and successful treatment with anti-inflammatory drugs is presumptive evidence of allergy. In this case there was no clinical

improvement of the cockatiel with a food elimination trial. The response to cyclosporine is questionable, although the owner believes that there was some improvement. The greatly diminished response of the avian patient to histamine administration has hindered the development of avian skin testing methods (Schmidt and Lightfoot, 2006). Environmental factors such as low humidity, changes in day-length, household aerosols and cigarette smoke may all can lead to poor feather quality (Gill, 2001). Autoimmune skin disease has not been documented in birds, but several cases with intra-epidermal pustule formation and acantholysis have been seen, unfortunately these few cases were lost to follow-up (Schmidt and Lightfoot, 2006).

In many skin diagnoses there are inflammatory lesions whose exact aetiology cannot be determined. Based on the pattern and type of inflammation, a tentative diagnosis may be made, but until more cases with complete histories and follow-up information become available, many lesions will have obscure origins (Schmidt and Lightfoot, 2006).

Despite the bird's appearance, its quality of life is good - it is still vocal, eating and active. The owner has indicated he will allow a full necropsy when the bird dies.

Things that I have learnt with this case;

Always remember the old saying : "common things occur commonly BUT always expect the unexpected"

Looks are not everything – even the ugliest of patients will be adored by somebody (Figure 5).

ACKNOWLEDGMENTS

I thank Jim Gill, David Phalen and Shane Raidal for the opinions they gave in this case.

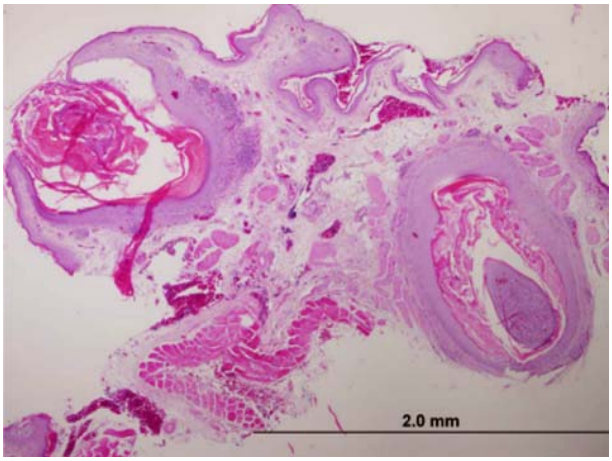

Figure 1 H&E

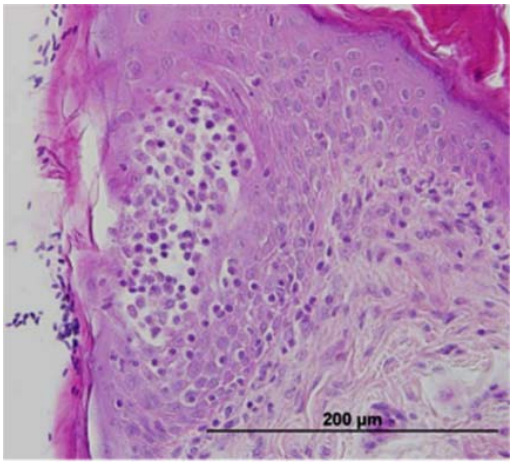

Figure 2 **H&E**

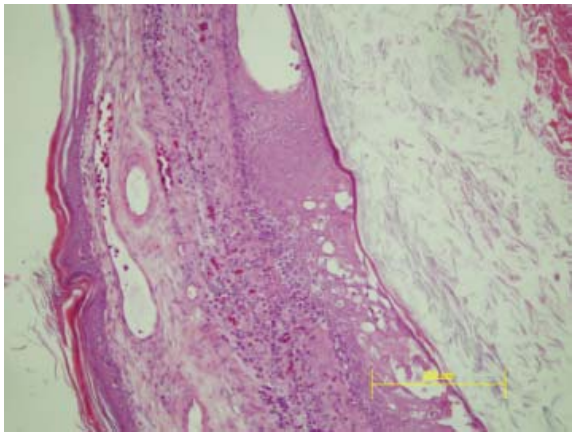

Figure 3: Subcutaneous keratogenous cyst H&E

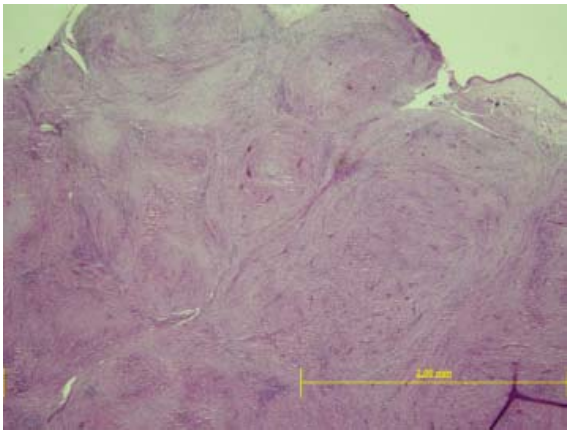

Figure 4: Subcutaneous myoma H&E

Figure 5: The Ugly Cockatiel

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